Society for Endocrinology

Imperial College London

Imperial Pituitary Masterclass Meeting

Monday 15th September 2025

IMPERIAL PITUITARY MASTERCLASS MEETING 2025

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

09.00 – 09:30 Registration.

SESSION 1

09:30 - 10.00 *Acromegaly and cancer.*
Dr John Ayuk, Queen Elizabeth Hospital, Birmingham.

10:00 - 10:20 *Two unusual faces of acromegaly: a high-risk surgical case with AVP deficiency potential and a rare SF1/PIT1 double-positive adenoma.*
H Imtiaz[1], S Gohil[1], N Reddy[1,2], MJ Levy[1,2].
1. University Hospitals of Leicester NHS Trust.
2. University of Leicester.

10:20 - 10:40 *Is there a need to improve the management of aggressive/ refractory acromegaly?*
B Patel[1], D Peters[1], K Meeran[1,2], F Wernig[1], E Hatfield[1], E Lim[1], Z Jaunmuktane[3], A Falconer[1], J Mariadas[1], R Nair[1], N Martin[1,2].
1. Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.
2. Imperial College London.
3. University College London Hospitals NHS Foundation Trust

10:40 - 11:00 *Beyond benign - An unexplained raised alkaline phosphatase level leading to the diagnosis of a Pit-1 positive mixed somatotroph-lactotroph pituitary neuroendocrine tumour with radiological features suggestive of potential metastatic disease.*
A Raju[1], L Bridges[2], A Martin[2], D Sivakumaran[1], G Bano[2].
1. St George's University Hospitals NHS Foundation Trust, London.
2. Kingston and Richmond NHS Foundation Trust.

11:00 - 11:20 *Isolated ACTH deficiency due to presumed postpartum lymphocytic hypophysitis and consequent empty sella syndrome presenting with severe symptomatic adrenal insufficiency.*
D Perera, S Nag, N Skariah.
South Tees Hospitals NHS Foundation Trust.

11:20 - 11:50 TEA & COFFEE BREAK

SESSION 2

11:50 - 12:10 *Preserved gonadal function despite panhypopituitarism.*
K Koysombat[1], P Behary[1], E Hatfield[1], M Patel[1], M Gonzalez[1], A Abbara[1,2], N Martin[1,2], F Wernig[1], K Meeran[1,2].
1. Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.
2. Imperial College London.

12:10 - 12:30 *A skull base eroding macroprolactinoma: the dilemma of treating conservatively, medically or surgically.*
O Abdalrazag, M Debono.
Sheffield Teaching Hospitals NHS Trust.

12:30 - 12:50 *Aggressive silent corticotroph adenoma with recurrent disease, pituitary apoplexy, and life-threatening complications: a longitudinal case report.*
A Rahman[1], Q Marwat[1], R Stanworth[1], H Ali[2].
1. Department of Endocrinology, Royal Derby Hospital, University Hospitals of Derby and Burton. Derby.
2. Department of Endocrinology, Queens Hospital Burton, University Hospitals of Derby and Burton. Burton.

12:50 - 13:10 *Cushing's disease; never an easy ride*.
J Lee Siew Hua[1], C Bartlett[1], R Gorrigan[1], H Marcus[2], WM Drake[1].
1. St Bartholomew's Hospital, Barts Health NHS Trust, London.
2. National Hospital For Neurology and Neurosurgery, Queen Square, London.

13:10 - 14:10 LUNCH

SESSION 3

14:10 - 14.40 *Acute management of dysnatraemia in pituitary/neurosurgical patients.*
Prof Mark Sherlock, Beaumont Hospital, Dublin.

14:40 - 15:00 *Clinical outcomes following supply driven transition from intranasal to oral desmopressin in AVP-D- A single centre experience.*
Dr Rupa Ahluwalia, Norfolk and Norwich University Hospitals NHS Trust.

15:00 - 15:20 *Challenges of a rapidly progressive pituitary metastasis – surgery, AVP deficiency, chemotherapy and palliative care.*
AW Paracha, M Levy, S Gohil, N Reddy.
Leicester Royal Infirmary, University Hospitals of Leicester NHS Trust.

15:20 - 15:40 *Hyponatremia in AVP deficiency during prolonged childbirth: the double-edged sword of desmopressin in prolonged labour.*
L Eltayieb, E Shakoor, M Dram, J Aikpitanyi, O Oluwaseun, F Hussein, H Wong, S Mukadam, A Mota, A Abdullah, M Ashwini, M Muhammad, B Hossain, G Simon, P Sivarajasinga ,E Phillips, F Fahal, G Mlawa.
Barking, Havering and Redbridge University Hospitals NHS Trust, Romford.

15:40 - 16:00 *Pituitary abscess: a rare cause of pituitary mass.*
M Blair, M Freel, S Hassan.
Queen Elizabeth University Hospital, NHS Greater Glasgow and Clyde.

Oral Presentation Abstracts

Two unusual faces of acromegaly: a high-risk surgical case with AVP deficiency potential and a rare SF1/PIT1 double-positive adenoma.

H Imtiaz[1], S Gohil[1], N Reddy[1,2], MJ Levy[1,2].

1. University Hospitals of Leicester NHS Trust, Leicester.
2. University of Leicester, Leicester.

Abstract
Acromegaly typically caused by GH-secreting pituitary adenomas can present with diverse clinical, radiological, and pathological features. We present two complex cases that illustrate the therapeutic challenges associated with atypical tumour morphology, surgical risks, and rare histological subtypes.

Case1
A 45-year-old man with a five-year history of reduced libido presented with clinical features of acromegaly. Investigations showed low testosterone with suppressed gonadotrophins. IGF-1 was elevated, and GH failed to suppress during an oral glucose tolerance test. MRI revealed a large pituitary lesion (11.5 × 8 × 18.5 mm) with suprasellar extension contacting the right optic nerve and chiasm. The tumour had an unusual “anchor-like” shape crossing the pituitary stalk. Methionine PET showed diffuse tracer uptake throughout the gland. Due to the tumour’s configuration, surgery carried a high risk of vasopressin deficiency and CSF leak. Visual field testing revealed mild right superior nasal quadrantanopia. Given these risks, lanreotide (somatostatin analogue) was initiated. Despite treatment, IGF-1 and GH levels remained elevated, although the patient reported minimal symptoms and imaging showed no significant tumour growth.

This situation poses a critical question: Should we continue suboptimal medical therapy or pursue high-risk surgery for potential cure and control?

Case 2
A 29-year-old man reported classical features of acromegaly since adolescence. IGF-1 was significantly elevated (811 ng/mL), and GH failed to suppress (nadir 7.4 µg/L). MRI revealed a large lesion (3 × 3.9 cm) with bilateral cavernous sinus involvement. Given the large tumour size, preoperative lanreotide was commenced, followed by transsphenoidal surgery. Postoperatively, he developed transient AVP deficiency requiring desmopressin. Histology revealed a rare combined PIT1/SF1-positive pituitary adenoma—a plurihormonal PitNET with dual lineage differentiation, which may have implications for tumour behaviour, hormone secretion, and recurrence risk. Given his young age and family history, genetic evaluation through a pituitary tumour gene panel was undertaken.

PIT1/SF1 co-expression is a more recently recognised subtype, what are the long-term prognostic implications of PIT1/SF1 co-expression compared to pure PIT1 or SF1 lineage tumours?

Discussion
These cases highlight the importance of individualized, multidisciplinary management in acromegaly. Rare histological subtypes and complex tumour anatomy may significantly impact treatment decisions. Advanced imaging, genetic evaluation, and careful surgical risk assessment are essential for optimizing outcomes in challenging presentations.

Is there a need to improve the management of aggressive/ refractory acromegaly?

B Patel[1], D Peters[1], K Meeran[1,2], F Wernig[1], E Hatfield[1], E Lim[1], Z Jaunmuktane[3], A Falconer[1], J Mariadas[1], R Nair[1], N Martin[1,2].

1.Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.
2.Imperial College London.
3.University College London Hospitals NHS Foundation Trust.

