CONCLUSION
Glucocorticoid replacement may precipitate diabetes insipidus in the setting of adrenal insufficiency. Low cortisol levels will stimulate the release of antidiuretic hormone and increase water reabsorption in the kidney. This effect can be inhibited by exogenous steroids which may then rapidly unmask or worsen central DI.

EP_A129

SPONTANEOUS RHINORRHOEA SECONDARY TO NORMAL-PRESSURE PARTIAL EMPTY SELLA SYNDROME

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INTRODUCTION/BACKGROUND
Empty sella syndrome is a disorder where the sella turcica is partially or completely filled with cerebrospinal fluid (CSF), resulting in compression and displacement of the pituitary gland. Empty sella is frequently coupled with increased intracranial pressure which causes spontaneous CSF leaks. Some people may experience normal-pressure CSF leaks, which can be due to idiopathic or congenital abnormalities. Both high-pressure and normal-pressure CSF leaks are commonly seen in middle-aged obese women.

CASE
A 43-year-old obese female with a BMI of 34.9 kg/m^2 presented with a two-day history of headache and clear nasal discharge persisting for a month. She reported no weakness or visual disturbances. Upon examination, her blood pressure measured 142/70 mmHg, her Glasgow Coma Scale was intact, and her neurological assessment revealed no abnormalities. The ophthalmological evaluation showed no signs of papilledema. Subsequently, a lumbar puncture was performed, indicating a normal opening pressure of 12 cm H_2O. Brain MRI revealed a partially empty sella, while CT imaging of the paranasal sinuses aimed to identify the cause of the CSF leak, was suggestive of cribriform plate dehiscence. Evaluation of anterior pituitary hormones yielded results within normal ranges. The case was then referred to a neurosurgical team for further evaluation and potential surgical intervention.

The occurrence of a spontaneous cCSF leak frequently coincides with the radiographic discovery of an empty sella. Its clinical manifestation can vary, encompassing symptoms such as headaches, rhinorrhoea, visual impairments, and hormonal imbalances. Assessing pituitary hormone levels is crucial, with hormonal supplementation warranted when deficiencies are identified. Surgical intervention is essential in managing CSF leaks to mitigate potential complications like meningitis and brain abscesses.

CONCLUSION
A CSF leak is important to be recognised at the initial presentation, as surgical intervention is required to reduce the complication of infections.

EP_A130

IMMUNOLOGICAL CROSSFIRE: ENDOCRINOPATHIES IN THE AGE OF IMMUNE CHECKPOINT INHIBITORS

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INTRODUCTION/BACKGROUND
Pembrolizumab is a PD1 receptor inhibitor which is a type of immune checkpoint inhibitor (ICI) for cancer therapy. Immune-related adverse events (IRAE) are more commonly encountered with increased use.

CASE
A 52-year-old male with recurrent renal cell carcinoma developed endocrinopathies following pembrolizumab. After failure of first-line treatment, he received the first dose of pembrolizumab in November 2022.

Six weeks later, he was symptomatic of hyperthyroidism with corresponding biochemistry of TSH <0.01 m IU/L (reference interval 0.55 – 4.8), free T4 of 28.8 pmol/L (reference interval 11.5 – 22.7) and free T3 of 9.7 pmol/L (reference interval 3.5 – 5.5). Following treatment with low dose and tapering carbimazole for eight weeks, he quickly became hypothyroid. Biochemistry showed TSH of 87.3 m IU/L, fT4 of 2.9 pmol/L and fT3 of 1.3 pmol/L. Anti-thyroid peroxidase and anti-thyroglobulin antibodies were negative. Carbimazole was discontinued and levothyroxine 50 mcg daily were commenced. Pembrolizumab was continued.

