



Imperial College
London

Imperial Pituitary Masterclass Meeting

Monday 14th September 2026

IMPERIAL PITUITARY MASTERCLASS MEETING 2026

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

08.45 – 09:15 Registration.

SESSION 1

CHAIRS: Dr Anjali Amin, Imperial College Healthcare NHS Trust, London.
Mr Ramesh Nair, Imperial College Healthcare NHS Trust, London.

09:20 - 10:00 *Aggressive Pituitary Tumours.*
Dr Ben Whitelaw, King's College Hospital NHS Foundation Trust, London.

10:00 - 10:20 C001: *Metastatic Corticotroph PitNET.*
H Casey, EM Freel.
Department of Endocrinology, Queen Elizabeth University Glasgow.

10:20 - 10:40 C002: *Two Decades of Relentless Recurrence: Cushing's Disease, Nelson's Syndrome and the Challenge of Managing a Progressive Corticotroph Adenoma.*
L Narayanan, A Juszczak.
Heartlands Hospital, Birmingham, University Hospitals Birmingham NHS Foundation Trust.

10:40 - 11:00 C003: *How To Manage Gigantism – A Race Against The Epiphyseal Plates.*
V Mourougavelou, E Benyei, P Alexandros Drakos, N Raoof, A Bourg, M Korbonits.
Centre for Endocrinology, William Harvey Research Institute, Faculty of Medicine and Dentistry, Queen Mary University of London, London.
Department of Endocrinology, Barts NHS Health Trust, London.
Department of Ophthalmology, Barts NHS Health Trust, London.
Department of Oncology, University College London Hospitals NHS Foundation Trust.
Department of Neurosurgery, National Hospital for Neurology and Neurosurgery, London.

11:00 - 11:20 C004: *Misinterpretation Of Sella Pus, Leading To Visual Loss.*
C Shepherd C, Z Reisz, JM U-King-IM, BC Whitelaw, E Maratos.
King's College Hospital NHS Foundation Trust.

11:20 - 11:40 TEA & COFFEE BREAK

SESSION 2

CHAIRS: Dr Emma Hatfield, Imperial College Healthcare NHS Trust, London.
Dr Zin Htut, Imperial College Healthcare NHS Trust, London.

11:40 - 12:20 *A Practical Guide To Pituitary Imaging.*
Dr Emma Lim, Imperial College London Healthcare NHS Trust, London.

12:20 - 12:40 C005: *Subtraction ¹¹C-methionine PET/CT After Osilodrostat Identified A Surgical Target In MRI-negative Cushing's Disease.*
Z Htut¹, J MacFarlane², R Nair³, D Gillett², H Cheow², M Gurnell², F Wernig^{1,3}
1. Imperial College London. 2. Cambridge University Hospitals NHS Foundation Trust. 3. Imperial College Healthcare NHS Trust London.

- 12:40 - 13:00 C006: *Recurrent Hypophysitis Of Unknown Cause: A Challenging Journey.*
K Athanasiadou, S Luo, S Khan.
Department of Diabetes and Endocrinology, University College London Hospitals
NHS Foundation Trust, London.
- 13:00 - 13:20 C007: *The Adenoma That Changed Its Mind: Evolution from Macroprolactinoma to Recurrent Acromegaly.*
M Mankumare, D Sharma, A Sinha, T Purewal.
Royal Liverpool University Hospital.

13:20 - 14:10 LUNCH

SESSION 3

- CHAIRS: Dr Alison Wren, Chelsea and Westminster NHS Foundation Trust, London.
Dr Marcus Martineau, West Middlesex University Hospital, London.
- 14:15 - 14:55 *Prolactinoma In Pregnancy.*
Dr Marie Freel, Queen Elizabeth University Hospital, NHS Greater Glasgow and Clyde.
- 14:55 - 15:15 C008: *Rapidly Deteriorating Vision In Pregnancy.*
N Osman, N Raoof, R Khan, WM Drake.
St Bartholomew's Hospital, Barts Health NHS Trust.
- 15:15 - 15:35 C009: *Management Of Acromegaly Where Fertility Preservation Is A Priority: Use Of Methionine PET Imaging To Guide Second Trans-Sphenoidal Surgery.*
R Valecha, N Karimaghahi, I Boughazi, A Wren.
Department of Endocrinology, Chelsea and Westminster Hospital NHS Foundation Trust.
- 15:35 - 15:55 C010: *When Surgery Can Safely Wait: Multidisciplinary Management Of A Pituitary Macroadenoma In Pregnancy.*
B Bhatt^{1,2}, H Hussain², K Shea², F Hussein², F Islam², F Issa², A Shoakazemi², G Mlaw².
1. University College London Medical School.
2. Queen's Hospital Romford, Barking, Havering & Redbridge Hospitals NHS Trust.
- 15:55 - 16:15 C011: *A Double Whammy - Persistent Thyrotoxicosis Following TSHoma Resection.*
A Al-Qaysi, A Pal, J Tomlinson.
Endocrinology Department, Oxford Centre for Diabetes, Endocrinology and Metabolism (OCDEM).
- 16:15 Closing remarks and feedback

Poster Presentation Abstracts C012 to C021

<https://www.imperialendo.co.uk/pit2026>

C001

Metastatic Corticotroph PitNET.

H Casey, EM Freel.

Department of Endocrinology, Queen Elizabeth University Glasgow.

A 41-year-old man presented in 2013 with florid Cushing disease complicated by type 2 diabetes, hypertension, central obesity, vertebral fracture and mood lability. Pituitary MRI demonstrated a macroadenoma with suprasellar and right cavernous sinus extension. Transsphenoidal surgery (TSS) confirmed a Crooke cell corticotroph pituitary neuroendocrine tumour (PitNET), with a Ki-67 index of approximately 2%. Residual tumour measured 15 mm and involved the right cavernous sinus.

Biochemical recurrence was confirmed by failure to suppress on overnight dexamethasone testing in 2014, prompting bilateral adrenalectomy. He subsequently underwent biochemical, ophthalmological and radiological surveillance for corticotroph tumour progression and Nelsons syndrome after bilateral adrenalectomy. Imaging remained stable until 2020, when the residual tumour enlarged. ACTH was 180 ng/L before and 88 ng/L after hydrocortisone administration concordant with the imaging increase.

Repeat debulking TSS in 2021 again demonstrated a Crooke cell corticotroph PitNET, now with a Ki-67 index of approximately 8% and p53 nuclear positivity in approximately 80% of tumour cells, demonstrating pathological evolution. Postoperative MRI showed residual tumour within the suprasellar cistern and right cavernous sinus. Fractionated radiotherapy (45 Gy) was completed in May 2022. By December 2025, on imaging residual sellar tumour burden had reduced substantially.

In July 2026, he presented with three weeks of lethargy, dizziness, anorexia and malaise. Serum sodium was 121 mmol/L, and left anisocoria was identified. CT demonstrated acute hydrocephalus and a retroclival dural-based mass causing mass effect, requiring cerebrospinal fluid shunting. Whole-spine MRI additionally demonstrated a large ventral C5 intradural extramedullary lesion causing cord compression without cord signal change, and a smaller non-compressive T6–T7 lesion. Excision of the C5 lesion showed metastatic neuroendocrine tumour with a Ki-67 index of approximately 15%, consistent with metastatic corticotroph PitNET.

He is currently receiving palliative radiotherapy, with systemic palliative chemotherapy planned.

Questions for discussion

At what point should serial pathological progression in an invasive Crooke cell corticotroph PitNET trigger whole-neuraxis and whole-body staging, despite apparent radiological control of the irradiated sellar disease?