Background
Up to a third of aggressive and/ or refractory pituitary tumours are growth hormone (GH) secreting and predominantly affect young patients. Despite their prevalence, there is no definitive consensus on the diagnostic criteria or treatment strategy for their management.

Clinical Case
A 23 year old man presented to ENT with right sided tinnitus. An MRI revealed a large sellar and suprasellar pituitary macroadenoma invading the right cavernous sinus and encasing the right internal carotid artery. Biochemistry demonstrated elevated baseline GH (7.26µg/L) and insulin-like growth factor-1 (IGF-1, 92.3nmol/L, RR 10.5-47.1) levels with failure to suppress GH following oral glucose (nadir 3.7ug/L). Secondary hypocortisolism, hypothyroidism and hypogonadism were also noted due to tumour mass effect on the pituitary gland. His genetic studies and prolactin levels were normal, and he had no evidence of cardiac disease.

He was commenced on lanreotide for GH reduction and prednisolone, levothyroxine and testosterone replacement. Given the size of the tumour and its proximity to critical structures, a combined approach of surgery followed by radiotherapy for residual disease was advised by the MDT. He underwent extensive debulking surgery and histology revealed a sparsely granulated somatotroph adenoma (Ki67-index 1-3%). Post operatively, his MRI demonstrated a small residual tumour, and his GH and IGF-1 levels were 1.85µg/L and IGF-1 60.5nmol/L, respectively.

At three months following surgery, his GH (3.0µg/L) and IGF-1 (84.5nmol/L) levels began to rise despite regular lanreotide, and his treatment was changed to pegvisomant. The patient was apprehensive about radiotherapy, and he was advised it could be delayed if the tumour size remained unchanged and he achieved sufficient reduction in IGF-1 with pegvisomant. At 12 months after surgery there was a two-fold increase in the size of the residual tumour. Given the lack of biochemical response to lanreotide, tumour enlargement on pegvisamont, and histological findings, pasireotide was initiated. Additional local treatment was discussed; the MDT recommended repeat debulking surgery followed by radiotherapy.

Questions
What predictive markers can aid in the diagnosis of aggressive/refractory acromegaly?
Should pasireotide have been used earlier?
What are the concerns about radiotherapy in a patient of this age?

Beyond benign - An unexplained raised alkaline phosphatase level leading to the diagnosis of a Pit-1 positive mixed somatotroph-lactotroph pituitary neuroendocrine tumour with radiological features suggestive of potential metastatic disease.

A Raju, L Bridges, A Martin, D Sivakumaran, G Bano.

St George's University Hospitals NHS Foundation Trust, London.
Kingston and Richmond NHS Foundation Trust.

A 29-year-old male was referred to Endocrinology clinic with a persistently raised Alkaline Phosphatase (ALP) of 153–174 U/L [ref: 30–130] with otherwise normal liver function tests (LFTs), on a background of 9-months of lethargy impacting daily functioning and creativity. His clinical examination was unremarkable. His initial endocrine work-up revealed hypogonadotrophic hypogonadism (calculated free testosterone 0.119 nmol/mL) and significantly raised IGF-1 levels at 74.1 nmol/L [ref: 7.2–26.0 nmol/L].
A subsequent oral Glucose Tolerance Test with Growth Hormone (GH) measurements confirmed GH excess (GH supressed to a nadir of 2.49 ug/L). His prolactin was markedly elevated at 88,041mIU/L [ref: 86–324].
A MRI pituitary showed a large macroadenoma measuring 31.4 x 21.2 x 31.3 mm with left cavernous sinus invasion and optic chiasm contact. His Humphrey visual field test was normal.
He was commenced on cabergoline with no significant biochemical or radiological response. Addition of a somatostatin analogue also yielded no significant effect. A Dotatate PET-CT demonstrated a suspicious lesion near the left fifth rib, raising the possibility of metastatic disease.
He underwent endoscopic transsphenoidal resection of the tumour with left cavernous medial wall resection and clearance of the medial compartment and lateral cavernous sinus anterior to the ICA.
His postoperative histology confirmed a Pit-1 positive mixed somatotroph-lactotroph pituitary neuroendocrine tumour (PitNET); Ki-67 index was low. With the lucent rib lesion, this may be a Metastatic PitNET.
This case highlights the importance of considering endocrine causes in patients presenting with non-specific symptoms such as fatigue; It also highlights that Acromegaly should be considered in the differential diagnosis of isolated elevations in ALP with otherwise normal LFTs (including gamma-glutamyl transferase), which suggests a bony origin for this. Finally this case demonstrates the need for continued efforts/focus on classifying pituitary tumours and better understanding of the prognostic implications of these.

Discussion Questions:
1. Based on your experience, how do Pit-1 positive mixed somatotroph-lactotroph PitNETs or metastatic PitNETs typically present and progress? What patterns or challenges have encountered/observed?
2. Where histological confirmation of metastatic PitNET is unavailable, what criteria or diagnostic strategies do you rely on to differentiate and guide subsequent management?

Isolated ACTH deficiency due to presumed postpartum lymphocytic hypophysitis and consequent empty sella syndrome presenting with severe symptomatic adrenal insufficiency.

D Perera, S Nag, N Skariah.

South Tees Hospitals NHS Foundation Trust.

Introduction
Isolated adrenocorticotropic hormone (ACTH) deficiency is a rare cause of adrenal insufficiency, characterized by low cortisol level and low ACTH. Diagnosis is often delayed due to its insidious presentation, which includes fatigue, weight loss, hypotension, and hypoglycemia.

We report a case of a 36 year old woman who developed isolated ACTH deficiency, likely secondary to silent postpartum lymphocytic hypophysitis and subsequent empty sella syndrome, presenting with profound symptomatic hypocortisolism.

Case Presentation
A 36 year old female presented one year postpartum with severe fatigue that began approximately 3 to 4 months after delivery, accompanied by significant weight loss(20 kg). She breastfed for six months but discontinued this due breast fibroadenomas. She denied headaches, visual symptoms, or any complications during pregnancy and delivery.

On examination, she was thin and pale with no buccal or gingival hyperpigmentation, vitiligo, or goitre. Blood pressure was 118/82 mmHg with no postural drop.

Investigations
Thyroid function in primary care showed subclinical hypothyroidism (TSH 12.58 mIU/L, FT4 10.4 pmol/for) which was treated with levothyroxine without any clinical improvement.

Whole-body cross-sectional imaging was normal.

Serum cortisol was undetectable (<14 nmol/L). ACTH stimulation test showed a flat response (baseline cortisol <14 nmol/L; 60-minute increment 16 nmol/l) confirming adrenal insufficiency.

Serum electrolytes, gonadotropins, prolactin, and estradiol levels were normal. Serum ACTH was undetectable (<2 ng/L), consistent with a diagnosis of isolated ACTH deficiency.

Pituitary MRI demonstrated an incidental right-sided tuberculum sellae meningioma (5 × 9 × 7 mm) and an empty sella with marked thinning of the pituitary gland (<1 mm).

The patient was started on glucocorticoid replacement therapy, which led to marked clinical improvement. Levothyroxine was discontinued, and thyroid function improved spontaneouly indicating that the subclinical hypothyroidism was a consequence of cortisol deficiency.

Points for discussion
Isolated ACTH deficiency often presents with nonspecific symptoms that require a high index of suspicion for timely diagnosis. Early recognition and prompt glucocorticoid replacement are crucial to improving outcomes and preventing potentially life-threatening adrenal crises. This case emphasizes that while postpartum fatigue is frequently attributed to psychosocial stressors related to newborn care, clinicians should remain vigilant for organic causes when symptoms are persistent, severe, or accompanied by unexplained postpartum weight loss.

Preserved gonadal function despite panhypopituitarism.

K Koysombat[1], P Behary[1], E Hatfield[1], M Patel[1], M Gonzalez[1], A Abbara[1,2], N Martin[1,2], F Wernig[1], K Meeran[1,2].

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We present the case of an 18-year-old man who presented with headaches, progressive visual disturbances, photophobia, neck stiffness, and fever. Computed tomography (CT) of the brain revealed obstructive triventricular hydrocephalus caused by synchronous lesions in the pineal and suprasellar regions.

