EP_A018

ACQUIRED HYPOALDOSTERONISM WITH RENAL TUBULAR ACIDOSIS TYPE 4 IN A DIABETIC PATIENT

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INTRODUCTION/BACKGROUND
Renal tubular acidosis (RTA) type 4 is an uncommon cause of hyperkalaemia with non-anion gap acidosis. Here we present a case of acquired hypoaldosteronism.

CASE
A 60-year-old female with poorly controlled diabetes (HbA1c 9.3%) and hypertension presented with a two-year history of multiple episodes of asymptomatic hyperkalaemia. She was not on RAAS blockade or drugs causing hypoaldosteronism. There was no family history of RTA. Physical examination was unremarkable. Laboratory investigations revealed hyperkalaemia (5.9 mmol/L), normal sodium (136 mmol/L), metabolic acidosis (pH 7.27; HCO_3 17.8) and impaired renal function (creatinine 118 mmol/L; eGFR 43 ml/min/1.73m^2). Serum cortisol was normal (517.7 nmol/L) and serum aldosterone was low (<103 pmol/L), with serum renin mildly elevated (125.60 mU/L; supine 4.2-59.7; upright 5.399.1). There was no sonographic evidence of any suprarenal mass. She was commenced on frusemide 20 mg daily. However, due to persistent hyperkalaemia, fludrocortisone 50 mcg daily was added.

RTA type 4 results from aldosterone deficiency or resistance and is associated with diabetes in 50% of cases. Hypoaldosteronism can be due to reduced stimulus to release (hyporeninaemic) or reduced synthesis or secretion (drugs like heparin, NSAIDs, ciclosporin; congenital adrenal hypoplasia); whereas aldosterone resistance can develop in target organs (pseudohypoaldosteronism) due to tubulointerstitial disease or diabetic nephropathy. Chronic hyperglycaemia can lead to diabetic and autonomic nephropathy resulting in juxtaglomerular apparatus injury or deficiency in the conversion of prorenin to active renin. However, renin levels were unexpectedly normal in our patient. Initial treatment includes a low-potassium diet, diuretics, ion-exchange resins, and in persistent cases, fludrocortisone.

CONCLUSION
Acquired hypoaldosteronism should be considered in the diabetic patient with mild non-anion gap metabolic acidosis and hyperkalemia, especially if disproportionate to the degree of renal impairment.

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AN AUDIT OF PATIENTS WITH TYPE 1 DIABETES MELLITUS IN RELATION TO GLYCAEMIC CONTROL AND METABOLIC PROFILE IN A TERTIARY CENTRE

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INTRODUCTION
Type 1 diabetes (T1D) is a chronic disease affecting children and young adults which poses challenges to healthcare systems due to its associated microvascular and macrovascular complications. There is currently very little data focusing on this issue in Malaysia.

We aim to review the glycaemic control, metabolic profile and prevalence of acute and chronic complications of patients with T1D at the Endocrine Institute.

METHODOLOGY
A retrospective review was conducted using electronic medical records of patients who consulted in outpatient clinics at the Endocrine Institute between 1st December 2021 to 30th November 2022. A descriptive analysis exploring the association between glycaemic control, metabolic profile, complications treatment regimens and patient characteristics was done using SPSS version 21.

RESULT
A total of 143 patients were included. Glycaemic control and metabolic profiles were notably poor in this population with mean HbA1c and FBS of 9.0% and 10.0 mmol/L, respectively. LDL were elevated (>2.6 mmol/L) in nearly 80% of patients and 57.4% of patients were overweight or obese. Microvascular complications were predominant, with at least one-third of patients experiencing diabetic nephropathy or retinopathy.

CONCLUSION
This study found that these patients had poor overall glycaemic control and metabolic profiles, resulting in high incidence rates of chronic complications. This warrants a review of the management of T1D with emphasis on multidisciplinary specialised care and support required by these patients to improve outcomes.