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SUPRASELLAR MOGAD: RARE ENDOCRINE MANIFESTATIONS OF HYPOPITUITARISM AND DIABETES INSIPIDUS

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INTRODUCTION

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an uncommon inflammatory demyelinating disorder of the central nervous system, with a reported prevalence of 1.3–2.5 per 100,000. Hypothalamic–pituitary involvement is rare and can mimic structural lesions, presenting with varying degrees of hypopituitarism and central diabetes insipidus.

CASE

A 30-year-old male presented with a 2-year history of poor concentration, blurring of vision and lethargy. Initial investigations revealed severe hypernatremia (serum sodium 165 mmol/L) and a 1.4 × 2.1 × 1.3 cm suprasellar cistern mass on CT scan, with differential diagnoses including meningioma and germinoma. Brain magnetic resonance imaging showed abnormal signals in the optic pathways and hypothalamus, raising suspicion of a demyelinating process.

Endocrine evaluation confirmed panhypopituitarism: elevated prolactin (1125.2 mIU/L), hypogonadotropic hypogonadism (follicle-stimulating hormone 0.7 IU/L, LH 0.3 IU/L, testosterone <0.35 nmol/L), central hypothyroidism (TSH 3.8 mIU/L, free T4 5.88 pmol/L) and low cortisol (33.1 nmol/L). Persistent hypernatremia (up to 171 mmol/L) with high serum osmolality (370 mOsm/kg) and low urine osmolality (237 mOsm/kg) confirmed central diabetes insipidus, as urine osmolality rose to 755 mOsm/kg following intravenous desmopressin.

He was commenced on sublingual desmopressin 60 micrograms twice daily, hydrocortisone (10 mg morning, 5 mg afternoon), levothyroxine 75 micrograms daily and monthly intramuscular testosterone 150 mg. Subsequent readmissions for generalized weakness and fever led to cerebrospinal fluid analysis and serum testing, which were positive for MOG antibodies and negative for aquaporin-4 antibodies, confirmed MOGAD.

During a third admission with recurrent generalized weakness, he responded favorably to intravenous methylprednisolone (1 g daily for 5 days), followed by a tapering oral prednisolone regimen.

CONCLUSION

MOGAD can involve the hypothalamic–pituitary axis and mimic a suprasellar mass. In patients with panhypopituitarism, central diabetes insipidus and compatible imaging, inflammatory demyelination should be suspected. MOG antibody positivity and response to corticosteroids support diagnosis and guide management.