Initial endocrine evaluation demonstrated panhypopituitarism (FT3 2.7pmol/L, FT4 7.1pmol/L, TSH 1.03mU/L, cortisol 162nmol/L, ACTH 7pmol/L, LH <0.1IU/L, FSH <0.1IU/L, IGF-1 17.8nmol/L, prolactin 39.4μg/L). However, despite undetectable gonadotrophin levels, interestingly, his testosterone levels remained normal (testosterone 21.8nmol/L). Testicular markers were also discrepant; with post-pubertal level of anti-Müllerian hormone (AMH) at 26.3pmol/L but a low inhibin B level at 23ng/L.

Although his testosterone level was post-pubertal, clinically he had features of arrested puberty including sparse facial and body hair, testicular volumes of 4–6mL on ultrasound, azoospermia during cryopreservation, and failure to retrieve sperm via percutaneous epididymal sperm aspiration. These biochemical-clinical discrepancies were initially unexplained, particularly in the absence of exogenous testosterone therapy. This prompted measurement of human chorionic gonadotrophin (hCG), a tumour marker for germ cell tumour, which was elevated at 55IU/L.

He was commenced on stress-dose hydrocortisone and levothyroxine replacement in preparation for an endoscopic biopsy of the suprasellar lesion. Histopathological analysis confirmed a diagnosis of primary intracranial germinoma.

The patient was commenced systemic anti-cancer treatment with sequential combination chemotherapy: Etoposide-Cisplatin (EP), followed by Etoposide, Cisplatin, Vincristine, Methotrexate, and Bleomycin (EP-OMB). This led to a decline in serum hCG which was paralleled by a decline in serum testosterone:

Pre-chemotherapy: hCG 55IU/L, Testosterone 21.8nmol/L, LH <0.1IU/L;

Day 7 post-chemotherapy: hCG 8IU/L, Testosterone 2.0nmol/L, LH <0.1IU/L;

Day 14 post-chemotherapy: hCG <2IU/L, Testosterone <0.5nmol/L, LH <0.1IU/L.

Intracranial germinomas are known to be able to secrete the 40-kDa isoform of hCG, albeit at lower concentrations than non-germinomatous germ cell tumours. Given the structural similarity between hCG and LH, hCG can activate LH/hCG receptors, thereby stimulating testosterone production independently of LH. This mechanism likely accounted for the initially normal testosterone level. Following chemotherapy and the normalisation of hCG, the underlying hypogonadotrophic hypogonadism became evident.

At present he is completing his chemotherapy with autologous stem cell transplant and remains on replacement hydrocortisone, levothyroxine and transdermal testosterone.

Discussion points

- What was the mechanism behind the normal testosterone levels despite panhypopituitarism and undetectable LH? →
  hCG bears 85% resemblance to LH structurally and thereby can stimulate Leydig LHCGR to secrete testosterone.

- Why did the testosterone level at presentation—despite being within the post-pubertal range—fail to result in a post-pubertal phenotype? →
  Serum hCG levels in germ cell tumours can fluctuate, in fact pre-operatively his hCG levels measured at Oxford was 9IU/L and testosterone level at that point was 1.3nmol/L hence this could account for the discrepant clinical phenotype compared to the pre-chemotherapy blood results.

- Fertility option in this case. The patient was prepubertal, will need pubertal induction with gonadotrophins in order for spermatogenesis to occur, however was this an option in the context of germ cell tumour? Would this be a possibility post chemotherapy and radiotherapy?

A skull base eroding macroprolactinoma: the dilemma of treating conservatively, medically or surgically.

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Sheffield Teaching Hospital NHS Trust.

Background
Prolactin-secreting tumours account for approximately 30-40% of pituitary adenomas. The vast majority are effectively managed with dopamine agonists, while a minority of cases may require surgery, radiotherapy or purely symptomatic treatment depending on individual clinical circumstances.

Case Presentation
A 28-year-male with autistic spectrum disorder presented with longstanding symptoms of fatiguability, lack of sexual drive and facial hypotrichosis. Biochemical evaluation revealed a prolactin of 144,026 m IU/L, with secondary hypothyroidism and hypo-gonadotrophic hypogonadism with a low testosterone level of 2.8 nmol/L, with an intact pituitary-adrenal axis and a normal IGF-1 of 238 ug/L. These were followed by an MRI pituitary, which showed a 35 x 34 x 26 mm tumour, extending inferiorly through the skull base and the optic chiasm was elevated by the tumour. His scan was carried out prior to the appearance of any compression symptoms, and genetic screening for MEN1 and AIP were negative.

The MDT discussed the dilemma of initiating dopamine agonist therapy, running the risk of precipitating a CSF leak due to the tumour's downward invasive growth, versus early surgical intervention if visual compromise were to develop. Given the absence of visual symptoms, the consensus was to proceed with low dose cabergoline alongside Levothyroxine, following pneumococcal and meningococcal vaccination, with close monitoring for CSF leakage. Testosterone replacement was suggested if a low testosterone persisted during follow-up.

After three months of treatment, prolactin level normalised to 374 mIU. However, testosterone showed only modest improvement, rising from 2.8 to 4.0 nmol/L. Transdermal testosterone replacement was introduced. One month later, a repeat pituitary MRI demonstrated a reduction in tumour size to 12x20x20 mm with no extension into the suprasellar cistern or mass effect on the optic chiasm, with no evidence of skull base erosion or invasion.

Conclusion
Treatment of aggressive macroprolactinomas often requires multidisciplinary consensus, particularly when there is suprasellar extension or skull base invasion or erosion. In selected cases, a pragmatic approach favouring medical management with close monitoring and patient education may be appropriate to avoid the risks associated with surgery.

Question
Would you have taken a different approach to this patient's management?

Aggressive silent corticotroph adenoma with recurrent disease, pituitary apoplexy, and life-threatening complications: a longitudinal case report.

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2. Department of Endocrinology, Queens Hospital Burton, University Hospitals of Derby and Burton. Burton.

Background
Silent corticotroph adenomas (SCAs) are rare non-functioning pituitary tumors that lack clinical features of hypercortisolism but often behave aggressively, with high recurrence rates and resistance to standard therapies.

Case Presentation
We present a 59-year-old male diagnosed in 2017 with a non-functioning pituitary macroadenoma (32 mm) following visual field loss. Imaging showed optic chiasm compression; hormonal workup revealed hypopituitarism. He was started on hydrocortisone, levothyroxine, and testosterone. Transsphenoidal surgery (TSS) led to visual improvement. Histopathology confirmed a type 1 silent corticotroph adenoma (ACTH-positive).

Tumour regrowth occurred in 2019, requiring repeat TSS. Histology again confirmed SCA with high p53 expression and MIB-1 index >3%, indicating elevated recurrence risk. Residual disease prompted conventional radiotherapy in 2020, complicated by pituitary haemorrhage. Growth hormone deficiency was confirmed in 2022 and treated briefly with GH therapy, which was discontinued due to lack of benefit.

Progression continued into 2023–2024 with cavernous sinus invasion, third nerve palsy, and worsening vision. Debulking surgery was performed in July 2024, followed by temozolomide chemotherapy in January 2025. The disease course was complicated by septic shock from necrotising fasciitis, requiring ICU admission, intubation, and lower limb amputation. MRI revealed further pituitary apoplexy. A DVT developed during admission; anticoagulation with rivaroxaban was initiated, later requiring IVC filter placement due to worsening pituitary bleed.

He was re-admitted with worsening symptoms and further radiological progression. Evaluation for additional radiotherapy is ongoing, pending review by a tertiary oncology centre.

Conclusion
This case highlights the aggressive nature of SCAs and the complexity of their management. Recurrent disease, apoplexy, and systemic complications necessitate a multidisciplinary approach. Could earlier identification of high-risk features and integration of novel therapies alter the trajectory of such challenging cases?

Cushing's disease; never an easy ride.

J Lee Siew Hua[1], C Bartlett[1], R Gorrigan[1], H Marcus[2], WM Drake[1].

1.St Bartholomew's Hospital, Barts Health NHS Trust, London.
2.National Hospital For Neurology and Neurosurgery, Queen Square, London.

