IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 3 Ser. 5 (March. 2024), PP 30-32
www.iosrjournals.org

An Unusual Case Of Underdiagnosed Secondary Hypertension: Conn's Syndrome

Mangukiya Axay Kantilal[1], Dhruv Gupta[1], Challapali Aditya[2]

[1,2] *Department Of General Medicine*

Vydehi Institute of Medical Sciences And Research Centre, Bengaluru

Abstract:
Conn's syndrome is a rare condition which is caused by increased aldosterone production with the culprit commonly being an aldosterone secreting adenoma of the adrenal gland. Conn's syndrome account for 0.05% to 2% of patients with secondary hypertension. Compared to primary hypertension, primary hyperaldosteronism causes more end-organ damage and is associated with excess cardiovascular morbidity, heart failure, stroke, non-fatal myocardial infarction, and atrial fibrillation. Cardiovascular morbidities can be mitigated by the means of simple and readily available screening methodologies and targeted therapies which in turn also improve blood pressure control. Despite these imperatives, screening rates for primary aldosteronism are low and mineralocorticoid receptor antagonists are underused for hypertension treatment. After the evidence of the prevalence of primary aldosteronism and its associated cardiovascular morbidities is summarized, a practical approach to primary aldosteronism screening, referral and management should be performed. Therefore, all physicians who treat hypertension should routinely screen appropriate patients for hyperaldosteronism.

Keywords:*conn's syndrome, primary hyperaldosteronism, secondary hypertension, hypokalaemia.*

Date of Submission: 13-03-2024 Date of Acceptance: 23-03-2024

I. Introduction:

One of the endocrine causes for secondary hypertension is primary hyperaldosteronism and accounts for 0.05-2%of patients with secondary hypertension [1]. However, recent studies have shown that using the plasma aldosterone/plasma renin activity ratio as a screening test followed by aldosterone suppression confirmatory testing has resulted in much higher prevalence estimates (51% of all hypertensives) for primary aldosteronism. [2]

J.w. Conn first described conn's syndrome in 1955 in a patient with a unilateral aldosterone producing adenoma.[3]

It is characterized by increased aldosterone production, suppressed plasma renin activity (pra), hypertension, hypokalaemia, and metabolic alkalosis. The term primary hyperaldosteronism is used to describe conn's syndrome and other aetiologies of primary hypersecretion of aldosterone. These include; bilateral adrenal hyperplasia, idiopathic hyperaldosteronism, in which the adrenals appear normal, and glucocorticoid-remediable aldosteronism in which acth stimulates aldosterone production. Rarely, large adrenocortical carcinomas, adrenal embryologic nest neoplasms within the kidney and ovary secrete aldosterone. Hypokalaemia in a patient with hypertension who is not on diuretics should be investigated for primary aldosteronism [4]. Ventricular fibrillation is the most common cause of sudden death. Coronary artery disease, cardiac valvular or myocardial diseases, and non-cardiac abnormalities may also lead to ventricular fibrillation.[5]

II. Case Report:

A 40-year-old female, hailing from west bengal, with a history of hypertension is presented. She was consulting for hypertension for 5 years on medications. The patient consulted for 2 months history of lower limb weakness, intermittent palpitations and dry mouth which was not relieved on medications to a general physician. Despite anti-hypertensive measures, her blood pressure was consistently elevated, therefore, she consulted a general physician for further evaluation and management. The patient has consulted on many instances for elevated blood pressure despite adequate anti-hypertensive medications. The patient was initially prescribed

cilnidipine, amlodipine, aldactone and prazosin and was maintaining a blood pressure of 140/100 mm of hg. The patient denied complaints of headache, diaphoresis, abdominal pain, excessive weight gain and purple striae on the abdomen. There was no history of similar complaints amongst immediate family members and close relatives. The patient denied history of surgery, trauma, or allergies in the family. Vital signs revealed a blood pressure of 130/110 mmhg, pulse rate of 80 beats per minute, oxygen saturation of 98% on room air and temperature of 98.2 degrees fahrenheit. Other physical examination findings were unremarkable.

Initial laboratory evaluation revealed hypokalemia (table-1), normal hematological parameters, and normal renal function (table-2).

Ultrasonography of the abdomen and pelviswas performed which revealed minimal free fluid in the pelvic cavity with normal kidney size, shape and corticomedullary differentiation.

