Adult E-Poster

EP_A066

LANGERHANS HISTIOCYTOSIS-RELATED HYPOPHYSITIS: A DISTINCT CAUSE OF CRANIAL DIABETES INSIPIDUS

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INTRODUCTION/BACKGROUND

Cranial diabetes insipidus (DI) is a rare manifestation of hypothalamo-pituitary axis disorders. Langerhans Cell Histiocytosis (LCH), a clonal disease of dendritic cells, can infiltrate multiple organs, including the pituitary gland. When the posterior pituitary is involved, LCH can cause secondary hypophysitis, leading to cranial DI. Due to overlapping symptoms with more common conditions, diagnosis can be delayed.

CASE

A 22-year-old female with a one-year history of bullous skin lesions and oral ulcers was initially diagnosed with pemphigus vulgaris and treated with prednisolone. However, a skin biopsy later confirmed LCH. Imaging, including CT of the brain, neck and thorax-abdomen-pelvis, showed multisystem involvement (skin, ear, thyroid, and thymus). Hence, she underwent chemotherapy.

After the first cycle of chemotherapy with vinblastine and high-dose dexamethasone, she developed vomiting and lethargy. Laboratory tests revealed a hyperosmolar hyperglycemic state (serum osmolality 323 mOsm/kg). Despite normalisation of her blood glucose, she developed marked polyuria (>13,000 mL/day). Further testing showed serum osmolality of 294 mOsm/kg and urine osmolality of 54 mOsm/kg. A desmopressin trial resulted in a >50% increase in urine osmolality (398 mOsm/kg at 2 hours; 511 mOsm/kg at 4 hours), confirming cranial DI. MRI revealed a 1.1 × 1.3 × 1.0 cm lesion in the pituitary infundibulum, consistent with LCH-related hypophysitis, along with empty sella syndrome, likely secondary to chronic pituitary involvement and glucocorticoid therapy. She was treated with sublingual desmopressin and high-dose steroids, showing clinical improvement.

CONCLUSION

This case highlights the need to consider cranial DI as a manifestation of systemic LCH, especially in young adults with multisystem disease. Given the overlap of symptoms, early recognition of cranial DI is crucial for timely management.

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DISCORDANT THYROID FUNCTION TESTS: DIAGNOSTIC CHALLENGES IN A PATIENT WITH A TSH-SECRETING PITUITARY ADENOMA

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INTRODUCTION/BACKGROUND

Discordant Thyroid Function Tests (TFTs) may present a diagnostic challenge in clinical practice. Assay interference must be excluded before proceeding to further investigations. Rarely, the underlying diagnosis may be a TSH-secreting pituitary adenoma (TSHoma) or resistance to thyroid hormone (RTH). Making a diagnostic distinction between these two conditions is important as their clinical management varies significantly.

CASE

A 47-year-old engineer was referred to the Endocrine clinic with symptoms of hyperthyroidism and discordant TFTs. These symptoms improved with propranolol and carbimazole. Only his paternal aunt has a goitre on family history. On physical examination, there was no palpable goitre. His TFT was discordant before starting carbimazole [TSH 4.32 mIU/L (NR:0.27-4.20), fT4 34.1 pmol/L (NR: 12.0-22.0), fT3 11.6 pmol/L (NR: 3.10-6.80)]. Discordance persisted despite using two different immunoassays after stopping carbimazole. Alpha-subunit was raised (2.47 IU/L; NR: 0.0-0.7). A pituitary MRI showed pituitary macroadenoma. Pituitary hormones were within normal limits except for an elevated IGF-1. An oral glucose tolerance test was inconsistent with acromegaly as nadir growth hormone was 0.8 mIU/L (0.27 g/L). Several tests distinguishing a TSHoma from RTH were unavailable, so the patient underwent a somatostatin test. Following octreotide, TSH was suppressed with low-normal free thyroid hormone levels, highly suggestive of a TSHoma.

Our patient underwent an endoscopic transsphenoidal hypophysectomy. The histology report confirmed TSH and growth hormone-secreting adenoma.