Four months later, he presented with lethargy and postural symptoms. A random serum cortisol level was undetectable at <14 nmol/L (reference interval 145 – 619) and ACTH level was inappropriately normal at 6 pg/ml (reference interval 0 – 46). He denied any polyuria, polydipsia, or

symptoms of mass effect. His gonadal function, prolactin and electrolytes were normal. There was marked clinical improvement following hydrocortisone replacement. MRI of the pituitary gland was completely normal. A diagnosis of ICI-induced hypophysitis and thyroiditis was made. He completed the nine cycles of pembrolizumab as initially planned with good cancer response. He remains well on levothyroxine and hydrocortisone replacement.

CONCLUSION

This case illustrates the typical sequelae of ICI-induced endocrinopathy of thyroiditis that occurred earlier and hypophysitis later. All patients in ICI should be monitored at close intervals for hormonal dysfunction and replaced as required.

EP_A131

CUSHING'S DISEASE PRESENTING YEARS AFTER PITUITARY APOPLEXY: A CASE REPORT

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INTRODUCTION/BACKGROUND

Pituitary apoplexy is a life-threatening condition resulting from haemorrhage or necrosis of a pituitary tumour. After an apoplectic event, recurrence of pituitary adenoma from the remaining pituitary tissue is still possible. We report an interesting case who had cushingoid features that manifested several years after her initial episode of pituitary apoplexy.

CASE

A 29-year-old female initially presented with a sudden onset of headache and diplopia. Clinically, she had left cavernous sinus syndrome. A cranial MRI showed a normal-sized pituitary gland with an internal haemorrhage suggestive of apoplexy and no filling defect within the cavernous sinus. She had hypocortisolism post pituitary apoplexy and needed hydrocortisone replacement. Her subsequent yearly MRI surveillance showed a normal-sized pituitary gland with no residual haemorrhage. Three years later, she exhibited typical full-blown cushingoid features of the moon face, facial plethora, proximal myopathy, hirsutism and purplish striae over the abdomen. Hydrocortisone replacement was withheld as repeated morning cortisol was 1002 nmol/L (145.4- 619.4) raising the possibility of endogenous hypercortisolism.

On further evaluation, she had an increased 24-hour urine-free cortisol, and abnormal serum cortisol during both overnight dexamethasone suppression test and low-dose dexamethasone suppression test. Laboratory findings strongly supported ACTH-dependent Cushing's syndrome whereby plasma ACTH was elevated at 20.58 pmol/L (1.6-13.9). However, her brain MRI did not show any evidence of a pituitary adenoma, hence underwent inferior petrosal sinus sampling to further evaluate the likely possibility of Cushing's disease.

CONCLUSION

This case demonstrates the possibility of recurrence Cushing's disease years after an episode of pituitary apoplexy, whereby the residual ACTH-producing adenoma might have grown slowly to form the second tumour. Therefore, long term monitoring is crucial for patients with pituitary apoplexy as they may develop tumour recurrence as well as hormonal deficiencies or excesses over time.

EP_A132

A CURIOUS CASE OF PITUITARY STALK THICKENING – LYMPHOCYTIC INFUNDIBULONEUROHYPOPHYSITIS

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INTRODUCTION/BACKGROUND

Hypophysitis is a rare inflammatory disorder that affects the pituitary gland and infundibulum. Lymphocytic infundibuloneurohypophysitis (LINH), one of the causes of primary hypophysitis, is a rare autoimmune inflammatory process that selectively affects the neurohypophysis and infundibulum, typically presenting with arginine vasopressin deficiency (AVP-D). Magnetic resonance imaging (MRI) with contrast demonstrates thickening of the pituitary stalk, enlargement of the neurohypophysis, or both with homogeneous enhancement. The inflammatory process in LINH can be self-limited and regression can be seen radiologically during follow-up.

CASE

A 22-year-old male presented with sudden onset polyuria and polydipsia in 2016, with clinical and laboratory findings consistent with AVP-D. Brain MRI demonstrated enlargement of the pituitary stalk, measuring 6 mm, and absence of the posterior pituitary bright spot. Other anterior pituitary hormones were normal, except for mildly raised prolactin levels. Investigations for secondary aetiologies were not significant. He was given a trial of glucocorticoid treatment. Serial brain MRI showed a reduction of the