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Case Report

A Case Report on Uncontrolled Arterial Hypertension as an Underlying Cause of Conn´s Syndrome

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Abstract

A 24-year-old woman who consulted for uncontrolled high blood pressure. Four years ago, was diagnosed with high blood pressure and was treated with many medications without achieving adequate control. On treatment with irbesartan, hydrochlorothiazide, amlodipine. Patient has not family history. Blood pressure of 180/110mmHg was found upon evaluation. Examinations showed evidence of hypokalemia and normal renal function. Chest X-rays and electrocardiogram were normal. Ultrasound Doppler of renal arteries without alterations. Blood aldosterone was measured with values of 22ng/dl and an abdominal tomography showed the presence of a left adrenal adenoma. Spironolactone was orally given at a dosage of 25mg every day, titration every week, reaching a dose of 100mg every day, an education plan with a salt-restricted diet and adequate physical activity was provided. So, it is concluded that Primary aldosteronism is the primary endocrinological cause of secondary hypertension and only 30% of patients have hypokalemia.

Keywords: High blood pressure; Primary hyperaldosteronism; Aldosterone.

Introduction

Primary hyperaldosteronism is the main endocrinological cause of secondary arterial hypertension, 35% of which is secondary to an adrenal adenoma (Conn syndrome). Patients with this disease are usually treated with multiple medications to control blood pressure without being able to achieve it. It is characterized by having low levels of renin and high levels of aldosterone and sometimes low blood potassium levels, its diagnosis requires a high clinical suspicion; once the diagnosis is confirmed, mineralocorticoid antagonists should be started as first-line treatment. The case of a patient is presented with hypokalemia and multiple medications to control blood pressure.

Case report

A case of 24-year-old female patient from Honduras, with a history of arterial hypertension 4years ago is presented. The

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patient consulted for a 3-month history of headache located in the occipital region, predominantly in the morning, throbbing, without irradiation, intensity 5-6/10, not relieved by acetaminophen. Patient has consulted on many occasions without BP control despite optimal doses of antihypertensive medications. Enalapril, olmesartan, amlodipine, hydrochlorothiazide, furosemide, irbesartan were prescribed progressively, maintaining blood pressure levels of 150/90mmHg with up to three medications together at optimal doses. Patient denied blurred vision, epigastric pain, fever, cough, dyspnea, chest pain, profuse sweating, palpitations. In the family history it was not found, surgery, trauma or allergies. Vital signs revealed respiratory rate of 17breaths/minute, blood pressure of 150/100mmHg, pulse rate of 74/minute and temperature of 37.1 °C. Other physical examinations were unremarkable. Initial laboratory examination showed hypokalemia and normal kidney function tests (Table 1). Chest X-ray, electrocardiogram and urine test were normal.

Type	Value	Unit
Albumin	3.9	g/dL
BUN (Blood Urea Nitrogen)	11	mg/dL
Chloride	104	mEq/L
Creatinine	0.8	mg/dL
Hb (hemoglobin)	13.2	g/dL
Hct (hematocrit)	40	%
Platelet count	327000	cells/mm^3

Table 1: Initial laboratory examination showed hypokalemia and normal kidney function tests.

Renal arterial Doppler USG was performed with the result of normal vascular structures, low resistance record and resistance indices without signs of dysfunction, with this result renal artery stenosis was ruled out and hyperaldosteronism studies were performed because the patient met criteria (Figure 1). All antihypertensive drugs were omitted, and hydralazine was given orally at a dosage of 25mg every 6hours. Hypokalemia was corrected and then blood aldosterone and plasma renin activity were measured (Table 2).

Type	Value	Unit
Potassium	3.3	mEq/L
Sodium	142	mEq/L
WBC (total white blood cells)	7200	cells/mm^3
Aldosterone, serum	22	ng/dL
Plasma renin activity; PRA	0.1	ng/mL/hour

Table 2: Aldosterone and PRA activity after correcting hypokalemia.

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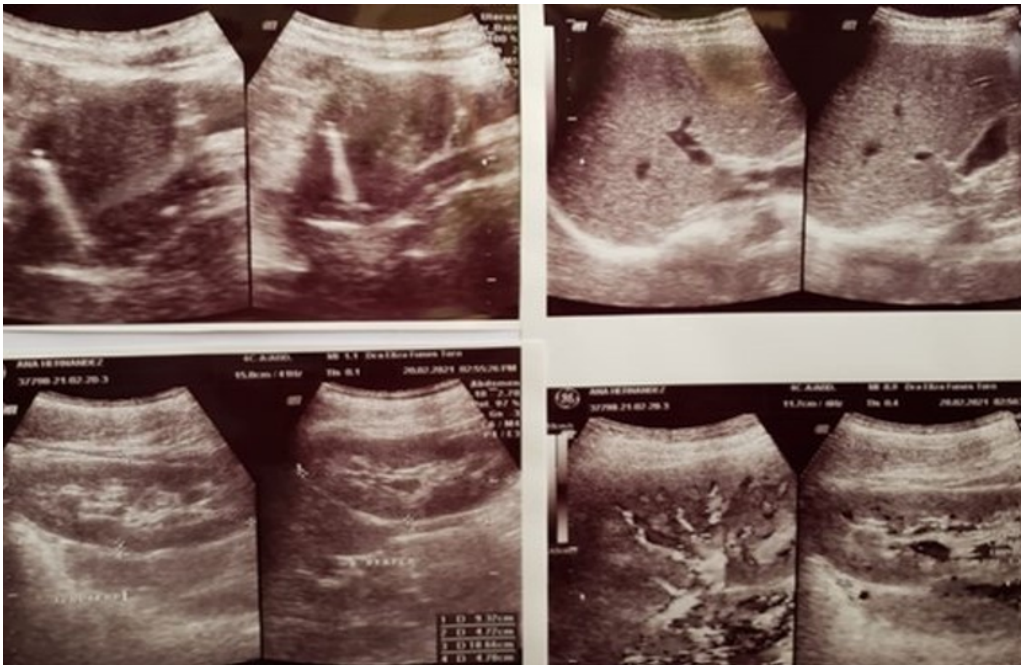

Figure 1: Renal arterial Doppler USG.

