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Case Report

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Primary aldosteronism: An underdiagnosed cause of hypertension

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Abstract

Primary aldosteronism (PA), the most common form of secondary hypertension, can be either surgically cured or treated with targeted pharmacotherapy. PA is frequently undiagnosed and untreated, leading to aldosterone-specific cardiovascular morbidity and nephrotoxicity. Thus clinicians should perform case detection testing for PA at least once in suspected patients with hypertension. We report a case of a young man presented with hypertension and recurrent hypokalemia. This case highlights the need for clinicians to orient themselves with the clinical and biochemical features of PA to avoid delays in diagnosis and specific treatment of PA to reduce morbidity and mortality. *[J Assoc Clin Endocrinol Diabetol Bangladesh, July 2023; 2 (2): 79-81]*

Keywords: Primary aldosteronism, recurrent hypokalemia, adrenal adenoma

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Introduction

Primary aldosteronism (PA) is characterized by increased aldosterone secretion and suppressed renin activity which would clinically manifest as hypertension, and hypokalemia and was first described by J. W. Connin 1955.[1] PA is one of the most prevalent forms of secondary hypertension.[2] The prevalence of PA is 5-13% of all patients with hypertension.[3] PA is frequently undiagnosed and untreated leading to aldosterone-specific cardiovascular morbidity and nephrotoxicity.[4] Classically PA presents as hypertension and hypokalemia but normokalemic hypertension is the most common presentation. Aldosterone-producing adenoma (APA) and bilateral idiopathic hyperaldosteronism (IHA) are the two most common subtypes of PA, less common causes are due to primary (unilateral) adrenal hyperplasia, aldosterone-producing adrenocortical carcinoma or familial hyperaldosteronism.[5] Over the past few decades, there has been considerable progress in understanding the genetics, pathophysiology, diagnosis, prognosis, and therapy of PA. Screening for PA is done by measuring the plasma aldosterone renin ratio.[6] Screening for PA is, however, only infrequently performed outside of specialized hypertension centers.[7] In patients suspected of having PA, the diagnosis can be confirmed by several techniques, including an oral salt loading suppression test, saline infusion test, fludrocortisone suppression test, or captopril challenge test.[8] Once the diagnosis is confirmed, a CT scan and/or adrenal venous sampling is used in patients with PA to determine whether unilateral adrenalectomy is likely beneficial.[6]

Case Summary

A 28 year-old-male presented with recurrent attacks of weakness in all four limbs and hypertension for two years. During the initial attack, he was found to be hypokalemic and hypertensive. His initial blood pressure was 170/110 mm-Hg and was treated with olmesartan and then cilnidipine with prazosin but with poor control (Figure-1). During this period he had several attacks of hypokalemia. He was hospitalized twice and managed with intravenous potassium. The weakness had no relation with taking carbohydrate meals and alcohol. He had no family history of hypertension and hypokalemia. Examination findings included uncontrolled blood pressure, and reduced muscle power (MRC grade 3/5) in all four limbs, with otherwise normal findings. Apart from hypokalemia,

serum corrected calcium, creatinine, urine routine examination, liver function test, and arterial blood gas analysis were normal. The USG of the whole abdomen and echocardiogram were normal. Serum aldosterone was elevated and plasma renin was suppressed as well as plasma aldosterone after saline infusion was not suppressed (12.4 ng/dl). The CT scan of the adrenal glands showed a small (17×15) mm rounded lesion seen in the adjacent/ lateral limb of the left adrenal gland (adrenal adenoma) (Figure-2).

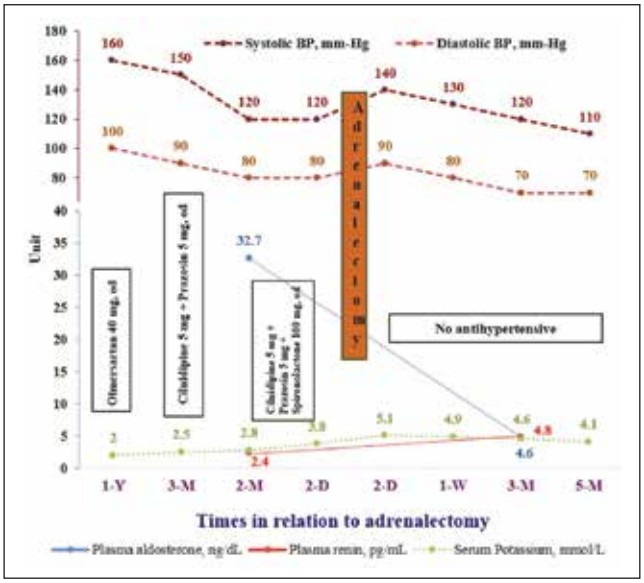

Figure-1: Blood pressure, antihypertensive, aldosterone, renin, and potassium levels in relation to adrenalectomy
Y- year, M- month, D- day

