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PRIMARY HYPERALDOSTERONISM (CONN SYNDROME)

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ABSTRACT
Primary aldosteronism is the most common endocrine disorder. which aldosterone production is inappropriately high for sodium status, inappropriate production of aldosterone causes hypertension, cardiovascular damage, sodium retention, suppression of plasma renin, and increased potassium excretion that excess level cause severe may lead to hypokalemia. It is commonly caused by an adrenal adenoma or hyperplasia and adrenal carcinoma or inherited conditions of familial hyperaldosteronism.

INTRODUCTION

Primary Hyperaldosteronism is otherwise called as Conn syndrome. It was named after J. W. Conn who first described it in 1955, in a patient who had hypertension with an aldosterone-producing adenoma. [1,2] It is a rare health problem that occurs mainly in the adrenal glands. Excess production of aldosterone leads to high blood pressure. This syndrome more common male than females. More commonly this syndrome in middle adult age group of 30-40 years. It is a rare syndrome 1 out 100 all cause the conn syndrome.

ROLE OF ALDOSTERONE

- Function of the hormone are.
- They help to regulate fluid and salt levels in the body that affect blood volume and blood pressure.
- They play a major role in stress maintenance and its change.
- They cause a faster heart rate and boost other systems that help to react quickly with a burst of energy when needed.
- Problems in the cortex or the medulla, then, can result in high blood pressure.[3]

DETECTION RATE OF PRIMARY HYPERALDOSTERONISM/(CONN SYNDROME)

Primary hyperaldosteronism is the most commonly occur to secondary hypertension and occurs in incidence rate 6% to 20% of adult hypertensive patients, higher in patients with resistant hypertension. The prevalence of 10% was noted when consecutive patients with hypertension were evaluated. However, the prevalence increased to 30% when aldosterone to renin ratio (ARR) was used as a screening method in general practice.[4,5]

As per the worldwide prevalence in unselected patients with hypertension is now considered to be low, perhaps about 2% or even less than 1%, although there are recent reports suggesting higher values of up to 10-15% of patients with hypertension.[6]

The prevalence out of Sixty-three (18%) of the 350 hypertensive patients (215 women and 135 men; age range, 23-75 year) were screened positive for primary aldosteronism. that primary aldosteronism occurs in at least 5% of the adult Asian hypertensive population, and approximately half of these individuals may have potentially curable, unilateral, aldosterone-producing adrenal adenoma.[7]

A total of 43 cases of PA were identified, giving a minimum prevalence of 9.2% (43/465). Potentially the prevalence may be up to 15% assuming that the ARR has a sensitivity of 93% (42/45) in predicting PA. This suggests that the prevalence of PA in Tayside is as high as that in the Australian hypertensive population.[8]

The prevalence of PA by age of hypertension onset was highest in age-group 30-39 years (31.3%). There is a high prevalence with young-onset hypertension should be considered as a separate high-risk category in PA.[9]

Causes of Primary Hyperaldosteronism

The are two main causes of primary hyperaldosteronism. It is important to differentiate

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the etiology because the treatment and prognosis are very different.

Solitary Adrenal Nodule

The most common cause (75% of the time) is a small (<2cm), benign tumor on one of your adrenal glands called an "aldosteronoma".

Bilateral Adrenal Hyperplasia

In the other 25% of cases, both of your adrenal glands become hyperplastic, or plump, and continuously produce aldosterone.[10]

Symptoms of Primary Hyperaldosteronism

Primary aldosteronism generally causes high blood pressure with low potassium. Other symptoms may include:

Excessive thirst, fatigue, frequent urination, headache., Muscle cramps., Visual disturbances. and Weakness or tingling.[10]

Diagnostic Evaluation of Primary Hyperaldosteronism

History and Physical Often, the patients are asymptomatic but may present with symptoms of fatigue, muscle weakness, cramping (secondary to potassium wasting), headaches, and palpitations. They can also have polydipsia and polyuria from hypokalemia-induced nephrogenic diabetes insipidus. Many patients are as a result of persisting hypokalemia and hypertension. Others may present with serious arrhythmias after being started on diuretics for hypertension.[11]

The 24 h urine aldosterone measurement is collected on the third day of oral salt loading; alternatively, a serum aldosterone is collected following intravenous saline infusion of 2 L over 4 h. Additional options include the fludrocortisone suppression test and captopril challenge test, which are less standardized, more difficult to perform safely (fludrocortisone suppression test) and prone to equivocal results (captopril challenge test). Patients with spontaneous hypokalemia, suppressed renin, and plasma aldosterone concentration >20 ng/dL are diagnosed with unequivocal PA from screening alone.[12,13]

Treatment for Primary Hyperaldosteronism

It includes laparoscopic resection for adenomas. This procedure will usually resolve the hypokalemia, but hypertension can persist in up to 65% of patients post adrenalectomy. Surgery is the preferred treatment for patients with unilateral aldosterone-producing adenoma. After unilateral adrenalectomy, almost all patients have the resolution of hypokalemia and moderate improvement in blood pressure control.[14]

In those who are unable to undergo surgery or have bilateral adrenal hyperplasia, mineralocorticoid antagonists such as spironolactone or eplerenone are an option. In a randomized study, the antihypertensive effects between spironolactone and eplerenone in patients with primary hyperaldosteronism were studied showing that spironolactone was more effective than eplerenone in controlling blood pressure.[15]

CONCLUSIONS

For high-risk groups of hypertensive patients and those with hypokalemia, we recommend case detection of primary aldosteronism by determining the aldosterone-renin ratio under standard conditions and recommend that a commonly used confirmatory test should identify the condition. Diagnosis of all patients with primary aldosteronism undergo adrenal CT SCAN as the initial study in subtype testing and to exclude adrenocortical carcinoma. Those who are unsuitable for surgery should be treated primarily with a mineralocorticoid receptor antagonist.

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