Due to the criteria of high suspicion of hyperaldosteronism, no confirmatory test was performed, and an abdominal axial tomography was performed with emphasis on the adrenal gland, observing the presence of a small iso-dense nodule with heterogeneous enhancement after the administration of contrast with measurements of 13x10.7x7.3mm suggestive of adenoma in the left adrenal gland. With the previous data, the cause of hyperaldosteronism was confirmed, obtaining the diagnosis of Conn's Syndrome. Hydralazine was omitted and spironolactone was started at a dose of 25mg orally every day, titration every week, reaching a dose of 100mg every day, an education plan with a salt-restricted diet and adequate physical activity was provided. At 4weeks, the patient had optimal blood pressure levels, 120/70mmHg, and no headache.

Discussion

Secondary (or remediable) hypertension is elevated BP due to a specific cause [1]. The proportion of patients with secondary hypertension who can eventually be cured is far greater than those with primary hypertension. This is particularly important in young hypertensive people (especially in children and adolescents) [2], who are more likely to harbor nearly all types of secondary hypertension with the important exception of atherosclerotic renovascular hypertension. The worldwide prevalence of hypertension is 30% that has been epidemiologically studied and well documented in literature [3].

Primary aldosteronism (PA) is characterized by unprovoked hypokalemia, hypertension, and majorly by inappropriate aldosterone production. Imbalance in the production of aldosterone is due to the non-activation of the renin angiotensin cascade. Excess aldosterone causes excretion of potassium at the distal convoluted tubule instead of retained sodium that results in the hypokalemia [4].

Primary aldosteronism is divided into two subtypes:

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1. Aldosterone-producing adenoma (APA): It is referred to a unilateral tumor of the adrenal cortex that cause autonomous secretion of aldosterone.
2. Bilateral adrenal hyperplasia (BAH): It is a type of PA excessive amounts of aldosterone is secreted by both the adrenals in response to angiotensin-II [4].

10% cases of the hypertension can be due to PA following the screening test of aldosterone/renin ratio (A/RR) or plasma aldosterone concentration (PAC) to plasma renin activity (PRA) (PAC/PRA). The screening policy included the screening test of the all the patients. The test revealed that majority of PA patients are normokalemic and only 9–37% of the patients show hypokalemia. The recent guideline of the Endocrine Society recommends the screening for high-risk groups. The Screening involves:

- Hypertension and sleep apnoea
- First-degree relatives of Hypertensive patients with PA
- Adrenal incidentaloma and Hypertension
- Hypertension and family history of early-onset hypertension or cerebrovascular accident at <40years of age
- Blood pressure (BP) >150/100mmHg to be sustained obtained three times on three different days
- Diuretic-induced or spontaneous hypokalaemia and Hypertension
- Hypertension (BP >140/90mmHg) resistant to three conventional antihypertensive drugs, or controlled BP when taking four or more antihypertensive medications

Case-detection testing can be accomplished by random morning paired measurements of blood plasma renin activity (PRA) and plasma aldosterone concentration (PAC). Before performing diagnostic studies, hypokalemia is optimal as the serum level of potassium are restored to normal as it reduces the secretion of aldosterone. A positive screening test for PA is PRA level less than 1ng/mL/hr with a high plasma aldosterone >15ng/dl.

A verificatory evaluation must be carried out to illustrate that high salt diet is not appropriate for aldosterone secretion. Endocrine Society Guideline put forward four different confirmatory tests generally that involves captopril challenge tests, fludrocortisone suppression, oral salt loading and saline infusion [4,5]. In spite of voluntary hypokalemia as well as hypertension, it is recommended that confirmatory testing is not at all essential that also imperceptible renin along with plasma aldosterone concentration >20ng/dl. If verificatory evaluation shows positive result, primary study of 2–3mm cuts of adrenal glands must be carried out by high-resolution CT scan for subtype analysis including the disbar large masses that ought to illustrate the adrenocortical carcinoma [6-8]. However, Unilateral laparoscopic adrenalectomy is a magnificent therapy is an alternative for the patient with the APA and unilateral hyperplasia [9]. PA conceivably cured medically if therapy involves the mineralocorticoid receptor blockade (spironolactone and epleronone). Moreover, avoiding tobacco, maintaining of the ideal body weight, a regular aerobic exercise and sodium-restricted diet (<100mEq sodium per day) may grant outstanding towards the success of the pharmacologic therapy.

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Conclusion

Primary aldosteronism is the first endocrinological cause of secondary hypertension. Only 30% of patients with primary aldosteronism have hypokalemia Case-detection testing positive for primary aldosteronism always requires a confirmatory test; the exception for confirmatory testing involves patients with hypertension, spontaneous hypokalemia, and marked primary aldosteronism (e.g., PAC >20ng/dL and PRA <1.0ng/mL per hour) Mineralocorticoid antagonists are the treatment of choice for primary hyperaldosteronism. All young hypertensive patients with high blood pressure despite pharmacological treatment, with a family history of cerebrovascular events at an early age, should undergo studies for primary hyperaldosteronism.

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