CONCLUSION

The case illustrates challenges in establishing the diagnosis of a TSHoma with resource limitations and supports using intramuscular octreotide LAR as a diagnostic tool. Appropriate evaluation of discordant TFT is paramount

to avoid unnecessary investigations and treatments. The somatostatin test can be useful and practical in differentiating TSHoma from resistance to thyroid hormone.

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LATENT AUTOIMMUNE DIABETES IN YOUTH PRESENTING AS YOUNG-ONSET TYPE 2 DIABETES: A CASE REPORT

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INTRODUCTION/BACKGROUND
Latent autoimmune diabetes forms a continuous age-related spectrum from LADY to LADA, where LADY exhibits greater autoimmunity. Latent autoimmune diabetes in youth (LADY) is diagnosed in individuals aged 15 to 29 years. A high prevalence of LADY is observed among youth with the T2DM phenotype. The TCF7L2 rs12255372 polymorphism is linked to an increased risk of developing T2DM at a young age and is associated with lower levels of GADA in individuals with either T2DM or latent autoimmune diabetes.

CASE
A 28-year-old female with no history of T2DM was admitted to the emergency room due to altered mental status. She had experienced a weight loss of approximately 10 kg and frequent nocturnal urination; however, she had never consulted a physician. Both her parents and her sister have a known history of T2DM. A physical examination revealed excess body weight and acanthosis nigricans. Laboratory results indicated elevated glucose levels, a high A1C level, a normal fasting C-peptide, negative ketones in the urine and a high HOMA-IR score. Following the patient's clinical improvement, we transitioned from insulin to oral hypoglycemic drugs. After several weeks, we identified a positive anti-GAD result and the TCF7L2 gene polymorphism, rs12255372 (G/T).

CONCLUSION
The clinical diagnosis of latent autoimmune diabetes (LAD) can be quite challenging. Young individuals exhibiting a T2DM phenotype should undergo assessment for pancreatic islet cell autoantibodies. Common TCF7L2 gene polymorphisms are linked to T2DM and latent autoimmune diabetes but not type 1 diabetes.

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SUSPECTED LEFT ADRENOCORTICAL CARCINOMA LATER DIAGNOSED AS EXTRA-ADRENAL COMBINED SCHWANNOMA AND GANGLIONEUROMA

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INTRODUCTION/BACKGROUND
Ganglioneuromas and schwannomas are both rare benign tumours. They arise from different types of nerve cells: ganglioneuromas from autonomic ganglion cells and schwannomas from nerve sheath cells (Schwann cells). A combined adrenal ganglioneuroma and schwannoma is extremely rare, representing 1.4% of adrenal incidentalomas.

CASE
We present a 41-year-old male with an underlying left ureteric calculus who was referred for left adrenal incidentaloma from CT urography (CTU). He had no history of paroxysms of headache, sweating or palpitations. There were no symptoms to suggest Cushing's or underlying malignancy. On examination, he was normotensive and there were no discriminatory features of Cushing's. Laboratory evaluation showed normal potassium (4.3 mmol/L), and the overnight dexamethasone test (ODST) was appropriately suppressed (12 nmol/L). 24-hour urine metanephrine was within normal range. Testosterone and DHEAS were within the normal range with 19 nmol/L and 3.830 umol/L levels, respectively. Initial CTU reported a left adrenal mass measuring 4.0 x 3.1 x 3.8 cm. The adrenal CT demonstrated a left adrenal mass measuring 4.1 x 3.2 x 4.0 cm, with a 39 Hounsfield unit, an absolute washout of 28% and a relative washout of 16%. These findings indicate an indeterminate adrenal mass with a differential of adrenocortical carcinoma or pheochromocytoma. He underwent a left adrenalectomy and was discharged well. Histopathological examination showed an encapsulated biphasic tumour. There were Verocay bodies, and the neoplastic cells were narrow, elongated and wavy with tapered ends, interspersed with collagen fibres, which are distinct characteristics of schwannomas. At the periphery of the tumour, a separate proliferation of spindle cells