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BALANCING DISEASE CONTROL AND METABOLIC HARM: A CASE OF IgG4-RELATED HYPOPHYSITIS

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INTRODUCTION
Immunoglobulin G4-related hypophysitis (IgG4-RH) is a rare fibro-inflammatory disorder affecting the pituitary gland. Glucocorticoids remain the first-line therapy, but their use may be complicated in patients with significant metabolic comorbidities. We report a case of suspected IgG4-RH presenting with hyperosmolar hyperglycemic state (HHS), highlighting the challenges of balancing disease control against glucocorticoid metabolic toxicity adverse effects.

CASE
A 32-year-old female with obesity and newly diagnosed diabetes mellitus was admitted with HHS. Prior to admission, she reported weight fluctuations, episodic headaches, progressive visual disturbance, and secondary amenorrhea. Following resolution of HHS, persistent polyuria of 10–16 L/day prompted further evaluation and led to a diagnosis of arginine vasopressin deficiency. Anterior pituitary hormonal work-up revealed hypogonadotropic hypogonadism. Pituitary magnetic resonance imaging demonstrated infundibular thickening measuring 0.5 cm, with concomitant marked bilateral parotid enlargement. Serum IgG4 was elevated at 2.26 g/L (0.63–2.01), raising strong suspicion for IgG4-RH with systemic involvement. Histopathological confirmation from the parotid gland biopsy was consistent with sialadenosis.

She was commenced on sublingual desmopressin and cyclical sex hormone replacement therapy. Given the provisional diagnosis of IgG4-RH, oral prednisolone 40 mg daily was initiated as a reduced induction regimen. However, treatment was poorly tolerated, with rapid weight gain from 93 to 100 kg and worsening glycemic control. Prednisolone was therefore tapered rapidly to 10 mg daily. Repeat imaging demonstrated interval improvement in infundibular thickening, but no functional endocrine recovery was observed.

CONCLUSION
This case illustrates the therapeutic challenge of managing IgG4-RH in the setting of pre-existing metabolic syndrome. Although glucocorticoids are effective for induction, their metabolic adverse effects may significantly restrict treatment tolerability. Early consideration of steroid-sparing agents, such as rituximab or azathioprine, may be important to achieve remission while minimizing glucocorticoid-related adverse effects.

EP_A116

A DIAGNOSTIC DILEMMA IN ECTOPIC ACTH SYNDROME: WHEN BIOCHEMISTRY AND IMAGING CONFLICT

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INTRODUCTION
Ectopic ACTH secretion (EAS) is a rare cause of Cushing's syndrome, accounting for 5–15% of cases. Pulmonary neuroendocrine tumors are the most frequent cause. However, primary tumor localization remains a significant diagnostic challenge, delaying effective treatment.

CASE
A 59-year-old female with diabetes mellitus presented with refractory hypertension and hypokalemia. Initial laboratory findings revealed a baseline cortisol of 1,164 nmol/L and morning adrenocorticotropic hormone (ACTH) of 19.5 pmol/L (normal <10.2 pmol/L), with an overnight dexamethasone suppression test cortisol of 624 nmol/L. Pituitary magnetic resonance imaging (MRI) and computed tomography (CT) scans of the thorax, abdomen, and pelvis were initially unremarkable. A PET scan identified mild hypermetabolism in the left adrenal gland and gastrointestinal tract, though colonoscopy revealed only chronic colitis. An intravenous desmopressin stimulation test demonstrated a 181% rise in ACTH and a 35% rise in cortisol, pointing toward pituitary Cushing's. However, repeat pituitary MRI remained normal.

A high-dose dexamethasone suppression test showed approximately 34% cortisol suppression (cortisol 1,128 nmol/L to 745 nmol/L), suggesting ectopic Cushing's syndrome. Bilateral inferior petrosal sinus sampling demonstrated a peak central-to-peripheral ACTH ratio of 1.8 on the right and 1.6 on the left, pointing toward ectopic Cushing's.

