Primary Hyperaldosteronism

Primary hyperaldosteronism (also known as Conn syndrome) is characterized by increased aldosterone secretion from an adrenal etiology. The etiology is commonly an adrenal adenoma or bilateral adrenal hyperplasia. Clinical features include hypertension, hypokalemic metabolic alkalosis, but no significant edema due to the aldosterone escape mechanism. Diagnosis is achieved with blood tests showing increased aldosterone levels and decreased renin. An abdominal CT scan can be used to identify the adrenal etiology. Management options include aldosterone antagonists and surgical resection.



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Etiology

Increased Aldosterone Secretion

Up-arrow Aldo-stereo

This disease is characterized by increased aldosterone secretion. Aldosterone acts on mineralocorticoid receptors, stimulating principal cells and alpha-intercalated cells of the collecting tubule. It induces the Na⁺/K⁺ pump and epithelial Na⁺ channels (ENaC). As a result, K⁺is secreted in the lumen along with H⁺.

Adrenal Adenoma

Adrenal Add (+) Gnome

A unilateral adrenal adenoma, known as Conn syndrome, is one of the two most common causes of primary hyperaldosteronism. It is an aldosterone-producing adenoma.

Bilateral Adrenal Hyperplasia

Bi-ladders Adrenal-hat Hiker-plates

Bilateral adrenal nodular hyperplasia is the other common cause of primary hyperaldosteronism. Hyperplasia occurs in the zona glomerulosa where aldosterone is secreted.

Clinical Features

Hypertension

Hiker-BP

High sodium reabsorption caused by excess aldosterone causes an increase in blood volume. This contributes to hypertension, which is seen in primary hyperaldosteronism patients. Patients often come with treatment-resistant hypertension and are typically young.

Hypokalemic Metabolic Alkalosis

Hippo-banana Metal-ball Elk-loser

Hypokalemic metabolic alkalosis can be present in primary hyperaldosteronism. Hypokalemia occurs due to potassium excretion via renal principal cells by aldosterone. Excess aldosterone will also decrease potassium reabsorption, resulting in high potassium in the urine. Hypokalemic patients can present with palpitations, fatigue, muscle cramps, and severe muscle weakness. Metabolic alkalosis occurs due to low H⁺ reabsorption and high H⁺ excretion due to an excess of aldosterone. High H⁺ excretion through alpha-intercalated cells leads to bicarbonate secretion, which contributes to metabolic alkalosis. Increased activity of the basolateral HCO₂/Cl⁻ exchanger is also involved in this process.

No Significant Edema

No-sign Edamame

Although it is intuitive to think that edema will result from increased blood volume and sodium levels because of the actions of aldosterone, this does not occur. The aldosterone escape mechanism goes into effect which responds to high aldosterone levels by increasing renal blood flow, thus natriuresis. This limits hypernatremia and edema.

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Diagnosis

Increased Aldosterone

Up-arrow Aldo-stereo

Aldosterone is the primary hormone secreted by the zona glomerulosa. Its production is stimulated by angiotensin II. The diagnosis of primary hyperaldosteronism can be confirmed by an elevated morning aldosterone to plasma renin activity ratio (higher than 20:1).

Decreased Renin

Down-arrow Wrenches

High aldosterone causes negative feedback on RAAS, resulting in low renin. The most common cause of low renin is primary mineralocorticoid excess.

Abdominal CT Scan

Cat-scanner Abdomen

Patients with suspected primary hyperaldosteronism should undergo an abdominal CT scan as the initial study, particularly to evaluate the adrenal glands. It can differentiate between unilateral adrenal adenoma, bilateral adenoma hyperplasia, and the possibility of adrenocortical carcinoma.

Management

Aldosterone Antagonists

Aldo-stereo Ant-toga

Aldosterone antagonists (e.g. spironolactone) are proven to be effective in treating primary hyperaldosteronism and reduce the plasma aldosterone level.

Surgery

Surgeon

Adrenalectomy is recommended in patients with unilateral adrenal adenoma. Patients with bilateral adrenal hyperplasia are better candidates for aldosterone antagonists.

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