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.

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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