Localization with DOTATATE PET-CT identified a 7 × 9 × 8 mm nodule in the right middle lobe. A wedge resection was performed. Histopathology confirmed a typical carcinoid tumor with clear resection margins. Immuno-histochemistry revealed tumor cells positive for CK AE1/

AE3, synaptophysin, chromogranin, and INSM-1, with weak positivity for ACTH. The latest 8 am cortisol was 117 nmol/L and ACTH 1.74 pmol/L, confirming biochemical cure. Hypokalemia resolved, with improvement in her metabolic profile.

CONCLUSION

This case highlights the diagnostic complexities of EAS, particularly when initial imaging is inconclusive and biochemical tests yield conflicting results. Timely, precise localization of the causative tumor is crucial for successful surgical intervention and to prevent severe complications of hypercortisolism, thereby improving patient outcomes.

EP_A117

FROM HYPERNATREMIA TO HYPONATREMIA: SEQUENTIAL CENTRAL AVP DEFICIENCY (AVP-D) AND CEREBRAL SALT WASTING (CSW) IN TUBERCULOUS MENINGITIS (TBM)

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INTRODUCTION

Electrolyte imbalance is common in central nervous system infections. Arginine vasopressin deficiency (AVP-D), Cerebral Salt Wasting (CSW), and Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) are important etiologies that can cause opposing extremes of serum sodium, posing diagnostic and therapeutic challenges. We report a rare case of transient central AVP-D followed by CSW secondary to tuberculous meningitis (TBM).

CASE

An 18-year-old female presented with a 1-month history of fever, reduced responsiveness, and visual hallucinations. Her Glasgow Coma Scale was E4V1M4 with neck stiffness and upper motor neuron signs. Initial investigations revealed severe hyponatremia (119 mmol/L) and communicating hydrocephalus with third ventricle ballooning on brain imaging. Coupled with a positive tuberculosis contact, anti-tuberculous therapy was initiated for probable TBM alongside 3% saline correction prior to insertion of external ventricular drain (EVD). Her condition deteriorated on day 5, requiring intubation for aspiration pneumonia. Repeated imaging showed worsening hydrocephalus, necessitating EVD revision. She subsequently developed polyuria (urine output [UO] 150–300 mLs/hour), with biochemical findings consistent with AVP-D (serum sodium 154 mmol/L; urine osmolality 96 mOsm/kg; urine sodium <20 mmol/L). Intravenous desmopressin 1 mcg was administered, and UO reduced to 30 mLs/hour. However, polyuria recurred on Day 9, accompanied by tachycardia, hypotension, and a rapid decline in serum sodium to 120 mmol/L. Diagnosis of CSW was established (urine sodium 204 mmol/L; urine osmolality 470 mOsm/kg). Oral fludrocortisone was initiated and titrated to 0.4 mg daily to maintain serum sodium >130 mmol/L. Due to persistent hydrocephalus, right ventriculoperitoneal shunt was inserted on Day 22, after which her UO gradually decreased, allowing tapering of fludrocortisone. She remains on fludrocortisone 0.1 mg daily with ongoing rehabilitation.

CONCLUSION

TBM can be complicated by SIADH, AVP-D, or CSW. Concurrent AVP-D and CSW have not been reported. This case highlights the dynamic electrolyte disturbances in TBM which may lead to diagnostic confusion and therapeutic error. Early recognition and tailored therapy, alongside definitive management to reduce intracranial pressure, are essential for optimal outcomes.

EP_A118

MASKING ANDROPAUSE BY A FUNCTIONING GONADOTROPH MACROADENOMA: DISCORDANT CLINICAL AND HORMONAL FINDINGS

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INTRODUCTION

Functioning gonadotroph adenomas (FGPAs) in males are rare and typically present with macroorchidism. In elderly patients, the presentation can be confounding. This case illustrates a unique scenario where a pituitary tumor masked the physiological decline of testosterone, maintaining the patient's stamina despite Introduction testicular involution.

CASE

A 65-year-old male was referred for a pituitary macroadenoma (10.7 × 10.6 × 11.4 mm) following progressive headaches. Remarkably, the patient denied symptoms of andropause; his stamina, mood, and libido were well-preserved. Investigations revealed elevated follicle-stimulating hormone (15.42 mIU/mL) and luteinizing