



Severe Hypokalaemia as a Cause of Reversible Diabetes Mellitus

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Authors' contributions

This work was carried out in collaboration between all authors. Authors OE and NH managed the patient. Authors OE, NH, NW and BJ wrote the manuscript. Authors NW, NH and BJ did the literature searches. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aims: To discuss the likelihood of severe hypokalaemia as a cause of reversible diabetes.
Presentation of the Case: A 46 year old patient presented to the Accident and Emergency unit (A&E) with a history of polyuria and polydipsia of recent onset. He was severely hyperglycaemic (glucose = 40 mmol/L, HbA1c = 11%), hypokalaemic (serum potassium < 2.0 mmol/L) and hypertensive [Blood Pressure (BP) = 180/115]. Conn's syndrome was confirmed by finding a raised serum aldosterone (1650 pmol/L; normal value < 440 pmol/L), suppressed renin (< 0.2 ng/ml/h; normal range: 0.2-2.5 ng/ml/h) and a left sided adrenal tumour (2.0 x 2.0 cm) on CT scanning. He was managed initially with IV potassium and insulin (initially 100 units daily) together with oral potassium, spironolactone 100 mg daily, lisinopril 20 mg daily and amlodipine 10 mg daily. After six days his potassium was 3.4 mmol/L and the IV potassium infusion was stopped. Twelve days later his fasting blood glucose, serum potassium and BP's were normal and the insulin and antihypertensive medications, apart from spironolactone, were stopped. He was discharged on spironolactone alone for four months during which time his blood glucose, blood pressure, and serum potassium levels remained normal and his HbA1c had fallen from 11.0 to 5.2%. He then

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underwent successful laparoscopic adrenalectomy and his serum aldosterone came down to 127pmol/L (within normal range). Histology confirmed the diagnosis of a Conn's tumour.

Conclusion: Although hyperaldosteronism per se predisposes to diabetes we suspect that this patient's rapidly reversible hyperglycaemia resulted primarily due to a failure of insulin secretion as a result of his severe potassium depletion.

Keywords: Conn's syndrome; severe hypokalaemia; reversible diabetes.

1. INTRODUCTION

Most cases of primary hyperaldosteronism (PA) are due to idiopathic adrenal hyperplasia and an adrenal tumour (Conn's syndrome) is rare. As many as 70 percent of patients with PA are normokalaemic at presentation and may have familial forms of the disease, such as glucocorticoid remediable aldosteronism (GRA) now referred to as familial hypertension type 1 (FH1) or non-glucocorticoid remediable disease FH type 2 [1-3]. The latter are considered to be rare in the West but recent observations suggest they may be common in Oman [4,5]. Familial Conn's syndrome is very rare and has been reported only as part of the multiple endocrine neoplasia syndromes.

In this report we describe a patient with Conn's syndrome and severe hypokalaemia who developed marked hyperglycaemia which was rapidly reversed with potassium replacement.

2. CASE REPORT

This 46 year old patient had been hypertensive for 14 years in spite of multiple antihypertensive medications. He was seen in the A&E unit in Sultan Qaboos University Hospital with a three-week history of polyuria, polydipsia and generalized ill health. He was found to have a random glucose of 40 mmol/L, potassium of 1.9 mmol/L and HbA1c of 11%. He weighed 102 kg (BMI = 30.1 kg/m²), with a BP of 180/115 mmHg. The ECG revealed runs of ventricular tachycardia. Clinically he did not have features of heart failure. There was a strong family history of hypertension: Both parents, who were consanguineous, and his only two siblings being affected. There was no family history of Type 1 or 2 diabetes mellitus. His investigation revealed a serum glucose of 40 mmol/L, potassium of 1.9 mmol/L, creatinine of 90 umol/L, sodium of 141 mmol/L, HbA1c of 11.0%, aldosterone of 1650 pmol/L (normal value < 440 pmol/L), and renin <

0.2 ng/ml/h. (normal range: 0.2-2.5). CT scan of the abdomen showed a left sided adrenal tumour, 2.0 x 2.0 cm (Fig. 1).