Abstract
A 15 year old female patient presented to a paediatric emergency department in an acutely confused state with a self-inflicted stab wound to her neck and chest. She had recently undergone attempted trans-sphenoidal surgery on two occasions for Cushing's disease (probable 3mm target lesion, with a marked central to peripheral gradient of ACTH on inferior petrosal sinus sampling) but without clinical or biochemical remission.

She was admitted to Paediatric Critical Care where an IV etomidate and IV hydrocortisone infusion as part of a 'block and replace' regimen was commenced. This admission was further complicated by the development of sepsis secondary to cellulitis. On account of the severity of the situation and a need for total resolution of Cushing's, a bilateral adrenalectomy was performed.

She was discharged from hospital on hydrocortisone and fludrocortisone. Post bilateral adrenalectomy, 9am cortisol levels were<20nmol/L; indicating that the adrenalectomy was complete. A year later, MRI scan showed the right sided lesion measured 7mm. Her treating paediatric team referred her for external beam pituitary radiotherapy (45Gy in 25#). She was referred for adult endocrine follow-up.

By age 22, she started to notice a persistent tan after a holiday to Turkey; ACTH levels post-hydrocortisone were 114ng/L (reference range<50). Her pigmentation became progressively more noticeable over 1-2 years, with ACTH values >2000ng/L in association with enlargement of the pituitary lesion to a maximum of 17mm, with radiological evidence of contact with and elevation of the right optic nerve and indentation into the right cavernous sinus. She recently underwent further trans-sphenoidal surgery and awaits further detailed biochemical evaluation.

This case illustrates that Cushing's syndrome can occasionally cause psychiatric emergencies; and highlights the need for regular monitoring of ACTH levels in those patients with Cushing's disease not cured by pituitary surgery who require bilateral adrenalectomy.

Challenges of a rapidly progressive pituitary metastasis – surgery, AVP deficiency, chemotherapy and palliative care.

AW Paracha, M Levy, S Gohil, N Reddy.

Leicester Royal Infirmary, University Hospitals Of Leicester NHS Trust.

Background
Pituitary metastasis, although rare, is a life-threatening condition often presenting with visual disturbances, hypopituitarism and AVP deficiency. The most common primary sites are breast and lung carcinomas. The prognosis is poor due to late diagnosis and the aggressive nature of metastases. We highlight such a case of non-small cell lung cancer (NSCLC) with pituitary metastasis with rapid progression, management challenges and unfavourable outcome.

Case Presentation
A 52-year-old male, ex-smoker, presented with headaches, visual disturbances and profound fatigue. Biochemistry confirmed hypopituitarism: Na 144mmol/L, LH <0.5iu/L, FSH <0.5iu/L, Testosterone <0.3nmol/L Cortisol 99nmol/L, IGF1 143ng/mL, Growth hormone 0.9ug/L, TSH 0.06mIU/L, fT4 7.4pmol/L, Prolactin 74mIU/L. Pituitary MRI revealed a heterogeneous enhancing intra and suprasellar mass, measuring 17x 16.6 x 16.4mm compressing the optic chiasm and consistent with a pituitary macroadenoma. Hormone replacement with hydrocortisone, thyroxine and testosterone was commenced.

Within two weeks, he re-presented with worsening headaches, confusion, polydipsia and polyuria and was subsequently started on desmopressin. Repeat pituitary MRI revealed rapid interval growth. With the suspicion of malignancy, CT CAP revealed a 5 cm lung mass, later histologically confirmed as NSCLC. An interval pituitary MRI 2 weeks later demonstrated further pituitary metastatic growth with developing hydrocephalus. He was discussed in the pituitary and lung MDTs and the pros and cons of debulking surgery were discussed with the patient and his partner who wished to proceed despite the risks, with the small hope of post-op chemotherapy if successful. Orbitozygomatic craniotomy with EVD and second surgery was needed. Post op, he was confused, and sodium/AVP-D management was difficult with hyper- and hyponatraemia and a ‘smart catheter’ was used to help with monitoring fluid balance. Unfortunately, he did not recover enough for chemotherapy and palliative care supported his terminal period. Despite his prolonged inpatient stay, his partner was thankful for the extra time surgery gave, as he would have died much sooner without surgery.

Discussion
This case highlights the aggressive course of NSCLC-related pituitary metastasis, the dilemma of whether to operate or not (question for the audience), difficult AVP-D management, use of a ‘smart catheter’ and the palliative care of a patient with pan-hypopituitarism.

Hyponatremia in AVP deficiency during prolonged childbirth: the double-edged sword of desmopressin in prolonged labour.

L Eltayieb, E Shakoor, M Dram, J Aikpitanyi, O Oluwaseun, F Hussein, H Wong, S Mukadam, A Mota, A Abdullah, M Ashwini, M Muhammad, B Hossain, G Simon, P Sivarajasinga, E Phillips, F Fahal, G Mlawa.

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Introduction
AVP deficiency can be challenging during pregnancy due to the physiological changes associated with gestation, which can impact both the mother and the baby. Pregnancy induces the production of vasopressinase enzyme from the placenta, which degrades vasopressin. In women with AVP deficiency, vasopressinase can exacerbate the condition during pregnancy, leading to worsening polydipsia and polyuria and therefore necessitating desmopressin dose adjustments and close monitoring.

Case Summary
A 38-year-old female with a known diagnosis of AVP deficiency secondary to hypophysitis. Her medication history includes desmopressin nasal spray 20mcg twice daily. She presented to the hospital on her estimated date of delivery and was referred to endocrinology due to polyuria and polydipsia likely due to increased placental vasopressinase activity. She was admitted in active labour and developed significant hyponatremia (serum sodium 122mmol/L, serum osmolality 270mOsm/kg, urine osmolality 630mOsm/kg), while on desmopressin therapy (20mcg BD). Desmopressin was withheld during labour to prevent worsening hyponatremia. Fluid input-output and sodium levels were strictly monitored.

Clinical Challenge
She was in a prolonged labour and the challenge was a risk of exacerbating hyponatremia if oxytocin was used for induction/augmentation of labour, as oxytocin has an antidiuretic effect similar to vasopressin.
She was admitted to HDU due to the risk of physiological production of oxytocin which can further exacerbate the hyponatremia. Plans were made for hypertonic saline administration if sodium levels worsened in case oxytocin was administered for induction of labour.

Outcome and Follow-Up
The patient delivered successfully; however, the baby was found to be hyponatraemia mirroring maternal sodium levels and was therefore admitted to NICU.

Case discussion and Conclusion
This case highlights the delicate balance between desmopressin therapy, fluid management and physiological changes in active labour. The interplay of placental vasopressinase, desmopressin, and oxytocin created significant clinical dilemmas in ensuring maternal and neonatal safety. It illustrates the need for multidisciplinary for close monitoring and management.

Questions for Discussion
Have you managed patients with AVP deficiency during labour, and how did you approach it? How do you safely manage desmopressin and balance fluids in a prolonged labour in woman with AVP deficiency, especially when oxytocin is needed?

Pituitary abscess: a rare cause of pituitary mass.

M Blair, M Freel, S Hassan.

Queen Elizabeth University Hospital, NHS Greater Glasgow and Clyde.

Abstract
Pituitary abscess is a rare condition and often presents non-specifically. In this case we discuss the presentation of a pituitary abscess diagnosed intra-operatively and subsequent endocrine complications.

A 25 year old man presented with three months history of visual disturbance, headache and fatigue. Medical history included autism, asthma and Tourette's syndrome. He was a farmer and usually fit and well. CT imaging was arranged by his GP in view of persistent visual symptoms. This identified a pituitary mass with compression of the optic chiasm and he was referred to Neurosurgery.

Investigations demonstrated hypopituitarism (cortisol 155nmol/L, testosterone 4.1nmol/L, free T4 4.7pmol/L) and the patient was commenced on hydrocortisone. Marginal contrast enhancement was noted on MRI and trans-sphenoidal surgery performed due to concern of apoplexy. Intra-operatively, a cystic lesion was visualised with the normal pituitary gland and stalk displaced posteriorly. Yellow viscous fluid was drained. Nine days later a second trans-sphenoidal surgery was performed due to concern regarding persisting collection on repeat MRI. Fluid samples grew staphylococcus aureus and finegoldia magna. Under the care of the Infectious Diseases team, the patient completed a six week course of linezolid, with the final weeks managed via the outpatient antibiotic service.