Any previous experience of such rare, aggressive tumour transformation?

C002

Two Decades of Relentless Recurrence: Cushing's Disease, Nelson's Syndrome and the Challenge of Managing a Progressive Corticotroph Adenoma.

L Narayanan, A Juszczak.

Heartlands Hospital, Birmingham, University Hospitals Birmingham NHS Foundation Trust.

A 36-year-old male diagnosed with Cushing's disease in 2002, underwent transsphenoidal adenomectomy (TSA) and pituitary radiotherapy in 2003, neither of which achieved remission. He subsequently underwent bilateral adrenalectomy in 2006 and commenced hydrocortisone and fludrocortisone replacement. He was lost to follow-up in 2011 and re-presented in 2021 with difficult-to-control hypertension, pre-diabetes, obesity (BMI 40.7), and obstructive sleep apnoea.

Notably, he had self-discontinued hydrocortisone for five years without experiencing an adrenal crisis.

On re-evaluation, MRI scan reported a pituitary macroadenoma measuring 14×18×14mm which doubled in size since 2011. CT adrenals revealed an 18mm soft tissue remnant adjacent to the right adrenalectomy clips, suggesting residual adrenal function. His ACTH was elevated at 350ng/L. Following pituitary MDT discussion, a diagnosis of Nelson's syndrome was confirmed.

A second TSA was performed in March 2022. Histology confirmed a corticotroph adenoma with Ki67 of 4%. Although immediate post-operative cortisol was suppressed, ACTH subsequently rose from 189 to 409 ng/L by August 2023. MRI demonstrated progressive right-sided residual disease abutting the optic chiasm. The MDT reviewed the case in 2023 and 2024, concluding that stereotactic radiosurgery (SRS) was not safe given the lesion's proximity to the optic nerve.

In September 2025, the patient presented acutely with right-sided third nerve palsy and visual deterioration, with imaging confirming further lesion growth. Following MDT in February 2026, a third TSA was performed in April 2026. Histology revealed a densely granulated corticotroph adenoma with Ki67 of 8%, reflecting increasing tumour aggressiveness. Post-operatively, ACTH fell from 629 to 335 ng/L; however, residual disease persisted in the bilateral cavernous sinuses. The MDT convened again in June 2026 and recommended further imaging and oncological review for consideration of radiotherapy or temozolomide.

Throughout this 24-year journey, coordinated MDT input from endocrinology, skull base surgery, neuro-ophthalmology, oncology, and weight management has been central to every major clinical decision.

Does a rising Ki67 index across sequential surgeries define this as an aggressive corticotroph tumour warranting chemotherapy? When SRS is precluded by optic nerve proximity, what is the optimal sequencing of debulking surgery, conventional radiotherapy, and medical therapy to balance tumour control with preservation of vision?

C003

How To Manage Gigantism – A Race Against The Epiphyseal Plates.

V Mourougavelou, E Benyei, P Alexandros Drakos, N Raoof, A Bourg, M Korbonits.

Centre for Endocrinology, William Harvey Research Institute, Faculty of Medicine and Dentistry, Queen Mary University of London, London.

Department of Endocrinology, Barts NHS Health Trust, London.

Department of Ophthalmology, Barts NHS Health Trust, London.

Department of Oncology, University College London Hospitals NHS Foundation Trust.

Department of Neurosurgery, National Hospital for Neurology and Neurosurgery, London.

Pituitary gigantism is a rare condition caused by growth hormone (GH)-secreting pituitary neuroendocrine tumours. Germline variants in the aryl hydrocarbon receptor-interacting protein (AIP) gene are associated with aggressive tumour behaviour and resistance to first-generation somatostatin analogues (1st-SSA).

A 17-year-old male presented with slipped capital femoral epiphysis. Over six months, he developed rapid linear growth, headaches, and cognitive decline. GH was grossly elevated ($>100 \mu\text{g/L}$, undiluted; normal $<1 \mu\text{g/L}$ post-glucose suppression) with only a modest elevation in IGF-1 ($750 \mu\text{g/L}$, $1.54\times$ and $1.50\times$ the age-adjusted upper limit of normal [ULN] of 487 and $500 \mu\text{g/L}$ for chronological and bone age), attributable to secondary hypothyroidism, hypoadrenalism and hypogonadism (testosterone undetectable; normal $9.9\text{--}27.8 \text{ nmol/L}$). Height 205cm, bone age 14y at chronological age 18.5y; MRI showed a pituitary macroadenoma ($37\times 36\times 25\text{mm}$) with cavernous sinus invasion and optic apparatus compression.

Three-month 1st-SSA treatment with hydrocortisone and suboptimal thyroxine replacement modestly reduced his GH ($58.9 \mu\text{g/L}$) and IGF-1 ($654 \mu\text{g/L}$; $1.34\times$ and $1.31\times$ ULN).

Transsphenoidal surgery identified a sparsely granulated somatotrophinoma (Ki-67 1-2%), with residual tumour remaining postoperatively; GH and IGF-1 fell further (29.6 and $540 \mu\text{g/L}$; $1.11\times$ and $1.08\times$ ULN). Genetic testing, indicated by young age and positive family history (mother: acromegaly, sister: macroprolactinoma), identified a novel truncating AIP variant [c.259C>T; p.(Gln87Ter)].

Due to NHS restrictions on combining GH-receptor antagonists with SSA, and given open epiphyses (7cm growth between hip and pituitary surgery) and AIP-related SSA resistance, pegvisomant monotherapy was initiated and titrated to 270mg/week over months. After starting testosterone replacement and fully normalising thyroxine, IGF-1 rose again, complicating disease control. He was referred for proton-beam radiotherapy for residual tumour.

The patient developed ocular discomfort after radiotherapy and was diagnosed with posterior blepharitis, with preserved visual function, attributed to acromegaly-associated meibomian gland dysfunction rather than radiation toxicity; it improved with topical lubricating eyedrops.

This case highlights diagnostic delay and treatment challenges in AIP-related gigantism with delayed epiphyseal fusion, identifying posterior blepharitis as an under-recognised GH-excess manifestation.

Questions for discussion

Should pegvisomant monotherapy be considered earlier in young patients with open epiphyses and AIP-related SSA resistance, to limit growth acceleration before definitive tumour control is achieved?

C004

Misinterpretation Of Sella Pus, Leading To Visual Loss.

C Shepherd, Z Reisz, JM U-King-IM, BC Whitelaw, E Maratos.

Kings College Hospital NHS Foundation Trust, London.

Background

Hypophysitis can mimic a variety of pituitary conditions, leading to delay in diagnosis and rapid worsening of visual function. We present a case where macroscopic features during neurosurgical biopsy initially misinterpreted as an infective process, led to a setback in effective treatment for inflammatory hypophysitis.

Case Presentation

A 71-year-old female with a history of stable Crohn's disease and treated breast cancer, presented with a rapid deterioration in temporal visual fields. MRI showed a suprasellar cystic lesion with extensive signal change in the optic pathways. She underwent open biopsy of the suprasellar lesion. The cyst contents were described as purulent. Histology was non diagnostic. Due to the macroscopic appearance, a diagnosis of abscess was made. The patient did not respond to empirical antibiotics and dexamethasone. Her vision progressively deteriorated over 6 months with visual loss in her right eye. Repeat imaging showed recurrence of the cystic suprasellar lesion. Imaging was reviewed and a radiological diagnosis of hypophysitis was made. She was transferred to our centre and initially empirically treated with high dose methylprednisolone and broad-spectrum antibiotics. However, her vision deteriorated further, and she therefore underwent a second biopsy. The cyst contents were creamy-white and did resemble pus but were more consistent with hypophysitis. There was improvement in vision post-operatively. Histology confirmed a chronic inflammatory infiltrate. The diagnosis was revised to steroid-resistant hypophysitis. After successful treatment with rituximab, there was radiological resolution of the cyst and stabilisation of her vision.

