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.

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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