antibody or causative agent cannot be identified. It is essential to take the necessary actions to eliminate other causes for the discordant TFT results and to prevent unnecessary thyroxine replacement. For this patient, any future TFT testing should be conducted at Lab A to rule out any potential assay interference with upcoming samples, if needed.

EP_A150

THE PARADOX OF PLENTY: WHEN GLUCOCORTICOID RESISTANCE SYNDROME MEETS SYSTEMIC LUPUS ERYTHEMATOSUS

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INTRODUCTION/BACKGROUND

Glucocorticoid resistance syndrome (GRS) is a rare condition characterized by biochemical hypercortisolism without the typical clinical manifestations of Cushing's syndrome. Patients with GRS exhibit elevated serum cortisol, increased 24-hour urinary free cortisol, normal to elevated ACTH, non-suppressed low-dose dexamethasone-suppression test results and preserved circadian rhythm, which are findings that help distinguish it from Cushing's disease. It is associated with various mutations in the NR3C1 gene, which encodes the glucocorticoid receptor. Clinical presentations can vary from being asymptomatic to exhibiting features of mineralocorticoid or androgen excess such as hypertension with hypokalemia or hyperandrogenism.

CASE

A 51-year-old female with type-2 diabetes mellitus, hypertension, and dyslipidemia presented with bilateral lower limb edema and intermittent facial flushing. Her BMI was within normal range, and her blood pressure and blood glucose were well-controlled. Notably, she had persistent hypokalemia and elevated cortisol levels. MRI of the pituitary revealed a partial empty sella with a suspected right-sided pituitary adenoma. Her bone mineral density was also normal. Inferior petrosal sinus sampling confirmed ACTH-dependent hypercortisolism. However, in the absence of clinical features of Cushing's syndrome, diagnosis of GRS was made.

She was started on dexamethasone, leading to significant reduction in cortisol levels over nine months. However, her condition was complicated by recurrent infections, soft tissue abscesses, and a newly diagnosed systemic lupus erythematosus (SLE) with concomitant lupus nephritis. Frequent steroid adjustments were necessary to manage autoimmune flares, which, in turn, increased her risk for opportunistic infections, culminating in severe Pneumocystis jirovecii pneumonia.

CONCLUSION

This case illustrates the diagnostic and therapeutic challenges of managing GRS, particularly when complicated by autoimmune disease and infection risk. While dexamethasone is effective in suppressing the HPA axis in GRS due to its glucocorticoid receptor affinity and mineralocorticoid-sparing properties, its use in patients with concurrent immunosuppressive conditions like SLE requires careful balance to avoid immunosuppression-related complications. Individualized steroid management is crucial to optimize outcomes and minimize adverse events.

EP_A151

DIAZOXIDE-INDUCED HYPERGLYCAEMIC CRISIS IN AN ELDERLY: A TRAP FOR THE UNWARY

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INTRODUCTION/BACKGROUND

Diazoxide inhibits pancreatic insulin secretion and is a well-established pharmacological agent for management of hypoglycaemia in insulinoma. Hyperglycemic emergencies associated with its use are rare, being mostly reported in the elderly and in children.

CASE

An 88-year-old female with hypertension and dyslipidaemia presented to the emergency room with syncope and was noted to be hypoglycaemic with capillary glucose of 2.6 mmol/L. She reported a year-long history of recurrent presyncopal episodes and early morning hunger pangs. Renal profile, 8 am cortisol, thyroid and liver function tests were normal. Laboratory tests confirmed endogenous hyperinsulinemia (random blood glucose: 1.7 mmol/L, serum insulin 373 pmol/L, C-peptide 3054 pmol/L) with negative sulfonylurea screening. CT imaging revealed a 0.4 x 0.9 cm hypodense lesion in the proximal pancreas. She was started on diazoxide and was advised glucose