Conclusion

This case illustrates the diagnostic uncertainty in hypophysitis, with infection an important differential. The finding of purulent material in a lesion should not be interpreted as diagnostic of abscess or infection since this finding can occur with some forms of inflammatory hypophysitis, as shown in this case.

C005

Subtraction ^{11}C -methionine PET/CT After Osilodrostat Identified A Surgical Target In MRI-Negative Cushing's Disease.

Z Htut¹, J MacFarlane², R Nair³, D Gillett², H Cheow², M Gurnell², F Wernig^{1,3}

1. Imperial College London.
2. Cambridge University Hospitals NHS Foundation Trust.
3. Imperial College Healthcare NHS Trust.

Background

Long-term remission in Cushing's disease (CD) depends on complete resection of the ACTH-secreting adenoma while preserving normal pituitary function, yet a substantial proportion of corticotroph adenomas are not visible on conventional MRI. ^{11}C -methionine PET/CT (^{11}C -MET PET/CT) can localise MRI-negative tumours, but corticotroph adenomas are typically less methionine-avid than other functioning adenomas, in part because chronic hypercortisolaemia suppresses corticotroph metabolic activity. We hypothesised that preoperative cortisol lowering with osilodrostat — more effective and better tolerated than older steroidogenesis inhibitors — would relieve negative feedback, increase ACTH secretion and tumoural amino acid turnover, and thereby amplify ^{11}C -MET signal in an otherwise occult adenoma.

Case

A 28-year-old man with a history of juvenile arthritis, prior hip replacement, and postoperative pulmonary embolism presented with resistant hypertension, centripetal obesity, purple striae, and rapid weight gain. Investigations confirmed ACTH-dependent hypercortisolism: post-1 mg dexamethasone cortisol 807 nmol/L (<50), ACTH 56.7 ng/L (10–30), and 24-hour urinary free cortisol 774 nmol/24h (0–164). Late-night salivary cortisol was 13.5 nmol/L (<2.6) and salivary cortisone 42.7 nmol/L (<18).

Inferior petrosal sinus sampling excluded an ectopic source, but MRI showed no discrete adenoma; baseline ^{11}C -MET PET/CT demonstrated only equivocal uptake adjacent to the left cavernous sinus, insufficient for surgical targeting. Metyrapone was poorly tolerated and was switched to osilodrostat, titrated to achieve eucortisolaemia; ACTH subsequently tripled from 56.7 to 164 ng/L. After three months of sustained eucortisolaemia, repeat ^{11}C -MET PET/CT showed markedly increased focal tracer avidity at the same left para-cavernous site, and a subtraction (delta) image of post-treatment minus baseline scans isolated a discrete functional target. PET-directed transsphenoidal surgery yielded a histologically confirmed corticotroph adenoma, with day 5 cortisol <28 nmol/L and sustained remission thereafter.

Conclusion

Deliberate preoperative cortisol lowering may act as a functional signal enhancer for corticotroph adenomas, converting an equivocal ^{11}C -MET PET/CT into an actionable surgical target, with delta imaging isolating the signal change. If reproducible in larger cohorts, this approach could improve first-operation cure rates in MRI-negative CD while sparing normal pituitary tissue.

Questions for discussion

1. What degree and duration of cortisol suppression best enhances ^{11}C -MET PET/CT signal?
2. How does preoperative osilodrostat affect early postoperative cortisol and interpretation of remission criteria?
3. Does it alter the severity or duration of postoperative adrenal insufficiency?

C006

Recurrent Hypophysitis Of Unknown Cause: A Challenging Journey.

K Athanasiadou, S Luo, S Khan.

Department of Diabetes and Endocrinology, University College London Hospitals NHS Foundation Trust, London.

A 35-year-old woman was investigated privately for a 21 mm suspected pituitary macroadenoma in December 2021 following investigations for headaches and secondary amenorrhoea. Biochemical investigations showed elevated prolactin 1,810 (102-496) mIU/L, free T4 8.89 (11.9-21.6) pmol/L, FSH 3.1 (3.5-12.5) IU/L, LH 0.7 (2.4-12.6) IU/L, oestradiol <18 (114-332) pmol/L, and 9am cortisol 43 (133-537) nmol/L. She was commenced on hydrocortisone, levothyroxine, and cabergoline.

In February 2022, she presented to University College London Hospital with worsening headaches. MRI demonstrated a partly solid and cystic pituitary lesion suggestive of pituitary apoplexy. Posterior pituitary bright spot was not demonstrated; however, the patient did not present signs of AVP-deficiency. Visual fields were intact and prolactin normalised. Cabergoline was discontinued, and repeat imaging 10 days later showed reduction in lesion size. One month later, she re-presented with recurrent severe headache. MRI pituitary demonstrated pathological enhancement of the pituitary stalk and hypothalamus, raising suspicion for inflammatory or infective hypophysitis. Extensive investigations including serum ACE, IgG4, autoimmune screening, and chest X-ray were unremarkable. FDG-PET showed mild activity at pituitary fossa. Planned pituitary biopsy was deferred due to clinical and radiological improvement.

Between 2022–2024, while residing in Amsterdam, she experienced recurrent severe headaches with multiple hospital admissions and repeated empiric treatment with intravenous antibiotics and corticosteroids for presumed meningitis. Serial cerebrospinal fluid samples demonstrated raised white cell counts without microbiological growth. She underwent three pituitary biopsies and sellar clearance showing significant inflammation and pus; however, no infective organisms were identified. Repeat FDG-PET imaging and immunological investigations remained non-diagnostic. A clinical diagnosis of hypophysitis of unknown cause was made. In February 2025, immunosuppression with pulsed methylprednisolone and azathioprine 50mg BD was initiated.

Following return to the UK, serial MRI imaging has demonstrated radiological improvement and the patient remains clinically well.

Questions for discussion

1. Does the clinical and radiological response to azathioprine and corticosteroids constitute supportive evidence of immune-mediated disease?
2. Given the relapsing course, which potential differential diagnoses should be considered?
3. How should management be altered in the setting of azathioprine-induced bone marrow suppression?
4. What is the role of emerging biomarkers in hypophysitis?

C007

The Adenoma That Changed Its Mind: Evolution from Macroprolactinoma to Recurrent Acromegaly.

M Mankumare, D Sharma, A Sinha, T Purewal.

Royal Liverpool University Hospital.

A man presented aged 25 with headache, and MRI brain incidentally identified a 2.6 cm cystic sellar mass. Baseline testing confirmed a macroprolactinoma (peak prolactin 16,000 mU/L; reference <350) with hypopituitarism: insulin tolerance testing showed subnormal cortisol and growth hormone responses, GnRH testing showed low testosterone (9 nmol/L) with sub optimal LH/FSH response, and TRH testing showed a low baseline free T4. He was managed with cabergoline, hydrocortisone and, later, growth hormone replacement (suspended in 2020 due to non-attendance). Prolactin remained well controlled from 2017.

In 2023, despite normalised prolactin, IGF-1 rose progressively to 107 nmol/L (reference 11–33), and an oral glucose tolerance test confirmed failure of growth hormone suppression (nadir 1.8 µg/L)— the tumour had evolved from a mono- to a dual-secreting adenoma, with radiological growth and optic chiasmal compression. Family history was striking: his mother is a carrier of an AIP mutation, and his half-brother required trans-sphenoidal surgery for acromegaly without an identifiable AIP mutation. Germline AIP testing in our patient was negative.

