CLINICAL VIGNETTE

Hyperaldosteronism and Hypertension

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Case Report:

A 65-year-old man was referred for evaluation of hypertension and hypokalemia, with potassium level as low as 2.6 mmol/L. The patient's blood pressure was 163/102 despite multiple anti-hypertensive medications, including lisinopril 40 mg two times a day and spironolactone 25 mg a day. He was also receiving supplemental potassium for his persistent hypokalemia.

The patient's hypertension and hypokalemia raised the possibility of secondary hypertension due to hyperaldosteronism. Laboratory tests revealed a serum aldosterone of 56 ng/dL; plasma renin activity of 0.2 ngmLhr; serum creatinine 1.5 mg/dL; serum CO2 content of 29 mmol/L with normal range of 20-29 mmol/L; serum potassium 3.6 mmol/L; serum osmolality 293 mosm/kg; urine potassium 29 mmol/L; and urine osmolality 281 mosm/kg with normal urinalysis. The patient's medications, including lisinopril and sprinolactone, were discontinued 3 weeks prior to these tests and the patient was changed to amlodipine 10 mg daily and KCL 20 meq a day at the time of the tests. The patient's calculated aldosterone /renin and transtubular potassium gradient were 280 and 8.4 respectively.

The high aldosterone /renin ratio (280) and persistent hypokalemia with high transtubular potassium gradient in the setting of hypertension were consistent with primary hyperaldosteronism. Abdominal MRI revealed enlargement of left adrenal gland (1.6 x 1.3 cm) compatible with an adrenal adenoma. The right adrenal gland was unremarkable. The patient underwent selective adrenal vein sampling with a significant difference in aldosterone concentration between left and right adrenal veins, 563 ng/dL in left adrenal vein compare to17 ng/dL in right adrenal vein.

The selective adrenal veins sampling confirmed the left adrenal adenoma as a functional aldosterone producing adenoma and the cause of hypertension and hypokalemia in the patient. He underwent laparoscopic left adrenalectomy with normalization of hypokalemia and hypertension a few weeks after the surgery. He continues to maintain normal blood pressure off medications.

Discussion:

The triad of hypertension, hypokalemia, and metabolic alkalosis is suggestive of mineralocorticoid excess. The most common cause of mineralocorticoid excess responsible for hypertension and hypokalemia is primary aldosteronism. Drug- resistant hypertension, early-onset hypertension or hypertension with hypokalemia should raise the possibility of secondary cause of hypertension due to primary aldosteronism. Primary aldosteronism is the most common cause of hypertension due to an endocrine cause. Hypokalemia and metabolic alkalosis are not consistent findings and some patients, particularly those with bilateral adrenal

hyperplasia, can have normal serum potassium.

Hyperaldosteronism can be primary or secondary[1]. Primary hyperaldosteronism is mainly due to an aldosterone-producing adenoma/carcinoma or bilateral adrenal gland hyperplasia and are associated with high plasma aldosterone concentration and low plasma renin activity. Secondary causes of hyperaldosteronism include renovascular hypertension, aortic coarctation, and renin-secreting tumors. These are mainly associated with high plasma renin activity, and contrast with primary hyperaldosteronism in which marked suppression of renin release leads to very low plasma renin activity[2].

A suppressed renin measurement is of diagnostic importance to distinguish between primary and secondary forms of hyperaldosteronism. More importantly, a plasma aldosterone-renin ratio is used to screen for and detect primary aldosteronism. A combined plasma aldosterone-renin ratio above 30 and plasma aldosterone concentration above 20ng/dl have both a sensitivity and specificity of 90 percent for the diagnosis of aldosterone-producing adenoma[3].

In cases detected by plasma aldosterone-renin ratio, additional tests are recommended to definitively confirm or exclude the diagnosis[4]. The confirmatory tests include oral sodium loading, saline infusion, fludrocortisone suppression and captopril challenge test. To further evaluate the patients imaging studies CT or MRI scan are used. CT scan is used as the initial study. The findings on adrenal imaging study combined with information obtained from selective adrenal vein sampling are used to guide treatment decisions in patients with primary aldosteronism. The fact that nonfunctioning unilateral adrenal macroadenomas are not uncommon in patients older than 40 years[5], adrenal venous sampling should be considered in this group of patients. In patients younger than 40 years with unilateral adenoma and normal contralateral adrenal gland on scanning, it is reasonable to proceed to adrenalectomy[6].

Unilateral laparascopic adrenalectomy is recommended in patients with unilateral PA[7]. In patients with PA secondary to bilateral adrenal disease, idiopathic adrenal hyperplasia, bilateral aldosterone-producing adenoma, and patients who are unable or unwilling to undergo surgery, medical treatment with a mineralcorticoid receptor antagonist, preferably spironolactone is recommended[8].

In patients with bilateral adrenal disease, glucocorticoid-remediable aldosteronism, the lowest dose of glucocorticoid that can normalize blood pressure and serum potassium levels is recommended as the first line of treatment[9].

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