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Case Report Open Access

An Elderly Primary Hyperaldosteronism Case with Atypical Presentation

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Introduction

Essential hypertension is a systemic disorder with multi organ involvement. Although affecting many organ and systems, main detrimental effects of hypertension involve heart, kidneys and eyes. Retinopathy is the most common complication manifested through progression of essential hypertension which is more common and together with more severe progression especially in uncontrolled hypertension [1].

Primary hyperaldosteronism (PHA) is one of the most common causes of secondary hypertension. Although 10% of the hypertensive patients have PHA, this clinical entity is still not well known and underdiagnosed. Unilateral aldosterone synthesizing adenoma, also known as Conn's syndrome, is responsible from 65% of all hyperaldosteronism cases [2].

It is well known that left ventricular hypertrophy and cardiovascular complications are significantly increased among PHA patients. It is essential to define these patients since they have higher morbidity and mortality rates when compared with the patients of same age, sex and arterial blood pressure levels [3].

Here, we will present a case with recurrent photocoagulations for retinopathy diagnosed with PHA when hypokalemia was detected on his preprocedural consultation for geriatric assessment.

Case Details

Fundus fluorescein angiography was planned to be performed to a 70 year old male patient after detecting hypertensive retinopathy in his ocular examination. He was consulted for geriatric evaluation before the procedure. He was on perindopril treatment, but he wasn't taking diuretic therapy. He had laser photocoagulation seven times for retinopathy. In his physical evaluation his arterial blood pressure was 140/80 mmHg, had no ankle edema but severe retinopathy. He was evaluated further because he had no other acute or chronic illnesses other than regulated hypertension but had severe retinopathy. He was hospitalized for further evaluation after his serum potassium level was detected to be 2.6 meq/l. In his venous blood gas sampling pH was: 7.47 (7.35 – 7.45) , HCO3 level was: 34 mmol/L (22.2-28.3) , pCO2 level was: 47 mmHg (35-48) in accordance with metabolic alkalosis. 24 hour urinary potassium level was 106 mEq/24 h (25-125). His supine and upright posture serum renin and aldosterone levels were measured with suspicion of hyperaldosteronism. His serum renin (upright) level was: 0.16 ng/mL/h (0.48-4.88), serum renin (supine) level was: 0.17 ng/mL/h (0.3-1.9), serum aldosterone (upright) level was: 90.4 mg/dL (2.7-27.2), serum aldosterone (supine) level was: 134.9 mg/dL (1.3-14.5). Plasma aldosterone/renin ratio was: 565. Renal arterial Doppler ultrasonography for evaluation of causes of secondary hypertension showed no clinically significant narrowing. Left ventricular hypertrophy was detected in echocardiography. Also his anterior pituitary hormone levels and urinary catecholamine metabolites for pheochromocytoma evaluation were in normal range. These findings were suggestive of primary hyperaldosteronism and a computed tomographical evaluation was performed for imaging. Hypodense solid mass with 30 × 20 cm in size in axial plane compatible with adenoma was detected on medial limb of right adrenal gland (Figure 1). He was diagnosed with primary hyperaldosteronism secondary to adrenal adenoma. He was offered adrenal venous sampling and surgery but patient refused further evaluation and procedures. His ambulatory arterial blood pressure level was between 140-150 mmHg systolic and 90-80 mmHg diastolic during his hospitalization. 25 mg/d spironolactone was started and his arterial blood pressure level was in normal range during his follow up in hospital. His potassium levels rose to normal range and he was discharged to follow up as outpatient.

Discussion

PHA cases are usually detected in patients between third and sixth decades. Hypertension resistant to medical therapy is common among this population. Severe hypokalemia is found to be less than 20% in some researches in literature. Weakness and muscle cramps secondary to hypokalemia are also common. Retinopathy is usually an expected complication of uncontrolled hypertension and diabetes. Severe retinopathy necessitating photocoagulations for recurrent times in any hypertensive patient with controlled arterial blood pressure levels with only one antihypertensive medication is a condition that should prompt clinicians for further investigation of possible underlying secondary hypertension [4,5]. In our case the patient was 70 years of age. Although his serum potassium level was severely low he had no related symptoms secondary to hypokalemia. Besides having controlled arterial blood pressure levels with only one medications he had severe end organ damage and he had gone through laser photocoagulation for seven times.

Presence of hypertensive retinopathy, coexisting severe hypokalemia without any accompanying etiology like diuretic use or insufficient oral intake, low plasma renin activity high plasma aldosterone concentration with an increased aldosterone to renin ratio and detection of a adrenal adenoma in his imaging tests primarily led us to diagnosis of PHA. No further diagnostic evaluation like salt loading test were performed to confirm the diagnosis because clinical and laboratory findings already strongly suggested the diagnosis. Although patient refusal for further evaluation and procedures prevented precise medical diagnosis of PHA either by adrenal venous sampling or surgical procedure, rising serum potassium levels to normal range and

regulation of arterial blood pressure levels after a trial with spironolactone (an aldosterone antagonist) supported the diagnosis.

As a result it should be kept in mind that any patient with hypertensive retinopathy necessitating medical intervention, with accompanying hypokalemia, despite not having resistant hypertension, even in advanced age, should be evaluated for PHA which is the most common cause of secondary hypertension.

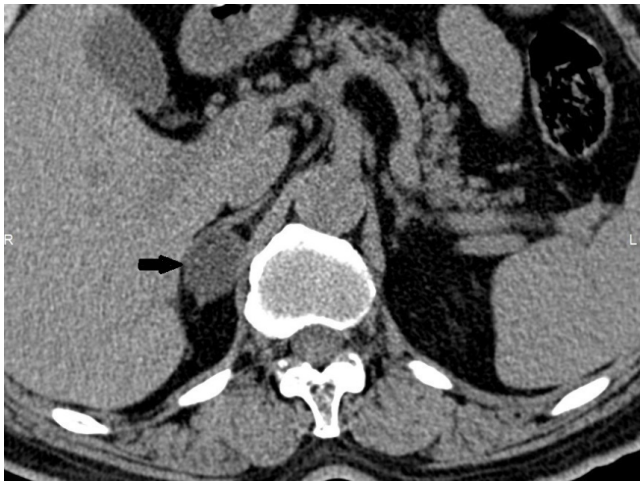

Figure 1: A hypodense lesion 30 × 20 cm size in medial limb of right adrenal gland in computed tomography is shown.

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