A contrast enhanced spiral axial scans of the abdomen were obtained on an 80 slice mdct scanner followed by coronal reformats which revealed a bulky left adrenal gland with a hypodense enhancing lesion in the body measuring 1.8 x 1.5 x 2.0 centimeters suggestive of an adrenal adenoma. (figure- 1)

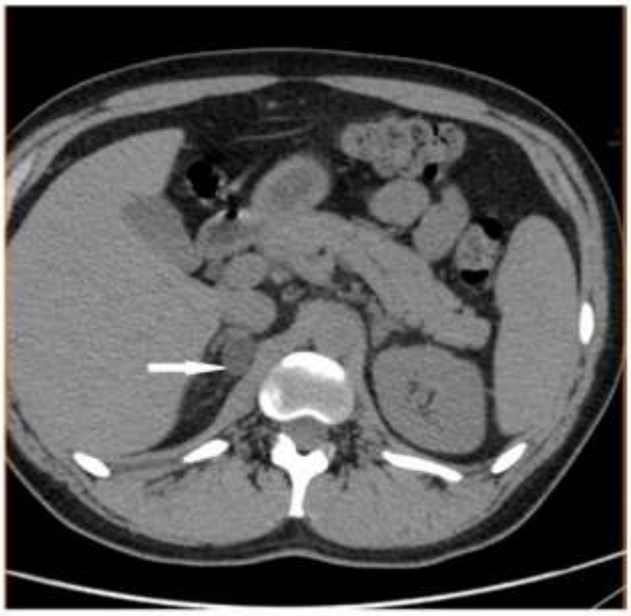

Figure-1. Cect of the abdomen showing a left sided adrenal mass

In view of a high suspicion of hyperaldosteronism, serum direct renin levels and aldosterone levels were obtained (table-3) which were found to be low (1 microiu/ml) and high (37.7 ng/dl) respectively. An elevated aldosterone to renin ratio of 37.7 suggested primary hyperaldosteronism.

Table-3:

Type	Results
Direct renin levels	1 microiu/ml
Aldosterone levels	37.7 ng/dl
Aldosterone to renin ratio	37.7

With the previous data and cect of the abdomen the cause of primary hyperaldosteronism was confirmed thereby obtaining a diagnosis of conn's syndrome.

Following the diagnosis of conn's syndrome, the patient was posted for laparoscopic left adrenalectomy. The pre-operative, peri-operative and post-operative periods were uneventful and there were no post-operative complications noted. Following the procedure, the patient was advised to withhold antihypertensive medications, and to continue anti-hypertensive medications if the blood pressure was above 140/90 mmhg. The patient was provided with an education plan of a salt-restricted diet and adequate physical activity. After 2 weeks of follow-up consultations, the patient had a resting blood pressure of 120/70 mmhg with no complaints of lower limb weakness or palpitations.

III. Discussion:

This case highlights a patient with what was thought to be essential hypertension but on proper investigation was discovered to have hypokalaemia and a unilateral adrenal mass. She presented with lower

limb weakness, intermittent palpitations, hypertension, and dry mouth. Laboratory examinations showed a suppressed renin level and an elevated serum aldosterone level.

Conn's syndrome is characterized by increased aldosterone production, suppressed plasma renin activity (pra), hypertension, hypokalaemia and metabolic alkalosis. The term primary hyperaldosteronism is used to describe conn's syndrome and other aetiologies of primary hypersecretion of aldosterone. These include; bilateral adrenal hyperplasia, idiopathic hyperaldosteronism in which the adrenals appear normal and glucocorticoid-remediable aldosteronism in which acth stimulates aldosterone production.

Patients with conn's syndrome present with hypokalaemia which may be severe. However, normal serum potassium levels do not exclude primary hyperaldosteronism, but in these patients, there is usually a history of low salt intake and salt loading usually unmasks the hypokalaemia [6]. A diagnosis of conn's syndrome is made with biochemical and imaging studies. A plasma aldosterone/renin activity ratio of >20 with a plasma aldosterone level equal to or more than 15ng/dl is highly suggestive and is the screening test of choice [7]. It has been suggested that captopril administration may optimize the pra test [8]. The most commonly used confirmatory test is a 24-hour urine aldosterone level obtained after 3 days of salt loading. The patient can be instructed to maintain a sodium intake of at least 200 meq/d (one teaspoon of salt 3 times daily) for 3 days. A 24-hour aldosterone excretion rate of greater than 14 mcg (with a concomitant 24-h urine sodium >200 meq) is diagnostic of primary hyperaldosteronism [9]. Postural studies are cumbersome and not routinely done. The salineinfusion test in which 2 litres of isotonic normal saline are infused over 2-4 hours and blood samples for aldosterone collected before and after the infusion can also be done. In patients with essential hypertension, plasma aldosterone reduces. While in patients with adenoma or idiopathic hyperaldosteronism, the plasma aldosterone fails to get suppressed. Thisdistinguishes primary aldosteronism from low-renin essential hypertension.