He required emergency trans-sphenoidal surgery in May 2024 for pituitary apoplexy. Histology confirmed a sparsely granulated, Pit-1-positive somatotroph adenoma (PitNET). Biochemical remission followed (GH nadir 0.20 µg/L on OGTT, normal IGF-1).

By 2025–2026, IGF-1 rose again to 56 nmol/L (reference <32) with impaired GH suppression on OGTT (nadir 0.8 µg/L), and MRI showed recurrence with bilateral cavernous sinus involvement, judged unresectable for cure. The pituitary MDT recommended radiotherapy, with a somatostatin analogue (lanreotide) to be started as interim therapy.

This case illustrates a rare secretory phenotype switch in a pituitary adenoma, the surveillance challenge this poses after apparently "cured" disease, and the central role of the multidisciplinary team in guiding management of recurrent, multiply-treated pituitary disease.

Questions for discussion

1. Should long-term surveillance of "burnt-out" or medically controlled macroprolactinomas include periodic IGF-1 screening, given six years of normal IGF-1 preceded overt acromegaly?
2. In AIP-negative patients with a strong familial pituitary adenoma history, what is the role of further genetic or cascade family screening?

C008**Rapidly Deteriorating Vision In Pregnancy.**

N Osman, N Raoof, R Khan, WM Drake.

St Bartholomew's Hospital, Barts Health NHS Trust.

A 34 year old woman presented 36 weeks into her first pregnancy with a 4 month history of progressive, painless monocular loss of vision in her left eye with particular decline in colour vision. Her medical history included depression and anxiety. Menses were regular prior to (non-assisted) conception, she reported no galactorrhoea, and there were no symptoms of cortisol excess, acromegaly or arginine vasopressin (AVP) deficiency. There were no signs of an endocrinopathy on examination. Formal visual field testing showed left superotemporal visual loss, visual acuity of 6/48 in the left eye and OCT showed mild left eye ganglion cell loss. Vision in the right eye was normal. MRI demonstrated expansion of the pituitary gland, a thickened pituitary stalk and left optic nerve compression. Routine haematology and biochemistry were normal. Thyroid function was in keeping with the third trimester of pregnancy (fT4 10pmol/L, TSH 1.63 mU/L) as was serum prolactin (5842 mU/L). Between the ophthalmic assessment and endocrine referral her vision rapidly deteriorated with development of a left afferent pupillary defect and extension of the visual field defect into the temporal region. In view of worsening vision with risk to sight loss, the decision was made to treat with corticosteroids (initially intravenous methylprednisolone 1g and subsequently prednisolone 40mg daily) with a recommendation for induction of labour which she declined. Spontaneous vaginal delivery occurred shortly after at 38+2 weeks gestation, following which the dose of prednisolone was tapered over several weeks.

Postpartum assessment showed resolution of the previous visual deficit and interval imaging showed significant reduction in pituitary size with clear visual pathways. Menses have returned. Complications related to a ventricular septal defect in her son have prevented breast feeding.

Questions for discussion

1. Faced with this scenario and the presented radiology, would others have managed this patient differently?
2. How should we advise her about future pregnancies?

C009

Management Of Acromegaly Where Fertility Preservation Is A Priority: Use Of Methionine PET Imaging To Guide Second Trans-Sphenoidal Surgery.

R Valecha, N Karimaghahi, I Boughazi, A Wren.

Department of Endocrinology, Chelsea and Westminster Hospital NHS Foundation Trust.

A 30-year-old woman presented with oligomenorrhoea and primary infertility. Pituitary profile revealed acromegaly with elevated IGF-1 (90 nmol/L, NR <39 nmol/L), unsuppressed growth hormone (GH) during oral glucose tolerance testing (OGTT; mean 23.3 µg/L, nadir 16.3 µg/L, NR <0.4 µg/L), and non-diabetic hyperglycaemia. MRI revealed a pituitary macroadenoma invading the left cavernous sinus.

Pre-treatment with lanreotide autogel 120 mg every 4 weeks for 4 doses did not reduce tumour size or improve biochemical activity. Transsphenoidal surgery (TSS) was performed, with near-complete resection. Cortisol deficiency developed postoperatively. At 3 months post transsphenoidal surgery, the GH excess had improved but was not normalised: OGTT GH mean 3.25 µg/L, nadir 3.16 µg/L, and IGF-1 68.3 nmol/L. Hyperglycaemia had resolved. Histology confirmed a sparsely granulated somato-mammotroph tumour, which is known to be less responsive to somatostatin analogues. Genetic screening, including AIP testing, was negative.

Despite persistent biochemical disease, regular menstrual cycles resumed, and the patient wished to prioritise pregnancy before further treatment. Cabergoline was offered; however, before commencing treatment, she conceived. The pregnancy was not complicated by hypertension or gestational diabetes, and a healthy baby was delivered. Shortly postpartum, GH and IGF-1 levels were very similar to pre-conception values, and she again chose to defer further treatment. Subsequent biochemical monitoring showed progressive disease activity, with rising IGF-1 levels (72.8 nmol/L at 12 months and 75.0 nmol/L at 19 months postpartum) and persistent failure of GH suppression during OGTT (nadir GH 3.75 and 4.79 µg/L, respectively). Following multidisciplinary review, repeat TSS was pursued.

A surgical target abutting and invading the left cavernous sinus was identified on MRI and methionine PET. Redo transsphenoidal surgery in April 2025 resulted in biochemical remission, with IGF-1 normalising to 35.5 nmol/L by July 2025 and remaining normal 9 months after surgery at 30.9 nmol/L in January 2026.

OGTT January 2026:

Time	GH (µg/L)
0 min	1.95
30 min	1.49
60 min	1.60
90 min	1.96
120 min	1.56

Pre-clinic blood tests in August 2026 showed a normal random GH of 1.01 µg/L but a mildly elevated IGF-1 of 42 nmol/L.

Management has been discussed regularly within the regional pituitary multidisciplinary team. The patient wishes to have another child, creating uncertainty regarding the timing and choice of second-line medical therapy and radiotherapy.

Questions for the discussion

1. What future therapies should be considered, either now or after her family is complete?
2. Would the panel recommend second-line medical therapy?
3. Should radiotherapy, stereotactic radiosurgery, or proton beam therapy be reconsidered?

C010

When Surgery Can Safely Wait: Multidisciplinary Management of a Pituitary Macroadenoma in Pregnancy.

B Bhatt^{1,2}, H Hussain², K Shea², F Hussein², F Islam², F Issa², A Shoakazemi², G Mlawa².

1. University College London Medical School.
2. Queen's Hospital Romford, Barking, Havering & Redbridge Hospitals NHS Trust.

Introduction

Pituitary macroadenomas in pregnancy present unique clinical challenges, requiring careful management of the complications associated with tumour enlargement, and the risks associated with surgical interventions. We present a case of an enlarging macroadenoma with progressive visual compromise in late pregnancy, where intensive surveillance enabled safe deferral of surgery.

Case Presentation

A 32-year-old female was initially referred in August 2025 with worsening headaches and dizziness, on a background of migraines with aura and coronary artery disease. Subsequent investigations identified a non-functioning pituitary macroadenoma measuring 15x13.5x14mm. Notably, previous imaging from 2024 demonstrated no pituitary masses, indicating substantial development over a 6-9 month period.

