

CLINICAL VIGNETTE

Hyperkalemia following Unilateral Adrenalectomy for Adrenal Adenoma

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Case Report

The patient is a 62-year-old man with a history of hypertension for three years. He was referred to nephrology for hypertension control, which was refractory to multiple lines of therapy. The patient reported systolic blood pressure reading as high as 190-200 mm Hg, and diastolic reading as high as 100-110 mm Hg despite receiving multiple antihypertensive medications.

The patient was asymptomatic and physical examination was remarkable only for elevated blood pressure systolic 188 mmHg and diastolic 104 mm Hg. Previous potassiums were low, ranging from 2.8 to 3.3 mEq/L. In the absence of diuretic therapy, the combination of hypokalemia and refractory hypertension raised concern for hyperaldosteronism and additional tests were ordered. Random serum aldosterone was 35 ng/dl, and random PRA was 0.2 ng/dl/hr. Kidney function was mildly reduced with a serum creatinine of 1.2 mg/dl and an estimated glomerular filtration rate of 57 ml/min. Electrolytes were normal except for a low serum potassium of 3.1 mEq/L. 24-hour urine catecholamines and renal ultrasound were normal. MRI scan of the adrenal glands showed a left adrenal mass measuring 1.7 X 2.1 X 1.8 cm and the patient was scheduled for resection.

The patient was treated preoperatively with spironolactone, potassium supplements and amlodipine with improved BP control and correction of the hypokalemia. He underwent left laparoscopic adrenalectomy without complication. Pathology confirmed a 1.6 cm benign adrenal cortical adenoma with normal remaining adrenal. The patient's BP remained well controlled postoperatively and his antihypertensive medications were discontinued.

The patient returned for follow-up 3 weeks after surgery, complaining of weakness and fatigue. He was tachycardic, with a heart rate of 108/min and blood pressure of 122/78 mm Hg sitting. The rest of his physical examination was unremarkable. Labs were remarkable for Na⁺ = 130, K⁺ = 6.6, Cl⁻ = 96, HCO₃⁻ = 25, blood urea nitrogen of 44 and

creatinine of 2.3 mg/dl and he was admitted to the hospital. Additional labs included normal cortisol levels and normal cosyntropin test. Serum aldosterone and PRA were 5.3 ng/dl and 2.1 ng/dl/hr respectively. The patient was treated with intravenous saline and a low potassium diet with increased fluid intake after discharge. On follow-up, his blood pressure ranged from 120-150/70-80 off antihypertensives. He remained free of hyperkalemia and his serum creatinine remained in the normal range.

Discussion

Primary aldosteronism (PA) is the most common cause of secondary hypertension associated with metabolic alkalosis and hypokalemia. Hypertension and hypokalemia are the usual presenting signs suggestive of over secretion of aldosterone and PA. The most common causes of PA are aldosterone-producing adenomas and bilateral idiopathic hyperaldosteronism. In patients with PA and adrenal adenoma, unilateral adrenalectomy generally corrects hypokalemia and cures hypertension in 60% to 87%¹⁻². Post adrenalectomy hyperkalemia has been reported in a subset of patients. In one study, 18 of 110 patients (16%) developed postoperative hyperkalemia, and six of these patients experienced prolonged hyperkalemia requiring treatment with mineralocorticoid therapy for 11-46 months³. Post adrenalectomy hyperkalemia in PA is believed to be secondary to hypoaldosteronism caused by suppression of the juxtaglomerular apparatus and the contralateral adrenal gland secondary to the volume-expanded state before unilateral adrenalectomy⁴. A retrospective analysis of 55 patients who underwent adrenalectomy for an aldosterone-producing adenoma, male gender, longer duration of hypertension, and higher preoperative serum creatinine were associated with greater risk of post-adrenalectomy hyperkalemia⁵. Some of these patients required prolonged mineralocorticoid treatment. Due to decreased intravascular volume associated with post-adrenalectomy hypoaldosteronism, acute post-op kidney injury can develop which can be treated with intravascular volume expansion.

Our patient was discharged from hospital after 24 hours of treatment with intravenous normal saline and a low potassium diet. He was followed in renal clinic and maintained a normal serum potassium level of 5.3 – 5.6 mmol/L with dietary modification. This case of post-adrenalectomy hyperkalemia illustrates the importance of meticulous monitoring of serum potassium to avoid life-threatening hyperkalemia after adrenalectomy.

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