Hyperaldosteronism Overview

Hyperaldosteronism is characterized by high aldosterone production from the adrenal gland. The aldosterone escape mechanism is an important concept that allows high aldosterone levels without corresponding edema and hypernatremia. Patients can present with hypertension and hypokalemic metabolic alkalosis. Hyperaldosteronism has several notable types including primary, secondary, and pseudohyperaldosteronism.

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Mechanism

Adrenal Glands

Adrenal Gland

Hyperaldosteronism is characterized by the increased production of aldosterone in one or both adrenal glands. It can be caused by an abnormality found in the adrenal gland itself (primary hyperaldosteronism) or outside the adrenal gland (secondary hyperaldosteronism).

Increased Aldosterone

Up-arrow Aldo-stereo

Hyperaldosteronism is characterized by an increased production of aldosterone which is secreted by the zona glomerulosa of the adrenal glands.

Aldosterone Escape Mechanism

Aldo-stereo Escaping

Typically, high aldosterone will cause a high sodium level in the blood. However, this does not always occur. Instead, increased blood volume due to sodium reabsorption by the excess of aldosterone leads to increased renal blood flow. This triggers natriuresis, which limits edema and hypernatremia.

Clinical Findings

No Significant Edema

No-sign Edamame

No significant edema will be present in primary hyperaldosteronism due to the aldosterone escape mechanism. As discussed previously, increased renal blood flow will increase natriuresis and limit edema.

Hypertension

Hiker-BP

High sodium reabsorption caused by excess aldosterone causes an increase in blood volume. This contributes to hypertension, which is seen in patients with hyperaldosteronism. 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 hyperaldosteronism. Hypokalemia occurs due to potassium excretion via renal principal cells due to 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. Increased 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.

Types

Primary Hyperaldosteronism

(1) Wand Hiker-Aldo-stereo

Primary hyperaldosteronism is characterized by an increase in aldosterone production due to an abnormality in the adrenal gland. It can be seen in patients with bilateral adrenal hyperplasia or adrenal adenoma. Diagnosis of primary hyperaldosteronism can be confirmed by an elevated morning aldosterone to plasma renin activity ratio (higher than 20:1).

Secondary Hyperaldosteronism

(2) Tutu Hiker-Aldo-stereo

Secondary hyperaldosteronism is characterized by an increase in aldosterone production due to an abnormality outside the adrenal gland. It occurs by activation of the renin-angiotensin-aldosterone system, which is stimulated by certain conditions such as juxtaglomerular cell tumors (renin-producing tumors), cirrhosis, heart failure, nephrotic syndrome, and renal artery stenosis. Diagnosis of secondary hyperaldosteronism can be confirmed by morning aldosterone to plasma renin activity ratio less than 10.

Pseudohyperaldosteronism

Sumo-Hiker-Aldo-stereo

Pseudohyperaldosteronism has a similar clinical manifestation to hyperaldosteronism but patients have low aldosterone levels. This is due to activation of the aldosterone receptor without the increase of aldosterone. Increases in cortisol or other steroid hormones with some mineralocorticoid activity can be responsible. It can occur due to direct mineralocorticoid effect from estrogens, fluorohydrocortisone, fluoroprednisolone, desoxycorticosterone, and /or the ingestion of high amounts of glycyrrhetinic acid (found in licorice).