The patient was re-reviewed in the first trimester of pregnancy, by both neurosurgical and endocrine teams. In the absence of visual compromise, conservative management was initially pursued, with monthly visual field testing and clinical review. However, in the third trimester, the patient reported worsening headaches and central visual blurriness. Repeat imaging revealed cystic changes and tumour enlargement to 18x19x24mm, with optic chiasm elevation and compression. Formal visual field testing demonstrated a superior quadrantanopia, prompting urgent multidisciplinary review.

Given the acute deterioration in visual fields and advanced gestation, management required careful coordination between the various clinical teams at our tertiary centre and the referring district general hospital. Consequently, a caesarean section was selected to optimise maternal and fetal safety, and pituitary replacement with Hydrocortisone 20mg and Levothyroxine 50µg daily was commenced until delivery. In the postpartum phase, serial visual field testing demonstrated consistent improvement in previous defects. On this basis, it was determined that neurosurgical intervention could be safely delayed for 4-6 weeks.

Discussion and Conclusion

This case exemplifies the complexities of managing progressive pituitary macro-adenomas in pregnant patients. Postpartum improvement in this case immediately alerted the risk-benefit balance of surgical intervention, emphasising the importance of regular re-assessment and re-evaluation. This case emphasises the importance of close, structured monitoring in enabling safe conservative management, preventing exposure to higher risk surgical procedures. It further demonstrates that dynamic multidisciplinary decision making is pivotal to achieving positive outcomes in complex pituitary cases during pregnancy.

Question for discussion

If you had this case in your centre – how would you have managed it?

C011

A Double Whammy - Persistent Thyrotoxicosis Following TSHoma Resection.

A Al-Qaysi, A Pal, J Tomlinson.

Endocrinology Department, Oxford Centre for Diabetes, Endocrinology and Metabolism (OCDEM).

Introduction

TSH-secreting pituitary tumours are rare causes of thyrotoxicosis and can be challenging to distinguish from other aetiologies. We describe a patient with a TSHoma who subsequently developed Graves' disease following pituitary surgery.

Case

A 57-year-old female was commenced on Levothyroxine abroad for unclear indications. Following her return to the United Kingdom one year later, Levothyroxine was discontinued after blood tests performed in primary care demonstrated thyrotoxicosis. However, biochemical thyrotoxicosis persisted on repeat testing using different assays, and her local team commenced Carbimazole in an attempt to normalise her thyroid function tests. This had no effect and was discontinued a few months later.

Subsequent assessment revealed persistent elevation of free thyroid hormones with non-suppressed TSH levels. Sex hormone-binding globulin was elevated, while the TSH α -subunit was normal. A thyrotropin-releasing hormone stimulation test demonstrated a blunted TSH response. An octreotide suppression test showed >50% initial TSH suppression, followed by non-sustained suppression at 24 and 72 hours. Genetic testing (panel R182) was negative for thyroid hormone resistance. Pituitary MRI revealed a 6 mm microadenoma. A TSH-secreting pituitary adenoma was diagnosed, and four weeks of treatment with octreotide was commenced prior to transsphenoidal resection. Histology confirmed a PIT1-lineage adenoma with thyrotroph morphology.

Initial post-operative thyroid function tests showed undetectable TSH with normal free thyroid hormone levels, suggestive of successful resection. However, she subsequently reported fatigue, and investigations revealed elevated free thyroid hormone levels with persistent TSH suppression despite not taking Levothyroxine. TSH receptor antibodies, which were initially negative, became positive during the post-operative thyrotoxic phase, and a thyroid uptake scan was consistent with Graves' disease. Treatment with Carbimazole resulted in normalisation of free thyroid hormone levels.

Conclusion

This case highlights the importance of a systematic approach to investigating abnormal thyroid function tests. Persistent or recurrent thyrotoxicosis following TSHoma resection may suggest residual or recurrent tumour; however, when TSH is undetectable, alternative aetiologies should be considered.

A Rare Case of Sellar Germinoma with Associated Lymphoid Hyperplasia Presenting with AVP Deficiency.

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Introduction

Sellar germinomas are rare intracranial germ-cell tumours that commonly involve the neurohypophysis and hypothalamic-pituitary axis. They can present with arginine vasopressin (AVP) deficiency (central diabetes insipidus), hypopituitarism and visual symptoms. The presence of prominent lymphoid infiltration may mimic inflammatory or autoimmune conditions, potentially resulting in delayed diagnosis. Early recognition is important, as germinomas are highly responsive to radiotherapy and chemotherapy and generally have a favourable prognosis.

Case

28-year-old man presented with a 5-year history of polyuria and polydipsia, drinking approximately 7litres of water daily. Past Medical history of hypothyroidism.

Blood test showed panhypopituitarism, with testosterone <0.1nmol/L, LH and FHS<0.3 IU/L, 9am cortisol 23nmol/L and reduced IGF-1 of 74µg/L. TSH 1.65mU/L with free T4 7.1pmol/L. Prolactin was within the normal range.

MRI of the pituitary demonstrated a large suprasellar mass measuring 27×14×19mm. The patient underwent an endoscopic extended trans-sphenoidal resection, complicated by a CSF leak requiring repair. The initial radiological and clinical differential included craniopharyngioma. However, histopathological examination demonstrated a germinoma with prominent atypical lymphoid infiltration, consistent with sellar germinoma with associated lymphoid hyperplasia.

He was diagnosed with panhypopituitarism and AVP deficiency and commenced on hydrocortisone, levothyroxine, desmopressin and testosterone replacement. Cerebrospinal fluid examination showed scattered reactive lymphoid cells with no atypical or malignant cells, and CSF was negative for malignant cells. CSF β-hCG and AFP were also within normal limits. He subsequently received proton beam radiotherapy.

Discussion

This case highlights the importance of considering sellar germinoma in patients presenting with AVP deficiency and evolving hypopituitarism, even when symptoms have been present for several years. AVP deficiency may precede the development of a radiologically apparent sellar lesion, potentially contributing to diagnostic delay. The prominent lymphoid component in this case further illustrates the potential for germinoma to mimic inflammatory or autoimmune pathology.

A sellar or suprasellar mass associated with AVP deficiency should prompt consideration of diagnoses beyond pituitary adenoma, including germinoma, craniopharyngioma, hypophysitis and other infiltrative lesions. Histopathological confirmation is therefore essential to establish the diagnosis and guide appropriate treatment. Germinomas are highly radiosensitive, and early multidisciplinary management involving endocrinology, neurosurgery, radiology and oncology is important to optimise outcomes.

Questions for discussion

In a patient presenting with AVP deficiency and subsequent hypopituitarism, what clinical or radiological features should raise suspicion of an underlying sellar germinoma rather than more common causes such as lymphocytic hypophysitis?

C013

Transient Panhypopituitarism Secondary to Rathke Cleft Cyst Presenting with Headache.

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Background

Rathke's cleft cysts (RCCs) are benign sellar/suprasellar lesions arising from epithelial remnants of Rathke's pouch and are usually asymptomatic, rarely progressing or causing new symptoms long term. Symptomatic presentation with acute headache and reversible hypopituitarism is rare and can clinically mimic pituitary apoplexy, posing a diagnostic challenge in the acute setting.