This patient developed the triphasic response following his pituitary surgery; within 24 hours of surgery he developed polyuria with a low urine osmolality (108mmol/kg) and rapid rise in serum sodium from 135 to 143mmol/L. At day nine post-surgery hyponatraemia developed (serum sodium 122mmol/L, urine sodium 124mmol/L, urine osmolality 926mmol/kg). Post-discharge testing six weeks later confirmed a diagnosis of arginine vasopressin (AVP) deficiency with ongoing polyuria, low urine osmolality and raised serum osmolality and desmopressin was commenced. The patient remains clinically well, continuing hydrocortisone with levothyroxine and testosterone replacement added after follow-up testing.

This case highlights the non-specific presentation of pituitary abscess, the importance of considering this as a differential diagnosis in an expanding pituitary mass and the value of multidisciplinary working to achieve optimum outcomes in this rare presentation.

Questions
Should this diagnosis have been suspected earlier in the clinical course and were there any findings which could have prompted earlier diagnosis?

Poster Presentation Abstracts
(view posters at https://www.imperialendo.co.uk/pit2025.htm)

When snoring means more: severe obstructive sleep apnoea as first clinical presentation of an endocrine disorder

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Introduction
Acromegaly is a rare chronic disorder caused by excessive growth hormone (GH) secretion, most commonly due to a pituitary adenoma (>95% of cases). Diagnosis typically occurs between 40 and 50 years of age. [1] Clinical manifestations often include acral enlargement, facial changes, and systemic complications. [2]

Case Presentation:
A 53-year-old man presented to the ENT clinic with dysphagia, difficulty breathing, excessive snoring at night and a blocking sensation in his throat, following a referral from his general practitioner. Examination revealed a left tonsillar mass extending to the base of the tongue and superiorly to the soft palate.
His past medical history included hypertension, back pain, and carpal tunnel syndrome. He was referred to the Endocrinology team by ENT due to acromegalic features. He reported a history of increased hand and shoe size over the past 13 years.
He later presented acutely with shortness of breath and was admitted to the respiratory ward, where he was treated for obstructive sleep apnoea (OSA).
Blood tests revealed elevated IGF-1 levels (824). He was started on lanreotide, initially 90 mg, later increased to 120 mg. His breathing has since improved, and he is scheduled for transsphenoidal surgery in the next three months as definitive treatment.

Conclusion: This case highlights the importance of recognising features of acromegaly, in the patients who present with upper airway obstruction. Obstructive sleep apnoea, which commonly presents with snoring, should be thoroughly investigated to identify potential underlying causes. In this case, the presence of OSA symptoms prompted further evaluation, ultimately leading to the diagnosis of acromegaly. As a result, Early identification and multidisciplinary management including ENT, endocrinology, and respiratory teams is important. The medical therapy with lanreotide provided symptomatic improvement, while transsphenoidal surgery remains the definitive treatment for his underlying pituitary pathology.

Question: Have you come across OSA or breathing difficulty as first presentation of Acromegaly?

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Unusual presentations of AVP deficiency presenting as cognitive impairment and renal function impairment

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Introduction
Cranial diabetes insipidus (DI) known as AVP deficiency is a rare disorder caused by impaired secretion in arginine vasopressin (AVP) in central DI or response in nephrogenic DI. Affects 1 in 25,000 people and usually presents with polyuria and polydipsia. The most common acquired causes include autoimmune, infection, primary or secondary tumours, neurosurgery and head trauma.
Here we discuss two cases in which AVP deficiency presented with cognitive and renal function impairment.

Case 1
31year-old female admitted with reduce oral intake, dehydration, cognitive impairment, and acute kidney injury (AKI). Past medical history TB meningitis which led to stroke. Blood test showed hypernatraemia 174mmol/L and MRI showed increase in size of lesion in left hypothalamus and midbrain with further new lesions. Plasma glucose 33.6 mmol/L suggesting new onset of Type 2 diabetes. Endocrine input was required as serum osmolality remained high despite treatment of hyperglycaemia. Serum osmolality was 314mosmol/kg, urine osmolality 481mosmol/kg. She was started on DDAVP which led to clinical improvement and normalised serum osmolality.
She was treated with quadruple anti-tuberculous medication for 14months and required second-line anti-tuberculous medication.

Case 2
54year-old male initially presented to different hospital with cognitive impairment, memory issues alongside worsening headaches. CT showed Chordoid Glioma of third ventricle requiring VP shunt, debulking of tumour and radiotherapy.
He was reviewed in endocrine clinic for low testosterone on background of third ventricle pathology and found to have high osmolality and was admitted to hospital for water deprivation test. He was diagnosed with AVP deficiency and full pituitary profile revealed partial hypopituitarism secondary to his third ventricle tumour.
He was treated with hydrocortisone, desmopressin, levothyroxine, testosterone.

Conclusion:
Here we discuss two cases in which AVP deficiency initially presented as cognitive impairment due to severe dehydration and AKI thought to be secondary to hypothalamic or brain injury. It is important that a multidisciplinary team approach is used for individualised management of these patients to achieve the best outcome.

Question:
1. Have come across unusual presentation of AVP deficiency?
2. Have you seen cognitive or renal impairment as initial presentation of AVP deficiency in your patients?

Long road to diagnosis: partial hypopituitarism and AVP deficiency in the context of a very rare multisystem disorder, Erdheim-Chester Disease.

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2. King's College Hospital

52-year-old male patient known to our service since 2003 for Type 1 diabetes developed polydipsia and polyuria in 2008. Investigations including water deprivation test confirmed AVP deficiency and Desmopressin was commenced.
He had partial hypopituitarism with hypogonadotropic hypogonadism. Contrast pituitary MRI in 2010 revealed small enhancing mass expanding the pituitary stalk. Differentials following discussion at St George's Pituitary MDT: Sarcoidosis, Germinoma or Langerhans cell histiocytosis. Biopsy was recommended, however patient not keen and the lesion was waxing and waning. He started Testosterone replacement therapy. HRCT in 2011 excludes
pulmonary sarcoidosis, serum ACE and protein electrophoresis are unremarkable. He was under regular endocrine follow up with surveillance scans.
Retrospective review of notes show complains of bilateral leg pain, muscle weakness, feeling generally unwell and tired.
He developed secondary hypothyroidism in 2016 with normal cortisol axis and was started Levothyroxine. Further imaging in 2018 shows enlarging of the pituitary lesion. Care taken over by different Consultant and further investigations were performed; normal repeat serum ACE, protein electrophoresis, AFP, hCG and PSA. Whole body PET CT was completed showing small focus within sella turcica but no other suspicious FDG avid lesions. Following re-discussion in Pituitary MDT patient agrees to proceed with biopsy, which was inconclusive. Post surgery he developed secondary hypoadrenalism and was commenced on replacement.
He continued to complain of significant lumbo-sacral pain radiating to legs, stiffness and intermittent claudication. He was referred to Rheumatology. Following MRI spine, local MDT discussion picked up sclerotic bony lesions, these were also seen on historic imaging. He was referred to Stanmore for tissue diagnosis, which was difficult due to sclerosis and non-diagnostic. CT CAP shows perirenal inflammatory changes and occluded right common iliac artery. Following multiple MDT reviews, Erdheim-Chester Disease was suspected, and he was referred to Haematology Specialist Centre for treatment.
Patient is currently on Trametenib since 2022 with significant improvement in symptoms resulting in much better quality of life and some improvements in MRI pituitary and endocrine function.

Discussion:
1. Any clues to diagnosis earlier?
2. Value of MDT involvement and Specialist Centres
3. Multiple symptoms - think of unifying diagnosis

Treatment and management of hypopituitarism on background of pituitary metastasis of multiple myeloma.

