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THE ROLE OF ALDOSTERONE EXCESS IN THE DEVELOPMENT OF CARDIOVASCULAR AND METABOLIC DISORDERS

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Abstract: Aldosterone is a mineralocorticoid hormone produced by the zona glomerulosa of the adrenal cortex and plays an essential role in sodium balance, potassium homeostasis, extracellular fluid volume, and blood pressure regulation. Excessive aldosterone secretion can lead to significant cardiovascular, renal, and metabolic disturbances. The aim of this study was to analyze the pathophysiological mechanisms and clinical consequences of aldosterone excess, with particular attention to primary aldosteronism. A literature-based analytical approach was used to evaluate the renin-angiotensin-aldosterone system, mineralocorticoid receptor signaling, renal sodium reabsorption, potassium excretion, vascular remodeling, myocardial fibrosis, insulin resistance, and diagnostic principles. The analysis indicates that chronic aldosterone excess promotes sodium retention, volume expansion, hypertension, hypokalemia, endothelial dysfunction, vascular stiffness, left ventricular hypertrophy, myocardial fibrosis, and increased cardiovascular risk. Aldosterone may also contribute to metabolic abnormalities through oxidative stress, inflammation, and impaired insulin signaling. Primary aldosteronism is now recognized as one of the most important potentially treatable causes of secondary hypertension. Accurate diagnosis requires appropriate screening using the aldosterone-to-renin ratio, confirmatory testing where indicated, adrenal imaging, and subtype evaluation. Treatment with mineralocorticoid receptor antagonists or adrenal surgery can significantly reduce cardiovascular risk. Understanding aldosterone biology is therefore essential for modern endocrine and cardiovascular medicine.
Keywords: aldosterone, primary aldosteronism, hypertension, mineralocorticoid receptor, hypokalemia, adrenal gland, cardiovascular risk, endocrinology.

Introduction

Aldosterone is a steroid hormone synthesized in the zona glomerulosa of the adrenal cortex. Its principal physiological function is to maintain sodium and potassium homeostasis and contribute to the regulation of extracellular fluid volume and arterial blood pressure.

Aldosterone secretion is controlled primarily by the renin-angiotensin system and serum potassium concentration. Reduced renal perfusion stimulates renin release from juxtaglomerular cells. Renin initiates formation of angiotensin II, which stimulates aldosterone secretion from the adrenal cortex. In the distal nephron, aldosterone binds to intracellular mineralocorticoid receptors and increases the expression and activity of sodium channels and sodium-potassium ATPase pumps. This enhances sodium reabsorption and potassium excretion.

Under physiological conditions, this mechanism is essential for maintaining circulatory volume and electrolyte balance. However, chronic inappropriate aldosterone secretion can become pathological. Primary aldosteronism is characterized by autonomous aldosterone production that is relatively independent of renin activity. Common causes include bilateral adrenal hyperplasia and aldosterone-producing adrenal adenoma.

The clinical importance of primary aldosteronism extends beyond elevated blood pressure. Aldosterone can directly affect the cardiovascular system, kidneys, adipose tissue, and metabolic pathways.

The aim of this study was to analyze the endocrine, cardiovascular, renal, and metabolic consequences of aldosterone excess and to review the major principles of diagnosis and treatment.

Materials and Methods

This study was prepared as a qualitative literature-based analytical review. Scientific publications and standard endocrinology sources concerning aldosterone physiology, mineralocorticoid receptor signaling, primary aldosteronism, secondary hypertension, cardiovascular remodeling, renal electrolyte balance, and metabolic disorders were analyzed.

The review focused on renal sodium transport, potassium excretion, plasma volume regulation, endothelial function, oxidative stress, cardiac fibrosis, insulin sensitivity, and diagnostic evaluation. Particular attention was given to the aldosterone-to-renin ratio, confirmatory testing, adrenal computed tomography, adrenal venous sampling, mineralocorticoid receptor antagonist therapy, and surgical treatment.

No original patient data were used. The Results section represents a synthesis of established endocrine and cardiovascular evidence.

Results

The analysis demonstrated that aldosterone excess first produces important renal effects.Aldosterone increases sodium reabsorption in the distal nephron, particularly within principal cells of the collecting duct.

This process increases extracellular fluid volume and contributes to sustained arterial hypertension.

At the same time, increased sodium reabsorption promotes potassium secretion into the urine.

Patients with severe aldosterone excess may therefore develop hypokalemia.

Hypokalemia can produce muscle weakness, fatigue, cramps, cardiac rhythm disturbances, and metabolic abnormalities.