Clinical Presentation

A 51-year-old male with low-risk prostate cancer on active surveillance presented with a 5-day history of dull, holocranial headache with periorbital pressure, worsened by straining, without neurological symptoms. Examination revealed newly elevated blood pressure (177/88 mmHg) with no neurological deficits, including absence of papilledema; the remainder of the examination was normal. Blood pressure showed a paradoxical rise despite sequential antihypertensive therapy (nifedipine 10 mg, then ramipril 5 mg), with ECG and urinalysis unremarkable for hypertensive end-organ damage; previous GP records confirmed baseline normotension. MRI brain revealed a 10 mm Rathke cleft cyst with an intracystic nodule. A 9 am pituitary hormone profile demonstrated central hypothyroidism (TSH 1.03 mIU/L [0.3-4.8], free T4 6.5 pmol/L 7.7-20.6) and hypogonadism (testosterone 1.7 nmol/L [8.4-27.4], LH 1.4 IU/L [1.5-9.3], FSH 2.9 IU/L [1.5-18]). IGF-1 was elevated at 40 nmol/L (7-25.6), but an oral glucose tolerance test showed well-suppressed GH (<1 ng/mL), excluding autonomous GH hypersecretion. Baseline cortisol was 289 nmol/L, and a short Synacthen test confirmed intact adrenal reserve (30-minute cortisol 525 nmol/L). Lumbar puncture was deferred as headache improved clinically. There was no erectile dysfunction and no history of exogenous testosterone or supplement use. The patient was managed conservatively with analgesia (paracetamol and ibuprofen) and antihypertensive therapy, both contributing to gradual blood pressure normalisation. Repeat testosterone at 7 days showed partial recovery to the low-normal range (10.8 nmol/L). Complete biochemical resolution of panhypopituitarism was confirmed at 2-month follow-up, without hormone replacement or surgical intervention. Repeat MRI then showed cyst shrinkage to an isointense 5 mm lesion.

Conclusion

This case illustrates that Rathke cleft cysts can cause acute, fully reversible multi-axis pituitary dysfunction without apoplexy. The headache and panhypopituitarism most likely reflect an inflammatory reaction to cyst content leakage during spontaneous cyst regression, with hormonal recovery paralleling structural shrinkage.

Questions for discussion

In patients presenting with acute headache and biochemically confirmed panhypopituitarism in the context of a Rathke cleft cyst without hemorrhagic MRI changes, should conservative management with serial hormonal and radiological follow-up be the preferred initial approach?

C014

CSF Rhinorrhoea in a patient with invasive Macroprolactinoma Following treatment with Cabergoline.

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Background

Prolactinomas are pituitary neuroendocrine tumours that commonly present with symptoms of hyperprolactinaemia or mass effect. Dopamine agonists, particularly cabergoline, are first-line therapy and can cause rapid tumour shrinkage. We describe a patient with an incidentally diagnosed large prolactinoma presenting during assessment for suspected stroke, complicated by CSF rhinorrhoea following treatment with cabergoline.

Case Presentation

A 50-year-old man presented with a one-week history of intermittent arm tingling followed by one day of right facial tingling, facial droop, slurred speech and right-sided numbness.

CT brain and angiography excluded acute stroke but unexpectedly demonstrated a 3-cm heterogeneous sellar mass. MRI showed a 23 × 20 × 30 mm sellar mass with erosion of the sellar floor and extension into the sphenoid sinus, with suprasellar extension and predominantly left-sided optic chiasm compression. He reported no headaches or visual symptoms, and Humphrey 24-2 visual field test was normal. Retrospective history revealed erectile dysfunction and a previous episode of transient unilateral clear nasal discharge one year earlier, suspicious for CSF rhinorrhoea. Biochemistry demonstrated profoundly elevated prolactin at 19,372 mIU/L (reference range 56–278 mIU/L), with low testosterone at 4.2 nmol/L (reference range 8.4–27.4 nmol/L) and low-normal LH (1.7 IU/L; reference range 1.5–9.3 IU/L) and FSH (1.8 IU/L; reference range 1.5–18.0 IU/L).

Cabergoline 250 micrograms twice weekly was commenced but discontinued after one dose following the development of clear unilateral nasal discharge within five days. The pituitary multidisciplinary team recommended surgical management, and he subsequently underwent transsphenoidal surgery.

Histopathology confirmed a lactotroph PitNET (prolactinoma), with immunohistochemistry showing strong positivity for Pit-1 and prolactin, focal positivity for pan- α -subunit, and a Ki-67 proliferation index of 1%.

Conclusion

Large prolactinomas may remain clinically subtle despite extensive invasive disease. Although dopamine agonists remain the preferred initial treatment for prolactinomas, rapid tumour shrinkage may unmask or exacerbate CSF leakage when the tumour has eroded the sellar floor. In such cases, multidisciplinary assessment is essential, and surgical repair may be required before reintroduction of dopamine agonist therapy.

Questions for discussion

Should patients with macroprolactinoma and extensive sellar floor erosion undergo primary surgical repair before initiating dopamine agonist therapy?

C015

Aggressive Pituitary Neuroendocrine Tumour with DICER1 Mutation: A Diagnostic Challenge.

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Introduction

Pituitary neuroendocrine tumours (PitNETs) account for approximately 15% of intracranial tumours and arise from anterior pituitary neuroendocrine cells. Although most are benign, their location and size can cause significant mass effects, including optic chiasm compression and cavernous sinus invasion, as well as endocrine dysfunction. Early diagnosis and treatment are therefore important. The pathogenesis of PitNETs involves genetic mutations, including GNAS in GH-secreting tumours and USP8 in ACTH-secreting tumours, as well as epigenetic and signalling pathway abnormalities. Treatment depends on tumour subtype and may include surgery, medical therapy, radiotherapy and, in aggressive cases, chemotherapy or targeted therapies.

Case

A 41-year-old woman presented with a 3-month history of progressive headaches and right-sided visual loss. She had no significant past medical history.

MRI demonstrated a large 2.5 × 2.5 × 2.4 cm sellar/suprasellar mass, compressing the optic chiasm and invading the right cavernous sinus. CT chest, abdomen and pelvis showed no evidence of malignancy, and MRI of the spine showed no metastatic disease.

She underwent trans-sphenoidal debulking surgery and subsequently developed AVP deficiency, requiring desmopressin.

Three weeks after surgery, she developed rapid tumour recurrence with worsening visual loss. She received four cycles of cisplatin and etoposide chemotherapy, followed by radiotherapy, with significant tumour reduction.

Histology demonstrated a malignant neuroendocrine tumour with a Ki-67 proliferative index of 90%. Further molecular investigation using methylation profiling identified an embryonal tumour with multilayered rosettes, while NGS demonstrated a DICER1 mutation. Pituitary blastoma was considered in the differential diagnosis.

Discussion

Although most PitNETs have an indolent course, a small proportion can behave aggressively, with rapid recurrence, local invasion and, rarely, metastatic disease. Histological assessment, immunohistochemistry and proliferative markers such as Ki-67 are important in identifying high-grade tumours.

This case highlights the diagnostic complexity of an aggressive sellar tumour with unusual histological and molecular features. The 90% Ki-67 index, rapid postoperative recurrence and aggressive clinical behaviour prompted early multidisciplinary management.

An MDT approach involving neurosurgery, endocrinology, oncology, radiology and pathology was essential in establishing the diagnosis and guiding treatment. The tumour responded to chemotherapy; however, the patient was counselled regarding the guarded prognosis and high risk of recurrence and metastasis.

Questions for discussion

1. In patients with rapidly recurrent, high-grade sellar tumours and unusual molecular findings, should early molecular profiling be routinely considered to guide diagnosis and treatment?
2. Given the rapid recurrence in this case, should chemotherapy be considered earlier in patients with high-grade PitNETs?"

C016

Pituitary apoplexy precipitated by immune thrombocytopenia.

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Background

Classical pituitary apoplexy is a clinical syndrome of sudden headache, vomiting, visual impairment and reduced consciousness caused by haemorrhage or infarction of the pituitary gland, with ocular palsies from cavernous sinus involvement. It usually reflects a tumour outgrowing its blood supply. Where the bleed instead follows failed haemostasis, the presenting signs and the management may both differ.