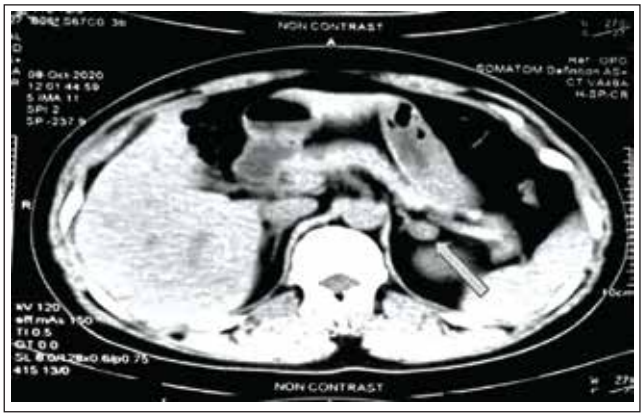

Figure-2: CT scan of the abdomen is showing a left-sided adrenal adenoma

After correction of hypokalemia and control of hypertension, he underwent laparoscopic left adrenalectomy and histopathology showed adrenal adenoma. After the operation, his blood pressure and potassium levels became normal without any medication (Figure-1).

Discussion

PA is one of the commonest causes of secondary hypertension and commonly occurs between 30 and 50 years of age. The uncontrollable synthesis of aldosterone leads to increased sodium reabsorption, kaliuresis, and renin suppression.[1,3]

All of them produce arterial hypertension and hypokalemia, which affects target organs (heart, kidneys, brain) more severely than essential hypertension, matched for age, blood pressure, and duration of hypertension.[9] PA is usually associated with hypokalemia and arterial hypertension due to excessive aldosterone but hypokalemia is not common and present only in 7% to 38% of cases.[6] PA may present as recurrent flaccid quadriparesis due to hypokalemia in a few of the patients.[6] Our patient presented with recurrent flaccid paralysis with young onset hypertension. In efforts to conserve medical costs and limit the consequences of false-positive case detection testing, the Endocrine Society guidelines on PA, recommend testing high-risk groups for PA.[6]

These groups include (i) sustained blood pressure above 150/100 mm-Hg (ii) resistant hypertension; (iii) hypertension and spontaneous or diuretic-induced hypokalemia iv) hypertension and adrenal incidentaloma (v) hypertension and sleep apnea; (vi) family history of early onset hypertension or cerebrovascular accidents at a young age (<40 years); and (vii) all hypertensive first-degree relatives with PA.[6] Our patient had hypertension of more than 150/100 mm-Hg and hypokalemia.

The diagnostic approach of PA can be summarized in three main steps i) case detection by plasma aldosterone concentration (PAC), plasma renin activity (PRA), and calculation of PAC/PRA Ratio calculation ii) confirmation of PA is based on oral sodium load test, saline infusion test, fludrocortisone suppression test and captopril challenge test iii) subtype testing by adrenal CT scan and may need adrenal venous sampling.[6]

In our patient's case detection is positive with elevated plasma aldosterone with low renin, confirmation by unsuppressed plasma aldosterone after saline infusion and subtyping by adrenal CT scan. We did not need adrenal venous sampling due to the young age of the patient with unilateral adrenal adenoma.

Surgery is the preferred treatment for patients with

unilateral disease. A marked reduction in aldosterone secretion and correction of hypokalemia in almost all patients is seen after unilateral adrenalectomy. Blood pressure control improves in all patients and normalization is around 30% of patients.[10] After surgery our patient is normotensive and normokalemic without any drug.

Conclusions

Diagnosis of primary aldosteronism is important because it can be either surgically cured or treated with targeted pharmacotherapy and reduce aldosterone-specific morbidity and mortality. This case highlights the need for clinicians to orient themselves with the clinical and biochemical, features of PA to avoid delays in diagnosis and specific treatment of PA to reduce morbidity and mortality.

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Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.
Ethics Approval and Consent to Participate
Written informed assent was taken from the patient.

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