However, many patients with primary aldosteronism have normal serum potassium concentrations. Therefore, absence of hypokalemia does not exclude the disorder.

Aldosterone excess suppresses renin secretion through volume expansion and increased blood pressure.

As a result, the characteristic biochemical pattern includes elevated or inappropriately normal aldosterone with suppressed renin.

This relationship forms the basis of the aldosterone-to-renin ratio used for screening.

The analysis also showed that chronic aldosterone excess has direct cardiovascular effects beyond its influence on blood pressure.

Mineralocorticoid receptors are expressed in vascular tissue and the myocardium.

Excessive receptor activation can promote endothelial dysfunction, oxidative stress, inflammation, and vascular remodeling.

These mechanisms contribute to increased arterial stiffness.

In the heart, aldosterone can stimulate fibroblast activity and collagen deposition.

This may lead to myocardial fibrosis and left ventricular hypertrophy.

As a result, patients with primary aldosteronism may have greater cardiovascular risk than patients with essential hypertension of similar severity.

Increased rates of atrial fibrillation, stroke, myocardial infarction, and heart failure have been reported in association with untreated aldosterone excess.

Renal damage is another important consequence.

Persistent hypertension and mineralocorticoid receptor activation can promote glomerular hyperfiltration, albuminuria, and progressive renal injury.

After treatment, glomerular filtration may decrease because correction of hyperfiltration unmasks underlying renal dysfunction.
The analysis further demonstrated a possible relationship between aldosterone excess and metabolic abnormalities.
Mineralocorticoid receptor activation can influence adipose tissue inflammation and insulin signaling.
Oxidative stress and inflammatory pathways may contribute to reduced insulin sensitivity.
Some patients with primary aldosteronism have an increased prevalence of metabolic syndrome and impaired glucose metabolism.
However, these metabolic relationships are influenced by obesity, age, hypertension, and other associated risk factors.
Primary aldosteronism can arise from unilateral or bilateral adrenal disease.
A unilateral aldosterone-producing adenoma may be treated surgically.
Bilateral adrenal hyperplasia is usually treated medically using mineralocorticoid receptor antagonists.
Adrenal imaging alone cannot reliably determine whether aldosterone excess is unilateral because incidental adrenal nodules are common.
For this reason, adrenal venous sampling is often used in appropriate surgical candidates to determine lateralization.

Discussion

The findings demonstrate that aldosterone excess should not be viewed solely as a disorder of sodium and potassium balance.
Its effects involve multiple organ systems and substantially increase cardiovascular risk.
One of the most clinically important observations is that primary aldosteronism is more common than historically believed.
It can occur not only in patients with severe resistant hypertension but also in individuals with less extreme blood pressure elevation.
Therefore, appropriate screening is important in selected hypertensive populations.
The aldosterone-to-renin ratio is widely used as a screening test because autonomous aldosterone production suppresses renin.
However, interpretation requires attention to medications, sodium intake, potassium concentration, posture, and sampling conditions.
Some antihypertensive drugs can alter renin or aldosterone levels and affect the ratio.
Hypokalemia itself may suppress aldosterone secretion and should therefore be corrected before diagnostic evaluation when possible.
Confirmatory testing may be required after a positive screening result.
Different approaches include saline infusion testing, oral sodium loading, captopril challenge, and other protocols.
The choice depends on clinical circumstances and local expertise.
Determining whether aldosterone excess originates from one adrenal gland or both has direct therapeutic importance.
Unilateral disease may be treated by adrenalectomy, whereas bilateral disease is generally managed medically.
Adrenal venous sampling remains an important technique for distinguishing unilateral from bilateral aldosterone secretion in many surgical candidates.
Mineralocorticoid receptor antagonists are central to medical treatment.

Spironolactone blocks the mineralocorticoid receptor and can reduce blood pressure, correct hypokalemia, and lower cardiovascular risk.
Its antiandrogenic effects can cause gynecomastia and sexual side effects in some patients.
Eplerenone is more selective for the mineralocorticoid receptor and may cause fewer sex-hormone-related adverse effects, although dosing and cost considerations may differ.
The cardiovascular consequences of aldosterone excess are especially important.
The higher incidence of atrial fibrillation and structural cardiac abnormalities suggests that aldosterone has direct myocardial effects.
Treatment can reduce left ventricular mass and improve cardiovascular outcomes.
This means that early recognition is important even when blood pressure can be controlled with conventional antihypertensive therapy.
The metabolic role of aldosterone also deserves attention.
Mineralocorticoid receptor activation has been linked with adipose tissue dysfunction, oxidative stress, and insulin resistance.
These pathways may partially explain the overlap between primary aldosteronism, metabolic syndrome, and type 2 diabetes risk.
However, more research is required to distinguish direct hormonal effects from the influence of obesity and chronic hypertension.
Overall, primary aldosteronism represents an important example of how endocrine hypertension differs biologically from essential hypertension.
Identifying the underlying hormonal mechanism allows targeted therapy rather than nonspecific blood pressure treatment alone.