Clinical case

An 83-year-old man with a 10mm macroadenoma acutely developed confusion, drowsiness, dizziness and a mild occipital headache over 4 days. He had noticed haematuria and easy bruising for 3 weeks.

He saw a neurologist privately and was referred to hospital only after a post-clinic blood test revealed platelets $11 \times 10^9/L$ [150-400].

On admission his sodium was 128mmol/L [133-146]. He had normal visual fields and acuity, and no ophthalmoplegia. CT head revealed an expanded pituitary containing high density suspicious for apoplexy, without chiasmal mass effect. The pituitary profile showed prolactin $<13\text{mIU/L}$ [86-324] and TSH 0.09mIU/L [0.27-4.2]; both had been normal 6 months earlier.

Blood film and bone marrow aspirate and trephine demonstrated increased megakaryocytes and peripheral consumption, confirming a diagnosis of immune thrombocytopenia (ITP). He received platelet transfusions, IV hydrocortisone and immunoglobulin 1g/kg, with rapid platelet recovery. Steroids were required for a dual purpose: to replace the hypothalamic-pituitary-adrenal axis and to treat ITP, so dose changes were approached cautiously. Haematology warned that the platelet response might have been steroid-dependent and that reduction could risk relapse of ITP.

His vision remained normal throughout and cognition improved. His TSH had recovered to 1.06mIU/L [0.27-4.2], without needing levothyroxine replacement therapy. He was discharged on prednisolone 3mg daily. Imaging at 3 months demonstrated involution of the lesion, with the stalk returned to the midline.

Conclusion

It can be just as crucial for the MDT to decide not to operate.

Questions for discussion

1. Apoplexy is a clinical diagnosis. Is it always visible on imaging?
2. How do we recognise apoplexy in a confused older patient whose vision is normal?
3. This lesion was only 10mm. Does the size of a macroadenoma predispose to bleeding, and should larger lesions change how patients are counselled?

C017

When biochemistry and imaging diverge: dopamine agonist-resistant macroprolactinoma.

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Background

Prolactinomas are the most common functioning pituitary adenomas and usually respond well to dopamine agonist (DA) therapy, with normalisation of prolactin and tumour shrinkage in most patients. DA resistance is uncommon, particularly in microprolactinomas. We describe a young patient with persistent biochemical disease despite escalating medical therapy and stable radiological appearances, highlighting the importance of multidisciplinary decision-making.

Case

An 18-year-old woman presented with a nine-month history of oligomenorrhoea progressing to amenorrhoea. Serum prolactin was 2396 mIU/L (reference range 102–496 mIU/L) and MRI demonstrated a 6 mm left-sided microprolactinoma. Cabergoline 500 micrograms weekly was commenced and progressively increased to 500 micrograms twice weekly and subsequently three times weekly due to persistent hyperprolactinaemia (2014–2691 mIU/L). Although menstrual cycles initially resumed, she subsequently developed increasingly infrequent cycles and secondary amenorrhoea. Adherence was repeatedly reviewed and confirmed. Serial MRI over six years demonstrated stable disease, measuring 6–7 mm without interval growth.

Mild biochemical hyperandrogenism raised the possibility of co-existing polycystic ovary syndrome; however, this was not considered sufficient to explain the degree of hyperprolactinaemia. Bromocriptine 1.25 mg twice daily was subsequently trialled but was poorly tolerated due to nausea and dizziness, with prolactin remaining elevated at 2131 mIU/L. Following regional pituitary multidisciplinary team (MDT) review, DA resistance was diagnosed. Cabergoline and bromocriptine were discontinued and combined oral contraceptive therapy commenced for oestrogen replacement and bone protection.

Given disease onset before 30 years of age, germline endocrine neoplasia testing (R217.1), including *AIP*, *CDC73*, *CDKN1B*, *MEN1*, *PRKAR1A*, *RET* and *VHL*, identified no pathogenic variants. Specialist pituitary review subsequently confirmed a 4 mm left-sided adenoma adjacent to, but not invading, the cavernous sinus. Transsphenoidal surgery was discussed, with an estimated >90% likelihood of cure, alongside fertility counselling.

Discussion

This case demonstrates marked biochemical persistence despite escalating DA therapy in the absence of radiological progression. MDT input was central to confirming DA resistance, excluding alternative and hereditary causes, addressing hypogonadism and bone health, and balancing fertility considerations against definitive surgical management.

Learning Points

- Consider DA resistance when prolactin remains elevated despite confirmed adherence.
- Consider hereditary endocrine neoplasia in young patients with pituitary disease.
- Early multidisciplinary review can guide fertility counselling and consideration of transsphenoidal surgery in medically resistant disease.

Questions for discussion.

In a young patient with a stable microprolactinoma and confirmed DA resistance, should transsphenoidal surgery be favoured over long-term hormonal management?

C018

Polyuria as a presenting feature of pituitary metastasis in HER2-positive breast cancer.

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A 47-year-old lady was referred to endocrinology due to a 6-month history of polyuria and polydipsia, drinking 7 litres of fluid per day and urinating every 2 hours overnight.

She had a background of HER2-positive breast cancer, treated with a left mastectomy, adjuvant chemotherapy, radiotherapy, and HER2-targeted therapy in 2021. In 2024 she had a recurrence and received further chemotherapy with an excellent treatment response. No checkpoint inhibitor therapy was given. One month prior to symptom onset she was started on maintenance HER2-targeted therapy (Pertuzumab/Trastuzumab).

HbA1c and calcium were normal. The patient underwent a water deprivation test which suggested AVP-deficiency; plasma sodium rose to 148mmol/L (NR 135-145mmol/L) with dilute urine (133mOsm/kg, NR 50-1200mOsm/kg), which rapidly corrected to 563mOsm/kg with DDAVP. Regular desmopressin was commenced.

A pituitary profile demonstrated low cortisol (40nmol/L, NR >350nmol/L), secondary hypothyroidism (FT4 8.6pmol/L, NR 9-23pmol/L; TSH 0.64milliunit/L, NR 0.3-4.2milliunit/L), and low LH (1.6unit/L, NR >20unit/L) and FSH (8.8unit/L, NR >20unit/L) relative to her post-menopausal state. Prolactin 1931milliunit/L (NR <550milliunit/L) suggested stalk disconnection. Prednisolone and levothyroxine were initiated. A subsequent SST showed an insufficient ACTH response (Cortisol <28; 95; 134nmol/L).

The pituitary MDT reviewed the patient's pituitary MRI which showed a new heterogeneously enhancing solid/cystic lesion encompassing the neurohypophysis and pituitary stalk, with loss of the normal posterior pituitary bright spot, and stretching of the optic chiasm. Visual fields were normal. Differentials included a primary neurohypophyseal/stalk lesion or hypophysitis. However, multiple new brain metastases were seen, so the pituitary lesion was presumed most likely a metastasis (no tissue confirmation).

Oncology confirmed a good prognosis of >1 year, however felt the lesion was too close to the chiasm for stereotactic radiosurgery. The neurosurgeons advised that debulking was technically possible and might make radiotherapy safer. The case was referred to the neuro-oncology MDT for decision on treatment; they elected for systemic therapy which the patient has now commenced.

Discussion

Pituitary adenomas almost never cause AVP-deficiency, whereas metastases typically seed the neurohypophysis and stalk. Anterior and posterior pituitary disease with a stalk lesion should therefore be presumed infiltrative or metastatic until proven otherwise.

