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

monitoring and dietary modifications. Her capillary blood glucose remained stable (5–7 mmol/L) on follow-up. However, weeks later, she presented again with reduced responsiveness. Investigations revealed overlapping diabetic ketoacidosis and hyperosmolar hyperglycaemic state with acute kidney injury (glucose 32 mmol/L, ketones 7.5 mmol/L, pH 7.2, HCO_3 15mmol/L, Na 162 mmol/L, urea 27 mmol/L, creatinine 309 mol/L, osmolality 362 mOsm/L). CXR showed right lower zone consolidation. She was treated with antibiotics and insulin, requiring up to 30 units per day when steroid was added for bronchospasm. After recovery and weaning of steroids, insulin was tapered off. However, she experienced further episodes of hypoglycaemia despite being off all glucose lowering medication. Diazoxide was resumed at 100 mg every other day. Family opted for nonsurgical management and she remained well with normal home glucose profile on follow up 3 months later.

CONCLUSION

This case highlights the rare but potentially life-threatening side-effect of diazoxide. The risk is heightened in the elderly, especially when confounded by renal impairment, high doses, intercurrent illness or steroid use. Awareness and vigilant monitoring are essential in the vulnerable to avoid adverse outcome.

EP_A152

THE ROLE OF DAPAGLIFLOZIN AS AN ADJUNCTIVE THERAPY IN SIADH-INDUCED HYPONATREMIA

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INTRODUCTION/BACKGROUND

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) leads to impaired water excretion and dilutional hyponatremia. Sodium-glucose cotransporter 2 inhibitors (SGLT2i), which were initially developed for diabetes and heart failure, have shown promise as a novel treatment for chronic SIADH-related hyponatremia based on recent studies.

CASE

We report the case of a 66-year-old male with comorbidities of systemic lupus erythematosus, heart failure and adrenal insufficiency on steroid replacement. His heart failure medications included furosemide, spironolactone, and dapagliflozin, which was initiated in May 2024. Prior to admission, his serum sodium levels ranged from 130–135 mmol/L. During his current hospitalization, he was treated for pneumonia and incidentally noted to be hyponatremic with a sodium level of 128 mmol/L. At this point, diuretics and dapagliflozin were withheld. He responded to fluid boluses given, showing an initial improvement in his serum sodium, which then plateaued, followed by a declining trend to a nadir of 115 mmol/L. Paired serum and urine samples sent were consistent with SIADH. Hormonal workup taken showed normal thyroid and cortisol level. The patient was then given hypertonic saline to correct the initial severe hyponatremia, followed by fluid restriction and oral salt. Despite an initial improvement, this effect was not sustained, with sodium levels remaining static at 125-126 mmol/L. Dapagliflozin was then reintroduced, resulting in progressive improvement in his serum sodium, which allowed for discontinuation of oral sodium supplementation. He showed progressive clinical improvement and was discharged well with a serum sodium of 138 mmol/L.

CONCLUSION

This case illustrates the potential benefit of SGLT2 inhibitors in managing SIADH-related hyponatremia. Reintroduction of dapagliflozin led to a sustained rise in sodium levels, even after discontinuing salt supplementation. SGLT2i may enhance free water clearance and could be considered as adjunctive therapy in chronic SIADH, alongside fluid restriction and sodium supplementation.

EP_A153

UNMASKING A HORMONAL CHAMELEON: TSHoma WITH HIDDEN ACTH CO-SECRETION

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INTRODUCTION/BACKGROUND

TSH-secreting pituitary adenomas (TSHomas) are rare and often misdiagnosed due to overlapping features with primary thyroid disorders. Even rarer are plurihormonal pituitary adenomas that co-secrete TSH and ACTH. We report a unique case where initial evaluation suggested a TSHoma, with ACTH co-secretion only suspected perioperatively based on clinical features and was later confirmed histologically.

CASE

A 41-year-old woman with a two-year history of hypertension and primary infertility presented with palpitations, heat intolerance, and insomnia. She had a history of