Conclusion

Aldosterone is an essential regulator of sodium balance, potassium excretion, blood volume, and arterial pressure.
Chronic aldosterone excess causes sodium retention, renin suppression, hypertension, and in some patients hypokalemia.
The pathological effects extend beyond the kidneys and include endothelial dysfunction, arterial stiffness, cardiac hypertrophy, myocardial fibrosis, renal injury, and increased cardiovascular risk.
Primary aldosteronism is a common and potentially treatable cause of secondary hypertension.
Diagnosis begins with appropriate biochemical screening using aldosterone and renin measurements and may require confirmatory testing, adrenal imaging, and adrenal venous sampling.
Unilateral disease may be treated with adrenalectomy, whereas bilateral disease is generally treated using mineralocorticoid receptor antagonists.
Early recognition and targeted treatment can reduce both blood pressure and the long-term cardiovascular consequences of aldosterone excess.

References

1. Funder JW, Carey RM, Mantero F, et al. The management of primary aldosteronism: case detection, diagnosis, and treatment. Journal of Clinical Endocrinology & Metabolism. 2016;101(5):1889–1916.
2. Скворцова, Л. А., Махмудов, Д. Э., Усманов, Д. Р., Адылходжаев, А. А., & Хегай, Л. Н. (2019). АСПЕКТЫ СПОРТИВНОЙ АДАПТОЛОГИИ ПРИ ИЗУЧЕНИИ ВИТАМИНА D 25 (OH) D У ФУТБОЛИСТОВ. In От фундаментальных знаний к тонкому владению скальпелем (pp. 51-54).

3. Хегай, Л. Н., Скворцова, Л. А., Махмудов, Д. Э., Усманов, Д. Р., Муслимов, О. Я., & Адылходжаев, А. А. (2019). СОДЕРЖАНИЕ 25-ОН-ВИТАМИНА D У ФУТБОЛИСТОВ В УСЛОВИЯХ СОЛНЕЧНОЙ РАДИАЦИИ УЗБЕКИСТАНА. In Actual Questions and Innovations in Science 2 (pp. 160-161).
4. Mavlyanov, I., Parpiev, S., Sadikov, A., Kurganov, S., & Makhmudov, D. (2020). Relative features of the PPARA (Rs4253778), PPARGC1A (Rs8192678) and PPARG2 (Rs1801282) polymorphisms genes in athletes engaged in cyclic types of sports. European Journal of Molecular and Clinical Medicine, 7(2), 1860-1869.
5. Пирназаров, М. М., Садиков, А. А., Махмудов, Д. Э., & Хегай, Л. Н. (2021). Магнитно-резонансная томографическая визуализация повреждений коленных суставов. Re-health journal, (1 (9)), 152-159.
6. Махмудов, Д. Э., & Саитов, Т. А. (2023). Анализ персонализированного питания высококвалифицированных спортсменов управленческих видов спорта. Klinik va profilaktik tibbiyot jurnali, (3), 79.
7. Махмудов, Д. Э., & Саитов, Т. А. (2023). Анализ персонализированного питания высококвалифицированных спортсменов управленческих видов спорта. Klinik va profilaktik tibbiyot jurnali, (3), 79.
8. Таралева, Т. А., Махмудов, Д. Э., Атабаева, С. М., & Сагитов, Д. М. (2026). ВАРИАБЕЛЬНОСТЬ СЕРДЕЧНОГО РИТМА В ЦИКЛИЧЕСКИХ ВИДАХ СПОРТА: СОВРЕМЕННЫЕ ПОДХОДЫ К ОЦЕНКЕ ФУНКЦИОНАЛЬНОГО СОСТОЯНИЯ СПОРТСМЕНОВ. South Aral Region Medical Journal, 2(Maxsus son-2), 587-593.
9. Махмудов, Д., Абдуллаева, Х., Таралева, Т., Аблялимов, Р., & Парпиев, С. (2025). АНАЛИЗ ФУНКЦИОНАЛЬНЫХ ПОКАЗАТЕЛЕЙ ФУТБОЛИСТОВ НА ЭТАПЕ ОТБОРА К СОРЕВНОВАНИЯМ. Медицина и спорт, (3), 162-162.