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

hormone (12.1 mIU/mL) with normal testosterone levels. Scrotal ultrasound showed bilateral testicular involution (right: 8.66 mL, left: 7.21 mL), contradicting the expected macroorchidism of FGPA. Although the patient felt his vision was normal, a confrontation test revealed temporal visual field defects, indicating chiasmatic compression. This case presents a striking clinical discordance where tumor-induced gonadotropin hypersecretion provided a supra-physiological drive to the involuting testes, maintaining testosterone levels and masking andropause. The primary indication for transsphenoidal surgery is the Introduction evidence of visual field defects. However, removing the adenoma will abruptly eliminate this "hormonal drive." Postoperative management will focus on rigorous hormonal monitoring. Testosterone replacement therapy is not immediately indicated; rather, it will be carefully planned only if postoperative evaluations demonstrate a significant hormonal decline accompanied by symptomatic hypogonadism. This tailored approach ensures that the benefits of intervention outweigh the potential risks in an elderly patient.

CONCLUSION

In elderly patients, FGPAs can mask age-related hormonal decline. In this case, clinicians must treat the patient, not just the tumor. Anticipating a potential decline in postoperative stamina is as crucial as the surgery itself. Comprehensive hormonal evaluation and a personalized approach to therapy are essential to preserve quality of life after the "masking" effect is removed.

EP_A119

WHEN HYPERPROLACTINEMIA FAILS TO SUPPRESS: THE SILENT GONADOTROPH IN A PITUITARY MACROADENOMA

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INTRODUCTION

Pituitary macroadenomas may present with mass effects, hypopituitarism, or hormone hypersecretion. Hyperprolactinemia, resulting from a prolactin-secreting tumor or stalk compression, typically suppresses gonadotropins. Thus, elevated follicle-stimulating hormone (FSH) and luteinizing hormone (LH) with low testosterone in this context are unusual. We report a macroprolactinoma with a clinically non-functioning gonadotroph adenoma.

CASE

A 69-year-old male presented with acute giddiness and headache while in Vietnam. He reported reduced libido but no visual symptoms or galactorrhea. Examination showed no neurological deficits, normal visual fields, and secondary sexual characteristics; bilateral testes volume of 25 mL. Magnetic resonance imaging (MRI) brain revealed a 1.6 × 2.2 × 2.1 cm pituitary macroadenoma compressing the optic chiasm and pituitary stalk.

Initial pituitary evaluation demonstrated hyperprolactinemia (prolactin >200 µg/L; normal 2.4–13.1), central hypothyroidism (T4 6.8 pmol/L [7.8–14.4], thyroid-stimulating hormone 2.3 mIU/L [0.38–5.3]), elevated FSH (21.5 IU/L [1.2–19.2]) and LH (191 IU/L [1.24–8.62]), low testosterone (8.88 nmol/L), normal insulin-like growth factor 1 (IGF-1) (68 µg/L [46.5–191.9]), and cortisol 237 nmol/L. Cabergoline was initiated at 0.25 mg twice weekly and titrated to 1 mg twice weekly over 4 months, alongside levothyroxine 25 µg daily.

Repeated MRI brain 2 months after cabergoline initiation showed a persistent macroadenoma (1.7 × 2.2 × 2.2 cm) with bilateral cavernous sinus extension. Prolactin decreased to <170 µg/L, central hypothyroidism persisted; levothyroxine was optimized, and hydrocortisone was initiated.

At the 5-month follow-up, prolactin further improved to 57 µg/L. Central hypothyroidism and borderline adrenal function persisted, requiring continued replacement therapy. Testosterone remained low-normal (10.2 nmol/L) despite elevated FSH (15.4 IU/L) and LH (92 IU/L). Follow-up MRI and hormonal reassessment were planned in May 2026.

CONCLUSION

Profoundly elevated prolactin level >200 µg/L and response to cabergoline reflect true prolactinoma rather than stalk effect. Persistently low testosterone with discordant high FSH/LH suggests a non-functioning gonadotroph component producing biologically inactive gonadotropins. Definitive diagnosis requires histopathological confirmation, while serial biochemical and radiological follow-up guides management and clarifies tumor subtype.