Case of prolonged hypoaldosteronism after unilateral adrenalectomy for Conn's syndrome.

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Introduction

Aldosterone is a mineralcorticoid produced by the zona glomerulosa of the adrenal cortex. Aldosterone producing adenomas (APA) and bilateral adrenal hyperplasia account for 95% of cases of primary aldosteronism. The most effective treatment for an APA is an adrenalectomy. Complications following an adrenalectomy include the suppression of the contralateral zona glomerulosa causing low levels of renin and hypoaldosteronism which in turn leads to hyperkalaemia and hypotension.

This case illustrates a prolonged case of hypoaldosteronism following an adrenalectomy for Conns.

Case

This is a case of a 52 year old man referred due to refractory hypertension. His systolic pressure ranged between 170mmHg to 230mmHg despite being on four antihypertensive medications.

Examination showed no acromegaly or cushoingoid features. Biochemical markers showed a hypokalemia (k 2.8) with a normal TFTs, 24 hour urinary catecholamines and dexamethasone suppression test. He had a renin level of 0.5 and an aldosterone level of 1050. MRI adrenals revealed a 18cm x1.4cm nodule on the left adrenal gland (Figure 1). Venous sampling showed excess aldosterone production and a laprascopic adrenalectomy occurred.

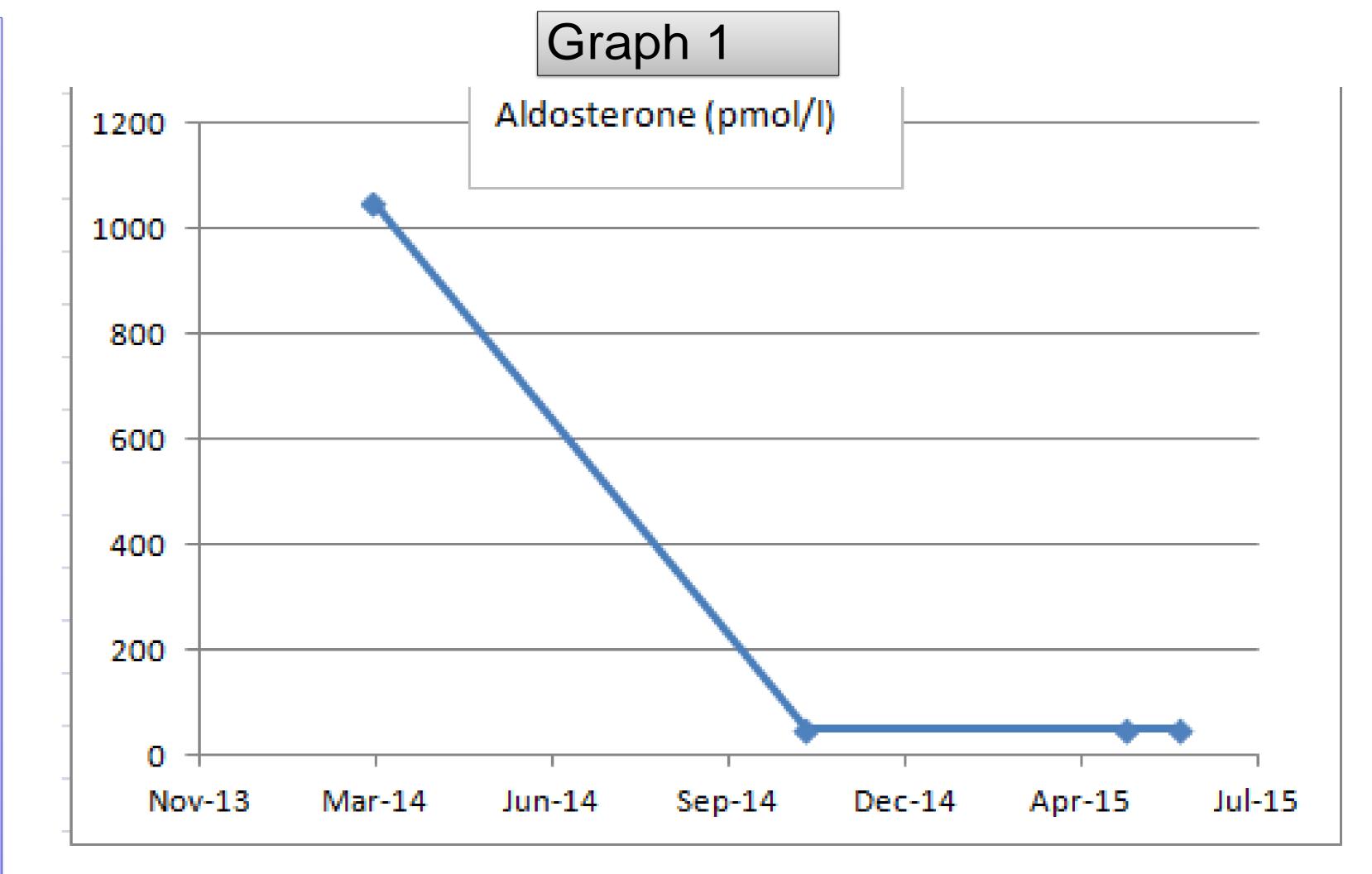
Post operatively he developed acute hypotension and acute kidney injury with a hyperkalemia. After discharge, he was found to have hypoaldosteronism with renin level 0.1 and aldosterone levels <100 (graph 1). He was commenced on fludrocortisone for 18 months and his AKI resolved. Whilst on fludrocortisone he underwent interval measurements of his electrolytes, renin and aldosterone.

Figure 1



Reference

Fischer. E, Hanslik. G, et al *Prolonged zona* glomerulosa insufficiency causing hyperkalemia in primary aldosteronism after adrenalectomy. J Clin Endocrinol Metab. 2012 Nov;97(11):3965-73.



Discussion

This case shows a post operative contralateral zona glomerolusa suppression causing hypoaldostronism with impaired renal potassium excretion leading to hyperkalaemia. Mechanisms include:

- Reduced renal perfusion following normalization of blood pressure post adrenalectomy can unmask renal impairment secondary to previous aldosteronism (via hypertension or via direct effects on fibrosis/inflammation)
- Delayed recovery of the remaining renin angiotensin-ZG function related to suppressed (secondary hypoaldosteronism) or elevated (primary hypoaldosteronism) renin levels.

Learning Points

To avoid this complication the ESE recommend:

- Stopping all potassium sparing agents
- Reducing anti hypertensive medications
- Plasma renin and aldosterone levels should be measured on day 1 post operatively
- 0.9% saline given without potassium replacement (unless hypokalaemia)
- A low potassium diet

In this case, this patient was on fludrocortisone therapy for 18 months. Predictive factors include age > 50, long duration of hypertensive, use of NSAIDS and size of adenoma.