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Introduction
Pituitary metastasis in haematological malignancies is rare, though incidence is rising with increasing cancer prevalence[1]. A 2014 review reported only 31 cases to date[2]. Approximately 7% of pituitary metastases are symptomatic; most are identified incidentally at post-mortem[1].
Clinical Case
A 65-year-old man with a high-risk IgG lambda multiple myeloma presented with headache and ophthalmoplegia. MRI pituitary showed a clival mass encasing the pituitary stalk.
SIADH was confirmed with serum sodium 129 mmol/L, serum Osm 265 mOsm/kg, urine Osm 654 mmol/L, and urine Na of 99 mmol/L. Testosterone 0.7 nmol/l, FSH was 4.2 IU/L and LH was 4.1 IU/L. TSH 0.48 mIU/L, FT4 11.4 pmol/L, prolactin 567 mIU/L and IGF-1 was 43.6 nmol/L. Cortisol was 352 nmol/L consistent with partial secondary hypopituitarism. High prolactin and IGF-1 likely non-specific in the context of malignancy.
Urgent Pituitary MDT recommended skull base radiotherapy for pituitary plasmacytoma. The patient was commenced on levothyroxine 100 mcg, testosterone gel, and dexamethasone 1 mg in between chemotherapy cycles where higher doses were used.
TSH rose to 8.17mIU/L with T4 10.3 pmol/l, 3-months following radiotherapy, indicating thyroxine non-compliance though further recovery of pituitary-axis.
Radiotherapy improved pituitary function: testosterone rose to 7.3 nmol/L at 5 months, normalised to 20.4nmol/L, and gel was stopped. Levels remained stable, (13.2 nmol/L), one year later.
At 12 months, patient was admitted with vomiting. Cortisol was 13 nmol/L. It was identified that he was non-compliant with steroid-replacement, possibly attributed to radiotherapy-related cognitive impairment. Endocrine nurse support was arranged to improve compliance with hormone replacement.
At 21 months post-radiotherapy, he remained independent with good quality of life and treatment compliance. CT-PET showed significant improvement in skeletal and extramedullary disease. Diplopia improved, though persistent visual-deficit required ongoing Ophthalmology follow-up.

Conclusion
This case underscores the high index of suspicion in myeloma patients presenting with headache or features of hypopituitarism requiring early diagnostic pituitary evaluation. MDT endocrinology input and regular follow-up are critical to guide and support haemato-oncology therapies. Heightened awareness among non-endocrine specialists and allied oncology healthcare professionals is essential.

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Pituitary apoplexy of an undiagnosed macroprolactinoma: to operate or not to operate?

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Abstract
A 28-year-old man presented to eye casualty with reduced vision in the right eye following a self-limiting headache 5 days previously. He had prior migraines with a similar episode 1 year ago. He recently noticed reduced libido and erectile dysfunction but no galactorrhoea.
Examination demonstrated significantly reduced right sided visual acuity (6/24) and colour vision (Ishihara 1/15). There was no ophthalmoplegia, or field defect on confrontation testing. An urgent outpatient MRI head and orbits was organised.
MRI showed a 38x28x30mm mixed solid and cystic pituitary lesion, with graduation of products suggesting subacute haemorrhage. The optic chiasm was compressed, particularly the right side, but with no T2 signal change in the anterior visual pathway. Biochemistry showed hyperprolactinaemia 29557mU/l (70-400); secondary hypothyroidism with thyroid stimulating hormone 1.87mU/l (0.3-4.20), T4 6.8pmol/l (9.0-23.0); hypogonadotrophic hypogonadism with testosterone 1.2nmol/l (10.0-30.0), luteinising hormone 1.0units/l (2-12); remaining pituitary function was normal.
The MRI results prompted admission, one week after initial presentation, by which time right sided acuity had further deteriorated to 6/60. He therefore underwent emergency trans-sphenoidal resection. His post-operative course was uneventful. Histology demonstrated a mixed lactotroph/somatotroph adenoma. Ki67 measured up to 7%, although this may be reactive to apoplexy.
At 3-month follow-up he felt his vision had normalised but described persistent loss of early morning erections. He had ongoing mildly elevated prolactin levels (804mU/l) and low fasted early morning testosterone (5.0nmol/l). MRI at 6 months showed no clear residual. Prolactin remains stable. He is being monitored with serial biochemistry and MRI planned at 1 year, with consideration of cabergoline if required. He is currently on no pituitary hormone replacement, and serum testosterone is improving.
This case demonstrates an atypical presentation of apoplexy with isolated visual loss and self-resolved headaches. It also demonstrates the challenges in deciding on surgical or medical management of pituitary apoplexy, particularly undiagnosed prolactinomas. However, in this case rapidly deteriorating visual acuity prompted emergency decompression, with subsequent excellent outcome and restoration of vision.

Questions for discussion:
1) Would you have proceeded with surgery or dopamine agonists at presentation?
2) Would you now pursue a watch-and-wait approach or treatment with cabergoline, or testosterone?

When sight and life collide.

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Abstract
A 19-year-old female with the BG of ADHD presented to A&E at 28 weeks of gestation presented with acute visual deterioration, prompting urgent ophthalmology referral. Examination revealed bitemporal hemianopia, left temporal optic disc pallor, complete loss of colour vision in the left eye, and mild colour vision reduction in the right eye. MRI orbit revealed a large pituitary macroadenoma causing optic chiasm compression and potential left anterior optic atrophy, without evidence of apoplexy.

Initial endocrine assessment was largely normal except for a raised prolactin believed to be consistent with pregnancy. (Prolactin 2399 mIU/L(109- 557), 9 AM Cortisol 388 nmol/L (102-535),ACTH 25(< 50), Free T4 12.9 pmol/L (9-19.1), TSH 1.20 mIU/L(0.5- 4.9), IGF-1 23.8 nmol/L(13.3- 59.9), LH (1.8-11.8), FSH < 0.1 (2.6-16.6). She presented again in A&E with reduced foetal movements and subsequently diagnosed with IUD. She becomes symptomatic as she developed dizzy episodes and worsening of visual symptoms, and urgent discussion with Neurosurgeon advised to transfer the patient immediately. She underwent transsphenoidal surgery (TSS). Histopathology confirmed a non-functioning gonadotroph adenoma.

Post-operatively, she developed diabetes insipidus on day 2 as she was passing 300-700 ml of urine per hour and was started on desmopressin and hydrocortisone. Follow-up MRI demonstrated residual anterior pituitary tissue extending into the suprasellar cistern, causing elevation of the peri-chiasmatic optic nerve. Ophthalmological follow-up showed normal colour vision in the right eye with mild improvement on the left, though the patient had persistent visual field defects with left temporal hemianopia consistent with optic neuropathy

This case highlights rare presentation of gonadotroph PitNET in pregnancy with optic neuropathy and IUD and underscores the importance of timely imaging, endocrine assessment, and surgical intervention.

Questions
Could earlier recognition of visual field loss and adenoma have altered the pregnancy outcome in this case?
What mechanism link pituitary adenoma in pregnancy with intrauterine demise, and how should this influence our diagnostic and management strategies?

A tired young man with enuresis nocturna.

PT Serrano, G Shelby, S Muquit, D Flanagan, I Dimitropoulos.

University Hospitals Plymouth.

Abstract
A 15-year-11-month-old boy presented with fatigue and poor growth compared to his peers and siblings, he had a history of nocturnal enuresis since age seven, managed with desmopressin. He also reported recent persistent polyuria and polydipsia, with a daily fluid intake of 2.5–3 liters with a body weight of 47.5 kg (~53–63 mL/kg/day). Biochemical evaluation demonstrated secondary hypothyroidism, hypocortisolism, and hypogonadism. MRI revealed a heterogeneous sellar mass, most likely arising from the posterior pituitary.

Following MDT discussion, the patient underwent endoscopic transsphenoidal resection. Histopathology was consistent with a benign cyst, most likely a Rathke's cleft cyst with xanthogranulomatous reaction or a sellar xanthogranuloma, with no evidence of malignancy. Post-operatively, he requires hormone replacement including hydrocortisone, levothyroxine, growth hormone, desmopressin, and testosterone. At one-year follow-up, serial MRI scans demonstrated no recurrence. Clinically, he achieved catch-up growth and pubertal progression while on regular endocrine replacement therapy. This case highlights the diagnostic challenges of paediatric pituitary masses and emphasizes the importance of early recognition, surgical intervention, and multi-disciplinary management.