Abdominal ct scan is the imaging procedure of choice. Overall, ct scanning has a sensitivity of 67-85% in patients with primary hyperaldosteronism. Adrenal venous sampling following co-syntropin stimulation is used in patients with equivocal ct scan findings.

The drug of choice is spironolactone in doses of up to 400 mg/day. Hypokalaemia tends to correct with adequate doses of spironolactone and potassium supplementation may not be required. Eplerenone is a newer mineralocorticoid antagonist with less anti-androgen and anti-progesterone effects and may be preferable. Amiloride, a potassium sparing diuretic may be used in patients who are intolerant of mineralocorticoid antagonists but it is not very effective and it lacks the mineralocorticoid receptor antagonist benefits. Unilateral laparoscopicadrenalectomy is the surgical procedure of choice in patients with conn's syndromeand its long-term cure rates average 69%. Unilateral adrenalectomy is a reasonable therapeutic option when a solitary unilateral macroadenoma (<1 cm) and normal contralateral adrenal morphology are found on ct in a young patient (<40 years old) with primary aldosteronism.

In our patient, the presence of low renin levels, elevated aldosterone levels, hypokalaemia, hypertension, and the presence of a unilateral adenoma strongly suggested the diagnosis of conn's syndrome. In addition, the patient has done well following left adrenalectomy. Post-surgical intervention, the patient recovered symptomatically and the hypertension came under control with minimal anti-hypertensive medications following which anti-hypertensives were discontinued.

IV. Conclusion:

This case points out the need for early evaluation of the young hypertensive patient especially in cases of a high suspicion of an adrenal aldosterone releasing adenoma. Early identification of the condition can lead to appropriate treatment intervention which include mineralocorticoid receptor antagonists and surgical removal of the adrenal adenoma. Such interventions will help in normalization of blood pressure levels and serum potassium levels thereby preventing future cardiovascular complications. Therefore all physicians who treat hypertension should routinely screen appropriate patients for hyperaldosteronism.

Refferences:

[1] Kaplan Nm. Hypokalemia In The Hypertensive Patient, With Observations On The Incidence Of Primary Aldosteronism. Ann Intern Med 1967; 66:10791090
[2] Rossi E, Regolisti G, Negro A, Sani C, Davoli S, Perazzoli F. High Prevalence Of Primary Aldosteronism Using Post-Captopril Plasma Aldosterone To Renin Ratio As A Screening Test Among Italian Hypertensives. Am J Hypertens2002;15:896902.
[3] Conn Jw. 1955 Presidential Address. Part I, Painting Background. Part Ii, Primary Aldosteronism, A New Clinical Syndrome. J Lab Clin Med 1955; 45
[4] Higham Pd, Adams Pc, Murray A, Campbell Rw. Plasma Potassium, Serum Magnesium And Ventricular Fibrillation: A Prospective Study. Q J Med 1993; 86: 609617.
[5] Falase A. O. Akinkugbe O.O The Cardiovascular System In: A Compendium Of Clinical Medicine 3rd Ed. Spectrum Books 2007:21.
[6] Gill Jr. Primary Aldosteronism: Strategies For Diagnosis And Treatment. The Endocrinologist. 1991; 1:365-369.
[7] Don Br, Lo Jc. Endocrine Hypertension In: Gardner D.G, Shoback D Eds. Greenspan's Basic And Clinical Endocrinology, 8th Ed. Philadelphia; Mcgrawhill 2007.

[8] Alper A, Ertan O, Izzet E, Arda S. Nese C. Adrenal Adenoma Presenting With Ventricular Fibrillation. Tex Heart Inst J 2005; 32:85-87.
[9] Castro Ol, Yu X, Kem Dc. Diagnostic Value Of The Post-Captopril Test In Primary Aldosteronism. Hypertension 2002; 39:935938
[10] Castro Ol, Yu X, Kem Dc. Diagnostic Value Of The Post-Captopril Test In Primary Aldosteronism. Hypertension 2002; 39:935938