Questions for discussion

In a patient with an expected survival over a year and no visual deficit, is there a role for debulking purely to create a safe margin for radiotherapy, or does an intact chiasm mean systemic therapy first? What would change your answer: field loss, or growth on interval imaging?

C019

Incidentally Detected Non-Functioning Pituitary Macroadenoma in a Patient with LADA and Metastatic Gastric Cancer.

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A 59-year-old woman with a 25-year history of LADA (HbA1c 8.4%, C-peptide 0.755 ng/mL – confirming absolute insulin deficiency) and stage IV HER2-positive gastric adenocarcinoma undergoing chemo-targeted therapy underwent whole-body ¹⁸F-FDG PET-CT for oncological staging, which revealed a focus of markedly increased FDG uptake in the pituitary region, measuring 1.7×1.2 cm axially (SUVmax 50.44), without measurable enlargement or sellar expansion.

Subsequent contrast-enhanced pituitary MRI confirmed a 1.2×1.2×0.8 cm mass in the right hemi-pituitary with infrasellar extension.

Anterior pituitary function was assessed: an earlier cortisol measurement was 159.5 ng/mL (50–250). Repeated morning cortisol was 319.0 nmol/L (171.0–536.0), with preserved nocturnal nadir (23:00 cortisol 82.3 nmol/L; reference 64.0–327.0), arguing against adrenal insufficiency; ACTH was 17.6 pg/mL (7.2–63.3). Prolactin was mildly elevated at 28.33 ng/mL (4.79–23.30), attributed to probable pituitary stalk compression. Growth hormone was 0.79 ng/mL (0.13–9.88) with low IGF-1 of 40.54 ng/mL (63.30–190.00). FSH was 52.5 mIU/mL (25.8–134.8), consistent with menopausal status. Aldosterone, renin, and aldosterone-renin ratio were within normal limits.

Goldmann perimetry demonstrated peripheral visual field constriction: right eye 25° superiorly and nasally with 5° inferiorly; left eye 15° in the superior, nasal, and inferior fields.

Given the absence of hormonal hypersecretion and the lack of visual field defects clearly attributable to chiasmal compression, the neurosurgical team concluded there was no current indication for surgical intervention; follow-up MRI and perimetry were recommended.

Questions for discussion

1. How should the markedly elevated FDG uptake (SUVmax 50.44) in a lesion of this size be interpreted — does this degree of metabolic activity warrant reconsideration of the "non-functioning" classification or closer surveillance?
2. Given the discrepant cortisol values and concurrent oncological treatment, what is the optimal approach to definitively exclude adrenal insufficiency in this patient before continuing chemotherapy?

C020

It never gets old: navigating Cushing's disease in a young adult.

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A 16-year-old female presented with an 18-month history of intermittent sweating, acne, hirsutism, oligomenorrhoea, 12kg unintentional weight-gain, purple abdominal striae and recurrent infections (pilonidal sinus and vulva boils). Private investigations showed random cortisol 1240nmol/L. Following NHS referral, her symptoms transiently settled and local investigations (blood pressure, overnight dexamethasone suppression test (ODST), urinary free cortisol (UFC) and pituitary profile) were normal.

Symptoms recurred four months later, prompting ICHNT referral. Examination revealed a round face, suprascapular fat pad, acne and marked abdominal striae without proximal myopathy or bruising. She was normotensive with intact visual fields and had never used exogenous steroids. She was studying for A levels, which was increasingly challenging with her symptoms.

Investigations confirmed ACTH-dependant Cushing's syndrome: UFC 1,150nmol/day (0-164nmol/day), late night salivary cortisol 22.2nmol/L (<2.6nmol/L) and cortisolone 98.4nmol/L (<18nmol/L), ACTH 77.6ng/L and unsuppressed ODST 580nmol/L (<50nmol/L). Dynamic pituitary MRI showed a left adenoma abutting but not invading the cavernous sinus. Inferior Petrosal Sinus Sampling excluded an ectopic ACTH source. She commenced rivaroxaban and metyrapone/prednisolone block-and-replace therapy.

The Imperial Pituitary MDT discussed and explored surgical logistics (linkage with school and young adult support services), care setting (paediatric vs adult including GOSH/Queen's Square paediatric neurosurgery input) and, given her age, her decision-making capacity. Joint adult-paediatric endocrinology assessment confirmed capacity. She and her family chose surgery at ICHNT, which was timed around her studies and half-term break.

She underwent endoscopic trans-sphenoidal resection of her pituitary microadenoma, with complete radiological resection. Histopathology confirmed a corticotroph pituitary adenoma (T-pit lineage) with high Ki67% 10-15%. Genetic testing for familial isolated pituitary adenoma was negative.

Post-operative cortisol was low, and she was discharged on prednisolone 6mg OD, which was slowly weaned off completely (balanced with her studies) following satisfactory cortisol day curves and normal 9am cortisol/ACTH off steroids.

Close monitoring with serial MRIs (3-monthly initially) showed no residual/recurrent disease. Off metyrapone, her late-night salivary cortisol (1.0nmol/L) and cortisolone (11nmol/L) are normal, and she has lost 5kg.

Questions for discussion

1. How should Cushing's disease be managed in young adult patients?
2. Should radiotherapy be considered given the high Ki67 index, despite ongoing biochemical remission?

C021

Four Doses, Dramatic Recovery: An Exceptional Response to Low-Dose Cabergoline in a Giant Macroprolactinoma.

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Background

Giant macroprolactinomas account for fewer than 5% of prolactinomas and typically present with profound hyperprolactinaemia, invasive tumour growth and visual compromise. Dopamine agonists are first-line therapy, although the speed and magnitude of response are variable. We present a young woman with a giant macroprolactinoma demonstrating exceptional biochemical, neuro-ophthalmological and radiological improvement after only four doses of low-dose cabergoline.

Case

A 20-year-old woman presented with a two-month history of headache, visual disturbance described as blind spots and absent withdrawal bleeding while taking the combined oral contraceptive pill. Formal perimetry confirmed bitemporal hemianopia. MRI demonstrated a 3.9 × 3.4 × 3.0 cm giant pituitary macroadenoma with suprasellar extension, optic chiasmal compression and bilateral cavernous sinus invasion. Serum prolactin was >100,000 mIU/L (reference range 102–496 mIU/L), confirming a giant macroprolactinoma.

Cabergoline 250 micrograms twice weekly was commenced with counselling regarding the rare risk of pituitary apoplexy. Serum prolactin fell to 2,129 mIU/L after the second dose, 749 mIU/L after the third dose and 397 mIU/L after the fourth dose, a reduction of over 99%. Visual field defects resolved after the third dose, while repeat MRI after the fourth dose demonstrated marked tumour shrinkage with decompression of the optic apparatus.

Discussion

This case demonstrates that even highly invasive giant macroprolactinomas may exhibit extraordinary sensitivity to dopamine agonist therapy. Rapid biochemical, neuro-ophthalmological and radiological improvement reinforces medical therapy as first-line treatment, even in patients with significant tumour burden. Predictors of such exceptional responses remain uncertain.

Learning Points

Low-dose cabergoline can induce dramatic early biochemical, neuro-ophthalmological and radiological improvement in giant macroprolactinomas. Early recognition, prompt treatment and multidisciplinary follow-up may avoid surgery even in markedly invasive tumours.

Questions for discussion

1. Which tumour characteristics predict an exceptional early response to dopamine agonist therapy?
2. Should early biochemical improvement alter the timing of repeat MRI and formal visual field assessment?
3. Should patients with giant macroprolactinomas and visual field defects routinely receive an initial trial of dopamine agonist therapy before neurosurgical intervention?