Adult E-Poster

EP_A148

CASE REPORT: RECURRENT UNILATERAL ALDOSTERONE-PRODUCING ADRENAL ADENOMA

https://doi.org/10.15605/jafes.040.S1.156

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INTRODUCTION/BACKGROUND

Primary aldosteronism (PA) is the most common cause of secondary hypertension. While adrenalectomy can be curative in unilateral cases, recurrence after total adrenalectomy is exceedingly rare. We describe a rare case of recurrent PA requiring two adrenalectomies, eight years apart.

CASE

A 43-year-old male was diagnosed with hypertension at age 30 and initially required four antihypertensive agents for blood pressure control. In 2012, biochemical screening confirmed PA, with an aldosterone-renin ratio (ARR) of 1991 and a post-saline suppression aldosterone level of 600 pmol/L. Adrenal CT revealed a hypodense lesion in the lateral limb of the left adrenal gland measuring 0.9 × 1.3 cm with HU of -4 to 20. Adrenal venous sampling (AVS) was performed but yielded inconclusive results due to failed cannulation of the left adrenal vein. In 2015, he underwent retroperitoneoscopic left adrenalectomy, with histopathology confirming an adrenal cortical adenoma. Postoperatively, his blood pressure improved and was maintained on a single antihypertensive agent.

Over the following years, his blood pressure gradually increased, requiring multiple medications. In 2023, repeat screening showed an ARR of 238 and adrenal CT showed a recurrent lesion in the left adrenal bed. A second left adrenalectomy was performed in December 2024. Postoperatively, his blood pressure normalized without the need for antihypertensives.

This case highlights the rare occurrence of recurrent PA after unilateral adrenalectomy. Possible mechanisms include residual hyperfunctioning adrenal tissue or the development of a new aldosterone-producing lesion in the ipsilateral adrenal bed. Some studies suggest that patients with certain genetic mutations such as KCNJ5 may be predisposed to developing multiple aldosterone-producing nodules, either at the time of initial surgery or later in the remaining adrenal tissue.

CONCLUSION

Recurrent PA after unilateral adrenalectomy is rare but clinically significant. Lifelong monitoring of blood pressure post adrenalectomy is essential. Repeat surgical intervention can achieve biochemical remission and restore blood pressure control in cases of recurrence.

EP_A149

WHEN NUMBERS DON'T ADD UP: DISCORDANT THYROID FUNCTION IN HIV INFECTION

https://doi.org/10.15605/jafes.040.S1.157

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INTRODUCTION/BACKGROUND

Thyroid function test (TFT) abnormalities in retroviral disease (RVD) are well documented, ranging from isolated low fT4 levels to overt hypothyroidism. However, careful evaluation of TFTs is essential as it presents a diagnostic challenge.

CASE

This is a case of a 28-year-old male with hypertension and end-stage kidney disease on peritoneal dialysis. He was diagnosed with vertical transmission of HIV since childhood, and the viral load is suppressed by regular antiretroviral therapy; oral lamivudine 500 mg daily, oral efavirenz 600 mg daily and oral abacavir 600 mg daily. He was referred for abnormal TFT, fT4 7.88 (9.01-19.05pmol/L), TSH 1.99 (0.35-4.94mIU/L). TFT was done for a pre-cadaveric renal transplant workout. Clinically, he is euthyroid without any palpable goiter. He denied consuming biotin-containing supplementation.

The results of the serial TFT showed a similar pattern. The pituitary hormonal workout excludes a central cause. Total T4 was normal at 68.1 (66-181 nmol/L). TSH assay interference was subsequently evaluated. Of the three different analyzers used (Lab A, B, and C), Lab A analyser displayed normal TFT results: TSH 1.93 (0.4-4 mIU/L), fT4 10.8 (10-26pmol/L), while the other lab shows low fT4 and normal TSH. Macro-TSH, heterophile antibody and rheumatoid factor interference run by Lab B were negative.

CONCLUSION

A low fT4 level combined with a normal TSH level may be affected by multiple factors, such as antiretroviral therapy (ART) and assay interference, as seen in this patient's case. Due to limited resources and testing capacity, the specific

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

https://doi.org/10.15605/jafes.040.S1.158

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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

https://doi.org/10.15605/jafes.040.S1.159

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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