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MALIGNANT HYPERNATREMIA COMPLICATING A HYPOTHALAMIC TUMOR: AN ENDOCRINE EMERGENCY

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INTRODUCTION

Hypernatremia is a common yet high-mortality electrolyte disorder. The hypothalamus maintains water homeostasis via thirst sensation and arginine vasopressin (AVP) secretion. Hypothalamic tumors, such as gliomas, can progressively destroy these osmoregulatory centres, leading to "malignant" hypernatremia (>180 mmol/L). We report a case of life-threatening hypernatremia in a patient with a progressive hypothalamic glioma, exploring its complex pathophysiology.

CASE

A 41-year-old female with a progressive high-grade hypothalamic glioma and persistent hydrocephalus presented with generalized weakness, reduced oral intake, and dehydration. Her initial Glasgow Coma Scale was E4V3M6. Laboratory investigations revealed malignant hypernatremia (serum sodium 209 mmol/L) and a serum osmolarity of 441 mOsm/kg. Despite life-threatening dehydration (urea 26.2 mmol/L, creatinine 343 umol/L), she was still able to deceptively produce urine output of 400 mL/day with a concentrated urine osmolarity of 890 mOsm/kg. A 1 mcg IV desmopressin trial reduced urine output to 60 mL/day and serum sodium by 10 mmol/L within 14 hours, confirming relative AVP deficiency. The patient's malignant hypernatremia was gradually corrected to 168 mmol/L over 1 week (8–12 mmol/L/day) using controlled intravenous hydration. However, her condition deteriorated due to hospital-acquired infection, and she succumbed 10 days after admission.

CONCLUSION

This case underscores several critical learning points for managing hypothalamic emergencies. First, hypothalamic tumors can reset the osmostat or destroy osmoregulatory centres, causing adipsic AVP deficiency. Second, clinicians must be alert to "masked polyuria" where severe hypovolemia reduces the glomerular filtration rate, causing urine output to appear "normal" despite underlying AVP deficiency. This state of "relative polyuria" is a hallmark of hypothalamic hypernatremia, thus indicating that a normal urine output does not rule out AVP deficiency. While desmopressin is indicated, its use in adipsic patients demands strict fluid titration to prevent iatrogenic hyponatremia. Rapid hypotonic correction carries a proven risk of cerebral oedema, and sodium measurement accuracy varies significantly across laboratory methods in extreme ranges.

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ISOLATED CRANIAL DIABETES INSIPIDUS UNMASKED AFTER HYPEROSMOLAR HYPERGLYCEMIC STATE IN TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Central diabetes insipidus (CDI) is characterized by impaired arginine vasopressin secretion, resulting in hypotonic polyuria and hypernatremia. In patients with coexisting diabetes mellitus, persistent polyuria may be misattributed to hyperglycemia, delaying recognition of a concurrent water balance disorder. CDI without identifiable structural abnormalities may be overlooked, leading to delayed diagnosis and complications.

CASE

We report a 30-year-old Malay female with underlying type 2 diabetes mellitus and premorbid obesity who presented with hyperosmolar hyperglycemic state, accompanied by persistent polyuria, polydipsia, weight gain, and amenorrhea. Despite resolution of hyperglycemia, she had ongoing polyuria with hypernatremia, raising suspicion of a concomitant water balance disorder.

Biochemical evaluation demonstrated a hyperosmolar state with serum osmolality 334 mOsm/kg and inappropriately dilute urine (urine osmolality 254 mOsm/kg), supporting impaired vasopressin activity. A modified water deprivation test showed a >50% rise in urine osmolality following desmopressin, consistent with CDI. Diabetes autoantibody screening was negative. Anterior pituitary function was preserved, with normal thyroid and adrenal axes (thyroid-stimulating hormone 3.30 mIU/L, cortisol 290.6 nmol/L), and gonadotropins not suggestive of hypopituitarism (luteinizing hormone 6.8 IU/L, follicle-stimulating hormone 24.6 IU/L).

Neuroimaging with computed tomography brain showed no structural hypothalamic–pituitary lesion. Magnetic resonance imaging of the pituitary demonstrated absence of the posterior pituitary bright spot, with preserved gland morphology and no focal lesion, supporting CDI without an identifiable structural cause.