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Primary hyperaldosteronism without suppressed renin: A case report

Sarath Bhaskar S

Junior Resident in General Medicine, Sree Balaji Medical College Hospital, Chennai

Corresponding author email: sarath_bhaskar1@yahoo.com

V Padma

Professor in General Medicine, Sree Balaji Medical College Hospital, Chennai

Murugaraj R

Junior Resident in General Medicine, Sree Balaji Medical College Hospital, Chennai

B Abhilash Nair

Junior Resident in General Medicine, Sree Balaji Medical College Hospital, Chennai

Kannan Meera Devi

Junior Resident in General Medicine, Sree Balaji Medical College Hospital, Chennai

Saketh Ramineni

Junior Resident in General Medicine, Sree Balaji Medical College Hospital, Chennai

Abstract---Primary Hyperaldosteronism characterizes elevated plasma and urinary aldosterone with suppression of plasma renin activity. Suppression can be due to sodium retention which is aldosterone dependent and extracellular volume expansion. Here a 29 year old male presented with uncontrolled hypertension, on work up he was diagnosed to have primary Hyperaldosteronism with normal plasma renin level. So with the available medical data on the patient as well as the publications on aldosterone/renin association in primary hyperaldosteronism was reviewed here to explain the rare finding.

Keywords---hypertension, hypokalemia, aldosteronism.

Introduction

Case Report

A 29-year old male, came with complaints of pain and swelling in the left knee for 2 days, patient had a alleged history of skid and fall from the bike and sustained injury to the left knee 2days back and also had a history of difficulty in walking and standing. No history of loss of consciousness/ENT bleed/Head injury/Chest Injury. No history of fever/cough/vomiting/loose stools. No history of any other comorbidities. Not a smoker/alcoholic .Normal bowel and bladder habits.

Examination

On examination patient was obese. He was conscious, oriented and afebrile. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy or edema. His Blood Pressure was 190/130mmHg, Pulse Rate was 98bpm and his saturation were 99% at room air. Cardiovascular system examination was normal and S1 and S2 was heard, no murmurs were present. Respiratory system examination showed Bilateral air entry. CNS examination didn't reveal any further significant findings and per abdomen examination did not reveal any anomaly like tenderness or organomegaly. Local examination of Left knee: Swelling and tenderness were present, range of movement was restricted and painful with sensation intact.

Investigations

Patient was admitted for further evaluation and management. CT left knee revealed GROSSLY COMMINUTED AND DISPLACED FRACTURE OF THE PROXIMAL TIBIA.CT Abdomen showing no significant abnormalities.

LABORATORY INVESTIGATIONS	RESULTS
<i>SERUM ELECTROLYTES</i>	
SODIUM	136mEq/L
POTASSIUM	2.90mEq/L
CHLORIDE	104.1mEq/L
<i>ENDOCRINOLOGY</i>	
ALDOSTERONE	370.68pg/mL
RENIN	39.74μIU/ml

Management

Patient developed repeated hypertensive episodes so the patient was started with, Calcium Channel Blocker and β Blocker. Correction of hypokalemia by potassium

chloride was effective. In further evaluation, thyroid function test, serum cortisol level, 24 hours urine VMA showed no insignificant findings, but there was elevated serum aldosterone level with normal plasma renin level with disproportional aldosterone/renin ratio then the patient was started along with potassium sparing diuretic for the maintenance of blood pressure. Following CT abdomen was done which ruled out adenomas, tumours in adrenals. After the control of the blood pressure surgical correction of fracture by Open Reduction Internal Fixation with plating of left proximal tibia was done. Post-operative period was uneventful. Patient was discharged with follow up medications.

Follow Up

Patient under regular follow-up.

Discussion

Hyperaldosteronism can be primary or secondary ⁽¹⁾. Primary aldosteronism is an endocrine condition which is considered to be a common cause for secondary hypertension and is also associated with treatment resistant hypertension. Adrenal aldosteronoma is a benign adrenal neoplasm, causes primary aldosteronism and is known as Conn syndrome. It is important to diagnose primary aldosteronism associated Hypertension as it can be cured with proper medical and surgical treatment. Hence initial screening, diagnosis confirmation and specific sub type of primary aldosteronism should be done. In distal convoluted tubule aldosterone induces Na reabsorption and increases the secretion of K⁺ H⁺ ions causing hypokalemia, hypernatremia and alkalosis.

Bilateral Idiopathic adrenal hyperplasia (IAH- 75% cases) is the most common cause of primary aldosteronism than Aldosteronoma (70% cases). Unilateral adrenal hyperplasia is very rare ⁽²⁾. These two conditions should be differentiated because the treatment for Aldosteronomas are surgical resection and that of IAH is with aldosterone antagonist.

Causes of aldosteronism are as follows.

- Familial forms of Hyperaldosteronism

Three distinct varieties exist:

Type 1 is Glucocorticoid remediable aldosteronism (GRA) which is due to aberrantly formed primary gene product that combines CYP11B1 gene that is Glucocorticoid responsive promoter of 11 beta hydroxylase gene with the CYP11B2 gene which is the coding region of aldosterone synthetase gene ⁽³⁾. When the promoter is not fully transcriptionally silenced, leads to over expression of aldosterone synthetase, thus increasing the synthesis and secretion of aldosterone. In this type hypertension responds to small doses of dexamethasone in addition to anti hypertensives. Type 2- the locus lies on band 7p 22 but the exact genetic abnormality is not yet identified⁽⁴⁾.

Type 3-Due to KCNJ5 K⁺ channel mutation⁽⁵⁾

- Aldosterone producing adenomas
- Aldosterone producing renin responsive adenomas
- Bilateral idiopathic adrenal hyperplasia
- Ectopic secretion of aldosterone
- Pure aldosterone producing Adrenocortical adenomas

Patients with uncontrolled hypertension and hypokalemia are first screened for primary hyperaldosteronism by measuring serum potassium, bicarbonate, magnesium and sodium levels and calculating the plasma aldosterone / plasma renin ratio. Diagnosis conformation is done by 24 hours urinary aldosterone excretion test, salt loading test and serum aldosterone levels. Sub type of primary aldosteronism can be identified by doing postural stimulation test, diurnal rhythm of aldosterone and furosemide stimulation test. Radiological investigations like high resolution, thin sliced adrenal CT with contrast can be done.

Primary aldosteronism is treated with calcium channel blockers, mineralocorticoid antagonists and glucocorticoids with regular follow up. Surgical intervention is needed in patients with typical aldosteronomas, primary adrenal hyperplasia and renin responsive adenomas. Morbidity and mortality are based on the severity of hypokalemia and hypertension. Severe hypokalemia can cause fatal cardiac arrhythmias. Ventricular hypertrophy, Acute coronary syndrome, stroke are some serious complications caused by chronic uncontrolled hypertension. Drug reactions and complications due to surgery can also occur. In the absence of hypertension, chronic aldosteronism itself can cause ischemic, hypertrophic and fibrotic injury. Other complication includes hypertensive nephropathy and retinopathy. It is very much important to diagnose and specifically treat primary hyperaldosteronism because in severe hypertension, young individuals will cause early hypertensive kidney damage and renal arteriosclerosis leads to escape of plasma renin activity.

Conclusion

In a young patient presenting with hypokalemia and severe hypertension a suspicion of primary hyperaldosteronism should be ruled out and patient should be treated appropriately to prevent morbidity and mortality.

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