In the shadows of cyclic Cushing's: an unlocalized ACTH source and fatal infectious complication.

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Introduction
Cyclic Cushing's syndrome (CS), once thought rare, is now recognized as more common and is characterized by intermittent hypercortisolism alternating with periods of normal cortisol secretion. Cycle duration varies from days to years, and diagnosis requires at least three cortisol peaks and two troughs. A recent meta-analysis (Nowak et al., 2023) identified pituitary tumours as the leading cause (67%), followed by ectopic (17%) and adrenal tumours (11%). Bilateral inferior petrosal sinus sampling (BIPSS) during hypercortisolaemia showed high accuracy in differentiating pituitary from ectopic sources, while testing outside active phases reduced diagnostic yield. Misclassification led to unnecessary surgery in 6% of cases. Compared to non-cyclic CS, patients with cyclic CS had lower remission rates and longer time to remission. Fluctuating cortisol levels contribute to diagnostic delays and misdiagnosis.

Case presentation
A 50-year-old woman, a former bodybuilder and model with no history of anabolic steroid use, presented with severe psychiatric symptoms culminating in self-harm. Initial investigations revealed markedly elevated serum cortisol (1656 nmol/L), ACTH (200 pmol/L), profound hypokalaemia (2.4 mmol/L), and urinary cortisol >1000 nmol/L, consistent with ACTH-dependent CS. Extensive imaging, including pituitary MRI, chest/abdominal CT, FDG-PET, octreotide, and 68-Ga PET scans, failed to localize the ACTH source. She responded well to metyrapone, with resolution of psychiatric symptoms and physical improvement. During clinical remission, BIPSS was attempted but proved inconclusive as she had cycled out. Metyrapone was gradually stopped, and monitoring resumed with twice-weekly late-night salivary cortisol and weekly overnight dexamethasone suppression tests. Recurrence of cyclical Cushing's occurred six months later, presenting with severe klebsiella pneumonia and marked hypercortisolaemia (4577 nmol/L). Despite intensive care, antimicrobial therapy, and etomidate infusion for cortisol control, she succumbed to sepsis. In cyclical ACTH-dependent Cushing's with occult source and inconclusive BIPSS, what could have been done differently to alter the outcome?

Discussion
This case highlights the complexity of diagnosing and managing cyclic CS. The unpredictable cortisol fluctuations complicate localization and delay treatment. Prolonged biochemical monitoring and timely invasive investigations during active phases are crucial. Missed diagnostic windows may increase morbidity and mortality, emphasizing the need for close clinical surveillance and early intervention.

Conservative management of pituitary apoplexy in a young woman with non-functioning pituitary adenoma.

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2. University Hospitals of Derby and Burton NHS Trust.

Background
Pituitary apoplexy is an uncommon but potentially life-threatening endocrine emergency. While surgery is often considered, especially in the presence of visual compromise, conservative management may be appropriate in selected cases. We present a young woman with pituitary apoplexy and a non-functioning pituitary adenoma who was successfully managed without surgical intervention.

Case Presentation
A 25-year-old woman with a history of hyperprolactinaemia (previously on cabergoline) presented in March 2019 with acute headache and transient right-sided weakness. MRI brain demonstrated a 9.5 mm pituitary microadenoma with haemorrhage consistent with pituitary apoplexy. Visual fields revealed a bitemporal hemianopia. She was commenced on intravenous hydrocortisone. Following neurosurgical review, a decision was made for conservative management as there was no evidence of chiasmal compression and her neurology was stable.

Serial MRIs demonstrated a reduction in adenoma size to 7 mm by mid-2019 and further regression to 3.5 mm by 2024. The patient developed transient ACTH deficiency requiring steroid replacement, but this resolved within months. Other pituitary axes remained intact. She experienced recurrent headaches and visual symptoms, ultimately attributed to chronic migraine, which improved with specialist management.

During her pregnancy in 2021–22, she was monitored jointly by endocrinology and ophthalmology. Despite worsening headaches, there was no radiological progression or new field defects, and she delivered safely without complication. At six years' follow-up, her adenoma has continued to regress, pituitary function remains normal, and she is well, with migraines managed separately.

Discussion
This case demonstrates that conservative management of pituitary apoplexy can be safe and effective in carefully selected patients. Surgery may not be necessary where visual compromise is mild or improving, there is no chiasmal compression, and endocrine deficits are manageable. Long-term outcomes can be favourable, including radiological regression and recovery of pituitary function.

Learning Point
Not all pituitary adenomas or cases of pituitary apoplexy require surgery. Conservative management with multidisciplinary follow-up can achieve excellent outcomes while avoiding the risks of operative intervention.

Tamoxifen as adjunctive therapy in a male with dopamine agonist–resistant macroprolactinoma.

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Background
Dopamine agonists (DAs) achieve remission in most prolactinomas, but a subset remains resistant despite maximal therapy. Invasive tumours further limit surgical options. Alternative pharmacological strategies are rarely reported.

Case
A 35-year-old man presented in 2020 with a macroprolactinoma (serum prolactin 28,056 mIU/L; ref 60–300). MRI revealed a 16 mm left-sided adenoma encasing the cavernous carotid, precluding curative surgery. Cabergoline was titrated cautiously due to a background of anxiety, reaching 6 mg/week by September 2022, achieving near-normalisation of prolactin and reduction in tumour size. Prolactin fell from >28,000 to 580 mIU/L (Jan 2023), but tumour volume remained stable (9 mm, unchanged 2022–2024). Serum testosterone was low-normal at 12.8 nmol/L (ref 10–30).

Tamoxifen 20 mg daily was introduced in January 2023 for gynaecomastia. This improved biochemical control, with prolactin 286–313 mIU/L (2023–24; ref 70–400) and testosterone stabilising mid-normal (18–23 nmol/L), alongside improved libido and muscle bulk. After stopping in July 2023, prolactin levels rose (567 mIU/L in October 2023) with testosterone levels declining (12.8). Rechallenge with tamoxifen (20 mg four times weekly from Oct 2023, then weekly from July 2024) again improved control (prolactin 287 mIU/L, testosterone 18.8). A further withdrawal in Feb 2025 led to prolactin rebound (495 mIU/L) and reduced testosterone (14.6) by Aug 2025. He has since resumed once-weekly 20 mg tamoxifen. The patient continues cabergoline 6 mg/week; dose reduction has not yet been attempted, but remains a goal, particularly given his anxiety and supramaximal cabergoline requirement.

Discussion
This case demonstrates tamoxifen's potential utility in DA-resistant prolactinomas, with biochemical and clinical benefit despite absent tumour shrinkage. The likely mechanism is antagonism of oestrogen receptor β in lactotrophs. Clinical use remains limited by the absence of long-term outcome data and thromboembolic risk.

Question
Can selective oestrogen receptor modulators provide a safe, effective adjunct in DA-resistant prolactinomas, and should they be prospectively evaluated to permit cabergoline dose reduction?

Protecting vision to support British Sign Language in a deaf patient with a progressive pituitary adenoma: a challenging case.

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3. Department of Clinical Oncology, Imperial College Healthcare NHS Trust.
4. Department of Radiology, Imperial College Healthcare NHS Trust.

Abstract
We present a 59 year old man, with a background history of complete sensorineural hearing loss and a learning disability, who can only communicate with British Sign Language (BSL). He underwent trans-sphenoidal surgery for a large pituitary tumour in another UK centre in 2010 with subsequent secondary hypothyroidism, hypogonadism and hypoadrenalism. He became blind in his left eye due to both the compressive effects of the tumour and also previous challenging glaucoma management leading to left optic nerve atrophy.

He was lost to follow up for several years but then presented to a local hospital and was referred to our Pituitary Multidisciplinary Team MDT for discussion in 2022. The residual pituitary tumour had slowly grown over the past five years, with sellar and suprasellar extension. This raised concerns about protecting his vision in the contralateral (right) eye, given that his remaining vision is essential to enable him to communicate via BSL. The decision was taken to perform further trans-sphenoidal pituitary surgery in 2023. This surgery was fortunately uncomplicated, and histology confirmed gonadotroph pituitary adenoma with a low proliferation index.

