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Delayed Diagnosis of Primary Aldosteronism in a Patient with Autosomal Dominant Polycystic Kidney Disease

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Xe Hui L, Nor Shaffinaz YA
Endocrinology Unit, Medical Department, Hospital Sultanah Bahiyah, Malaysia

INTRODUCTION

Hypertension is a common manifestation for both autosomal dominant ppolycystic kidney disease (ADPKD) and primary aldosteronism (PA). The occurrence of PA in ADPKD patients is extremely rare. The presence of multiple renal cysts makes identification of adrenal adenomas very challenging. Approximately 5 to 10 percent of adults with hypertension have a secondary cause. Hypertension is a common early finding in ADPKD, occurring in 50 to 70 percent of cases before any significant reduction in glomerular filtration rate. Cross-sectional and prospective studies report PA in >5% and possibly >10% of hypertensive patients, both in general and in specialty settings. PA is a condition well worth detecting because it is associated with excessive morbidity. We report an interesting case of a man with hypertension secondary to ADPKD being diagnosed with PA 9 years later.

CONCLUSION

Patients with PA have higher cardiovascular morbidity and mortality than age- and sex-matched patients with essential hypertension and the same degree of BP elevation. Diagnosing and treating patients with PA ameliorate the impact of this condition on important patient outcomes. Only a minority of patients with PA (9 to 37%) present with hypokalaemia, with hypokalaemia probably present in only the more severe cases. PA should be suspected when hypokalaemia occurs in a patient with hypertension. The echogenicity of the adrenal glands is similar to that of the retroperitoneal fat. It is even more difficult to visualize the adrenal glands on the ultrasound of a patient with polycystic kidneys. Hence, the methods of choice in the assessment of adrenal pathologies are computed tomography and magnetic resonance imaging.

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Lactic Acidosis and Transaminitis in a Type 1 Diabetes Patient with Recurrent Diabetic Ketoacidosis

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Xe Hui L and Shueh Lin L
Endocrine Unit, Medical Department, Penang Hospital, Malaysia

CASE

We report an interesting case of a 16-year-old boy with type 1 diabetes mellitus, who had 14 admissions for diabetic ketoacidosis (DKA) since 2016. During many of his admissions for severe DKA, despite resolution of DKA and being clinically well, he continued to have lactic acidosis and markedly elevated aminotransferases. Lactic acidosis is a common finding in DKA, whereas glycogenic hepatopathy (GH) is a rare complication of poorly controlled diabetes mellitus characterised by transient liver dysfunction, elevated liver enzymes and associated hepatomegaly. Lactic acidosis in DKA is multifactorial in aetiology, from anaerobic glycolysis due to inadequate tissue perfusion and oxygenation, as well as metabolic derangements in DKA itself contributing to elevated lactate levels. On the other hand, the pathophysiology of GH is incompletely understood, and clinical characteristics have not been fully characterised. It is believed to be the consequence of recurrent fluctuations in glucose level with hyperglycaemia, hypoglycaemia and hyperinsulinization.

CONCLUSION

Awareness of the occurrence of lactic acidosis and glycogenic hepatopathy in patients with uncontrolled diabetes presenting with DKA should be increased among clinicians to guide further management appropriately. With this, we can increase our patient data pool to better understand patient characteristics and associated risk factors.