

Secondary Hyperaldosteronism

Secondary hyperaldosteronism is characterized by an increase in aldosterone production due to an abnormality outside the adrenal gland. Etiologies include renal artery stenosis, reninoma, and chronic disease of the liver, kidney, or heart. Clinical features include edema, hypertension, and hypokalemic metabolic alkalosis. Diagnosis is achieved with blood tests showing the increase of aldosterone and renin levels. Management includes treating the underlying disorder and aldosterone antagonists.



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Etiologies

Renal Artery Stenosis

[Kidney Artery of Stone](#)

Renal artery stenosis due to fibromuscular dysplasia or atherosclerosis can cause reduced blood flow through the kidneys. This will induce aldosterone secretion, resulting in secondary hyperaldosteronism.

Reninoma

[Wrench-gnome](#)

Reninoma, also called a juxtaglomerular cell tumor, is a renin-producing tumor that induces the activation of the renin-angiotensin-aldosterone system. These will result in secondary hyperaldosteronism.

Chronic Disease of Liver, Kidney, or Heart

[Crone Stacked by Liver, Kidney, and Heart Diseased](#)

Activation of the renin-angiotensin-aldosterone system can happen due to reduced perfusion through the kidney, resulting in secondary hyperaldosteronism. It may occur due to decreased circulating fluid volume, chronic disease of the liver, chronic disease of the kidney, and low cardiac output due to chronic heart disease.

Clinical Features

Edema

[Edamame](#)

The increase of aldosterone in secondary hyperaldosteronism will increase sodium levels and blood volume. This will result in the accumulation of fluid in the body, known as edema. It's different from primary hyperaldosteronism, which has no significant edema due to the aldosterone escape mechanism.

Hypertension

[Hiker-BP](#)

High sodium reabsorption caused by excess aldosterone in secondary hyperaldosteronism causes an increase in blood volume. This contributes to hypertension, which is seen in patients with hyperaldosteronism.

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 HCO_3^{-}

255, 255); bottom: -0.25em; box-sizing: border-box; color: rgb(39, 43, 51); font-family: ;">2</sub>/Cl⁻ exchanger is also involved in this process.

Diagnosis

Increased Aldosterone

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⁺.

Increased Renin

Up-arrow Wrenches

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 shown by high renin levels. Diagnosis of secondary hyperaldosteronism can be confirmed by morning aldosterone to plasma renin activity ratio less than 10.

Treatment

Treat Underlying Disorder

Treating Underlying Attacker

Treatment of secondary hyperaldosteronism is based on the etiology. For example, renal artery stenosis can be treated with revascularization, surgery for reninoma, and other treatments that can be used for chronic disease of the liver, kidney, or heart.

Aldosterone Antagonists

Aldo-stereo Ant-toga

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