At a recent Pituitary MDT review, two years after his last surgery, imaging showed that the solid component of the residual tumour has increased in size indicating mild interval growth with difficulties viewing the optic chiasm. Currently, vision in his right eye is uncompromised. Following MDT discussion, further surgery was not recommended in view of two previous surgeries and high risk of cerebrospinal fluid leak. Also, there were some concerns about whether elective radiotherapy was in the patient's best interests currently, particularly regarding challenges communicating the requirements and risks of radiotherapy and the difficulties he may experience completing radiotherapy. However, this needs to be balanced against the risk of deterioration of his vision over time, due to further tumour growth, with the consequences of this on his ability to communicate.

Discussion
Are there any alternative options to consider for this patient to try and stop tumour growth?

Does anyone have any experience of delivering fractionated radiotherapy in a patient like this with challenges around communicating the risks and benefits of radiotherapy?

Incidental pituitary macroadenoma with biochemically active acromegaly discovered during ENT investigation.

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1. St Mary's Hospital, Imperial College Healthcare NHS Trust.
2. West Middlesex University Hospital, Chelsea and Westminster Hospital NHS Foundation Trust.

Background
Incidental pituitary macroadenomas are increasingly detected on cross-sectional imaging. Accurate interpretation of hormonal investigations, including dynamic tests, is crucial to avoid misdiagnosis and guide appropriate management.

Case Presentation
A 70-year-old man underwent MRI of the sinuses for chronic rhinosinusitis, which incidentally revealed a pituitary macroadenoma without optic chiasm compression but abutting the right cavernous sinus. Past medical history included atrial flutter (post-ablation), asthma, and non-diabetic hyperglycaemia. Morning pituitary profile demonstrated elevated IGF-1 (105.9 ug/L; RR 3.5-32) and GH (41.50 ug/L - elevated), while the rest of the pituitary profile was normal.

Following review in endocrinology clinic, he reported no classical symptoms of growth hormone (GH) excess. Examination showed coarse facial features and a hoarse voice, which he stated was longstanding. No other stigmata of hormone excess were present.

An oral glucose tolerance test (OGTT) was performed: GH failed to suppress adequately, with a single low value considered spurious by multidisciplinary team (MDT) consensus. The biochemical profile was consistent with active acromegaly.

Management and Outcome
The patient was referred for ENT management of rhinosinusitis and neurosurgical evaluation prior to transsphenoidal resection. Lanreotide 60 mg every 4 weeks was initiated. He awaits formal visual field assessment and neurosurgical review.

Conclusion
This case emphasises the importance of interpreting dynamic endocrine tests in the context of clinical features and imaging findings, recognising potential spurious results to ensure accurate diagnosis and timely management of functional pituitary adenomas.

Question
How would you interpret the one-off low GH result at the OGTT?

Difficulty identifying clinical signs of Cushing's disease with concomitant glucagon-like peptide-1 receptor (GLP-1) agonist use.

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1. St Mary's Hospital, Imperial College Healthcare NHS Trust.
2. West Middlesex University Hospital, Chelsea and Westminster Hospital NHS Foundation Trust.

Background
Cushing's disease typically presents with a constellation of symptoms associated with hypercortisolism, including weight gain, hypertension and diabetes. Diagnosis may be challenging with concomitant use of weight loss medications, potentially masking the classical features of Cushing's disease.

Case Presentation
A 53-year-old menopausal female with a background of asthma presented with weight gain and a large neck lipoma. Her medications included hormone replacement therapy, Symbicort and prednisolone during asthma exacerbations.

She was prescribed GLP1- agonist for weight loss by her endocrinogist. Her initial weight was 105kg, and with semaglutide therapy, she achieved a weight of 85kg and continuing to lose weight.

Investigations
9am Cortisol 326 nmol/L, ACTH 53.0 ng/L (RR<30)
ONDST: cortisol 304 nmol/L, ACTH not done
Midnight Salivary Cortisol: 3.8 nmol/L & 4.0 nmol/L (RR <2.6)
Midnight Salivary Cortisone: 28.8 nmol/L & 30.6 nmol/L (RR <18)
LDDST: 48-hour cortisol 207 nmol/L. ACTH not done
HDDST - ACTH suppressed to 14 ng/l
24-hour urine cortisol - 113 & 206 nmol/day (RR 0-164)
HbA1c 33 mmol/mol (RR <48)

MRI pituitary - 5 mm cystic pituitary microadenoma

These findings suggest centrally driven hypercortisolemia. However, the patient did not exhibit classic clinical features of Cushing's disease, including hypertension or hyperglycemia (HbA1c 33mol/mol), likely secondary to ongoing semaglutide use.

Management and Outcome
The case was discussed at the pituitary multidisciplinary team meeting with the impression of Cushing's disease and given the presence of a target lesion that could be amenable to surgical intervention, the patient will undergo inferior petrosal sinus sampling prior to a neurosurgical consultation.

Conclusion
This case displays the diagnostic challenges in considering potential Cushing's disease when the only presenting feature is obesity, which is being effectively managed with GLP-1 agonist therapy. Given the increasing use of GLP-1 agonist, clinicians should remain vigilant in considering Cushing's disease even when classical features are absent.

Questions
As the use of GLP-1 agonists increases, will this result in missed diagnosis of Cushing's disease due to lack of classical Cushing clinical features?
If so, how can we address this if the only presenting feature is obesity? Should all obese patients undergo baseline ONDST?

When thirst fails: managing arginine vasopressin deficiency in the absence of osmoregulatory drive.

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Case Presentation
A 20-year-old female presented with secondary amenorrhea and right-sided inferior temporal quadrantanopia. MRI pituitary revealed a 27mm cystic, rim-enhancing sellar/suprasellar lesion compressing the optic chiasm and hypothalamus which was in keeping with a craniopharyngioma. She underwent elective neuronavigation-guided endoscopic endonasal extended trans-sphenoidal surgery (TSS) on June 12, 2025 achieving near-total resection, leaving a small portion of the tumour. The normal pituitary gland was identified and preserved during the procedure. Histology confirmed an adamantinomatous craniopharyngioma, CNS WHO Grade 1.

Following surgery, the patient developed arginine vasopressin deficiency (AVP-D) characterised by marked polyuria and rapid onset severe hypernatremia (serum sodium rising from 142 mmol/L pre-op to 176 mmol/L within 18 hours). Urine specific gravity was consistently low (<1.005), confirming dilute urine. She exhibited confusion, disorientation, and impaired memory with no recall of recent events one day post-operatively and was commenced on desmopressin 0.5 mg subcutaneously (SC) on 12/06.

Impaired thirst sensation and ongoing confusion complicated fluid monitoring and the desmopressin was changed to an oral prescription with a gradual dose increase up to 300 mcg QDS. Due to the dose escalation, serum sodium did not remain within the reference range for 15 days post-operatively. This was also thought to reflect increased insensible losses due to the hot weather during this period and impaired thirst. Oral fluid intake was supported by prescribing water on the drug chart. Serum sodium gradually improved with careful fluid management, although episodes of agitation and fluctuating mental status persisted.

She also had evidence of adrenal and thyroid insufficiency. Peri-operative glucocorticoid replacement continued as prednisolone 4 mg and levothyroxine titrated to 100 mcg daily. She had neurocognitive impairment with persistent anterograde amnesia. Hypothalamic dysfunction caused by the initial tumour and potential damage post-operatively resulted in loss of satiety and weight gain of around 10 kg since admission; Semaglutide 0.25 mg SC weekly was initiated to treat likely hypothalamic obesity.

Discussion
This case highlights the complex endocrine, cognitive, and autonomic challenges in managing craniopharyngiomas with hypothalamic involvement. Long-term multidisciplinary follow-up is essential, with ongoing neuropsychological support, endocrine optimization, and careful consideration of timing of adjuvant radiotherapy.

Question
Any other ideas for management of patient with post operative AVP-D and loss of thirst regulation