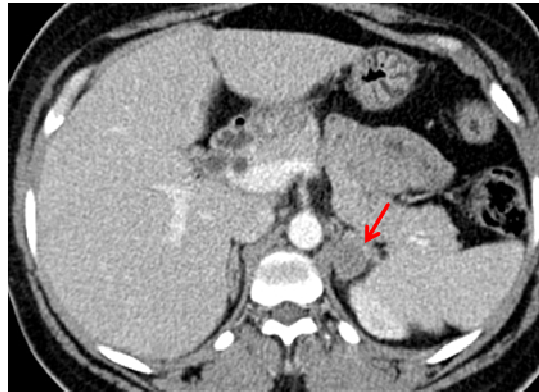


Fig. 1. CT abdomen showing a left adrenal tumour, 2 x 2 cm

After receiving 1 mg dexamethasone in the evening, his morning serum cortisol was suppressed to less than 50 nmol/l. His blood count, liver, thyroid and bone profiles were normal. A diagnosis of Conn's syndrome was suspected and he was started on an IV infusion of potassium (KCL), and insulin 100 Units (initially) daily together with oral KCL 600 mEq three times daily, spironolactone 50 mg twice daily, amlodipine 10 mg and lisinopril 20 mg daily after withdrawing blood for renin and aldosterone levels. The IV KCL infusion was stopped after six days when the serum potassium was 3.4 mmol/L, random blood glucose levels were around 8 mmol/L and the BP was 140/90 mm Hg. Insulin was discontinued after seven days and after 12 days all other medications except spironolactone were stopped. At this time the serum potassium was 4.7 mmol/L, blood glucose was 4 mmol/L and the BP was 110/70 mm Hg. He was discharged after 22 days and the treatment was continued with spironolactone alone until his surgery (Fig. 2).



Fig. 2. Surgical specimen showing a typical Conn's adenoma with a well circumscribed golden yellow tumour

2.1 Outcome and Follow Up

He received spironolactone 50 mg twice daily for four months. His blood glucose, serum potassium and BP's remained normal during follow up. The HbA1c fell from 11.0 to 5.2%. (Fig. 3). Laparoscopic adrenalectomy was carried out and his adrenal tumour was successfully removed (Fig. 2). He remained very well for the next 3 years but could not be followed because the patient went back to his home country.

3. DISCUSSION

The classical features of mineralocorticoid-induced disease; a poor response to multiple conventional antihypertensive medications, hypokalaemia with cardiac arrhythmias, polyuria,

and polydipsia [1] were seen in our patient. A most unusual feature was the severe reversible hyperglycaemia (blood glucose of 40 mmol/L) at presentation which was rapidly corrected after 12 days with potassium supplementation and spironolactone. The prevalence of diabetes mellitus is increased in hyperaldosteronism [6] which may predispose to the development of diabetes in one of two ways: Either by directly inhibiting insulin receptor function or by causing hypokalaemia. The former causes reduction of sensitivity to insulin and the latter results in impaired insulin secretion [7-9]. Although reversal of the diabetic state can occur after removal of a Conn's tumour [10] it can be hypothesized that severe hypokalaemia was the main cause of our patient's diabetes. This idea is supported by the rapid reversal of his diabetic state, normalization of the HbA1c level and the persistence of euglycaemia with correction of his serum potassium levels without the need for any diabetic medication. An additional interesting feature of this case was the family history of hypertension; Conn's syndrome is rarely familial and usually occurs in association with other syndromes. However, the presence of such a family history should alert the physician to the possibility of aldosterone-induced disease. Two small studies from Oman suggest that two-thirds of all such patients may have one or more forms of familial hyperaldosteronism [4,5].

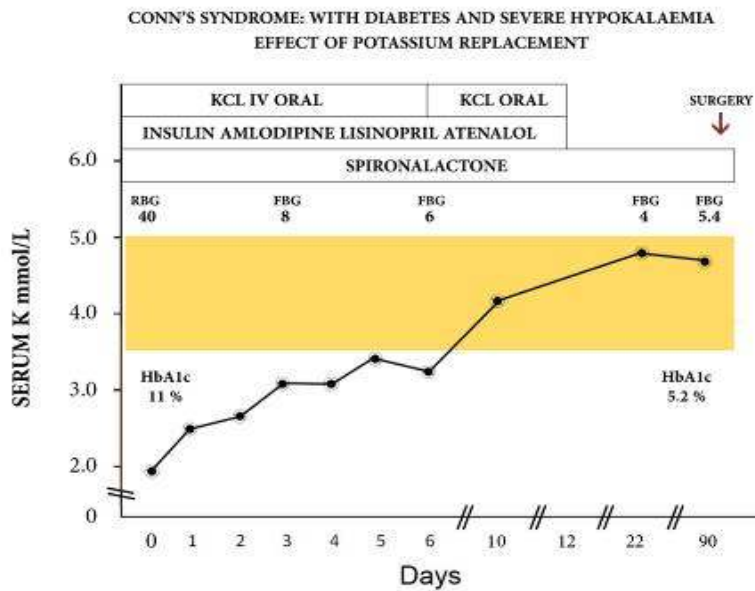


Fig. 3. Conn's syndrome with diabetes and hypokalaemia: Showing a rapid effect of treatment on the patients fasting blood glucose levels. All anti-diabetic medications were stopped on day 12. (RBG- random blood glucose, FBG- fasting blood glucose)

4. CONCLUSION

We present a unique case of Conn's syndrome with severe hyperglycaemia and hypokalaemia which corrected rapidly with potassium replacement and spironolactone. Impaired insulin secretion resulting from potassium depletion was the most likely cause of the hyperglycaemia.

CONSENT

Written informed consent was obtained for publication of the submitted article and accompanying images from the patient and his family.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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