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TOO LOW FROM A SELF-BLOW: DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN A CASE OF HIRATA'S SYNDROME

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INTRODUCTION

Non-diabetic hypoglycemia in older adults warrants careful evaluation across insulin-mediated and non-insulin-mediated causes. We present a challenging case of insulin autoimmune syndrome (IAS; Hirata's syndrome).

CASE

An 85-year-old female presented with severe hypoglycemia (capillary blood glucose [CBG] 1.8 mmol/L) with reduced consciousness, preceded by 2 months of recurrent dizziness relieved by food intake. Appetite and weight were stable. Past medical history included stage 3 chronic kidney disease and osteoporosis, but no diabetes. Medication review revealed a recent 2-week course of traditional supplement, long-term Neurobion®, but no agents with recognized hypoglycemic potential. Physical examination showed a moderately built elderly female with a body mass index of 24.3 kg/m² and no Cushingoid features.

Recurrent hypoglycemia (CBG nadir 1.5 mmol/L) occurred in both fasting and postprandial states, fulfilling Whipple's triad. Baseline investigations showed an estimated glomerular filtration rate of 42 mL/min/1.73 m², AM cortisol of 700 mmol/L, and unremarkable liver and thyroid function tests. During a hypoglycemic episode (CBG 2.3 mmol/L), plasma insulin and C-peptide were inappropriately raised at 203.6 mU/L (reference interval [RI]: 3.0–25.0) and 33 ng/mL (RI: 0.9–7.1), respectively, confirming endogenous hyperinsulinemic hypoglycemia. Polyethylene glycol precipitation showed 21% insulin recovery, raising suspicion for an autoimmune cause. Elevated insulin autoantibodies (175 AU/mL; RI <20) confirmed IAS. Computed tomography of the abdomen and endoscopic ultrasound excluded a pancreatic neuro-endocrine tumor.

Nutritional management comprising frequent low glycemic index feeds and uncooked cornstarch was commenced. Diazoxide 100 mg TDS caused fluid overload and severe hyponatremia, while hypoglycemia persisted at lower doses, necessitating discontinuation. Subcutaneous octreotide 100 mg QID was required to control hypoglycemia. Prednisolone 30 mg BD improved glycemic stability. Insulin autoantibodies titer remained 175 AU/mL at 2 weeks; reassessment was conducted at 4 weeks with consideration for biologics if persistent.

CONCLUSION

Endogenous hyperinsulinemic hypoglycemia, after excluding insulinoma, should raise suspicion for IAS. Management includes removing triggers, supportive care, and immunomodulatory therapy in severe cases.

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NOT JUST ANOTHER CASE OF TYPE 2 DIABETES IN AN ADOLESCENT

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INTRODUCTION

Cushing's disease (CD) in adolescents may present with subtle clinical features, resulting in a significant diagnostic challenge. We report a case of a young lady whose CD initially masqueraded as Type 2 diabetes (T2D), highlighting the difficulties in differentiating early hypercortisolism from T2D.

CASE

A 12-year-old female was incidentally diagnosed with diabetes during routine medical screening. Examination revealed an overweight female without the classical features of Cushing's syndrome. Due to the presence of acanthosis nigricans, a diagnosis of T2D was initially made. Her diabetes remained well-controlled with a single oral glucose-lowering drug.

The diagnostic challenge became apparent over time. She experienced delayed menarche at the age of 17, and a diagnosis of Cushing's syndrome was suspected when she subsequently developed hypertension and reduced bone mineral density. Biochemical evaluation was consistent with adrenocorticotropic hormone (ACTH)-dependent hypercortisolism, evidenced by the failure of serum cortisol suppression on low dose and overnight dexamethasone suppression tests. Her 24-hour urinary cortisol was elevated twofold, and plasma ACTH was elevated (17.8 pmol/L). MRI demonstrated a right-sided pituitary microadenoma (0.3 × 0.5 × 0.3 cm), and inferior petrosal sinus sampling confirmed the diagnosis of CD. She