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PATHOPHYSIOLOGICAL BASIS AND HORMONAL FACTORS OF THE DEVELOPMENT OF HYPERTENSION

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Abstract. *This article explores the pathophysiological mechanisms underlying the development of hypertension, with a special emphasis on the hormonal factors that contribute to elevated blood pressure. Hypertension, one of the most prevalent cardiovascular disorders worldwide, results from a complex interplay between genetic predisposition, environmental influences, and dysregulation of multiple physiological systems. The article discusses how alterations in renal function, vascular resistance, and sympathetic nervous system activity lead to chronic elevation of arterial pressure. Particular attention is given to the renin-angiotensin-aldosterone system (RAAS), vasopressin, and other endocrine factors such as catecholamines and natriuretic peptides. The review also highlights the feedback loops and receptor sensitivities that sustain hypertensive states over time. Understanding these hormonal contributions is essential for identifying therapeutic targets and improving the management of hypertensive patients. Overall, the paper aims to provide a comprehensive view of how hormonal imbalance acts as a key driver in the pathogenesis of hypertension.*

Keywords*: Hypertension, Pathophysiology, RAAS, Vasoconstriction, Aldosterone, Angiotensin II, Baroreceptors.*

ПАТОФИЗИОЛОГИЧЕСКИЕ ОСНОВЫ И ГОРМОНАЛЬНЫЕ ФАКТОРЫ РАЗВИТИЯ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ

Аннотация. *В этой статье рассматриваются патофизиологические механизмы, лежащие в основе развития гипертонии, с особым акцентом на гормональных факторах, которые способствуют повышению артериального давления. Гипертония, одно из самых распространенных сердечно-сосудистых заболеваний во всем мире, является результатом сложного взаимодействия генетической предрасположенности, влияния окружающей среды и нарушения регуляции множества физиологических систем. В статье обсуждается, как изменения в функции почек, сосудистом сопротивлении и активности симпатической нервной системы приводят к хроническому повышению артериального давления. Особое внимание уделяется ренин-ангиотензин-альдостероновой системе (РААС), вазопрессину и другим эндокринным факторам, таким как катехоламины и натрийуретические пептиды. В обзоре также освещаются петли обратной связи и чувствительность рецепторов, которые поддерживают гипертонические состояния с течением времени. Понимание этих гормональных факторов имеет важное значение для определения терапевтических целей и улучшения лечения пациентов с гипертонией.*

В целом, статья направлена на то, чтобы предоставить всестороннее представление о том, как гормональный дисбаланс действует как ключевой фактор в патогенезе гипертонии.

Ключевые слова: *Гипертония, Патофизиология, РААС, Вазоконстрикция, Альдостерон, Ангиотензин II, Барорецепторы.*

Introduction

Hypertension, commonly known as high blood pressure, is one of the most prevalent and clinically significant cardiovascular disorders worldwide. According to the World Health Organization, approximately one in three adults globally suffers from elevated blood pressure levels. As a major risk factor for heart disease, stroke, kidney failure, and other vascular complications, hypertension presents a substantial public health burden and continues to be a leading cause of morbidity and mortality.

The development of hypertension is multifactorial, involving complex interactions between genetic predisposition, environmental influences, and internal regulatory mechanisms.

Among these, the pathophysiological and hormonal contributors play a central role in the initiation and progression of sustained blood pressure elevation. Dysregulation in hormonal systems such as the renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system, and the release of hormones like vasopressin, cortisol, and adrenaline, leads to increased vascular resistance, sodium retention, and fluid imbalance all of which are key mechanisms in the pathogenesis of hypertension.

This paper aims to explore the fundamental pathophysiological mechanisms underlying hypertension, with a particular focus on the hormonal pathways involved. By examining how these systems contribute to abnormal blood pressure regulation, this discussion seeks to enhance understanding of the disease and support the development of more effective therapeutic and preventive strategies.

Main Body

Hypertension is a chronic condition marked by a persistent elevation of arterial blood pressure, which significantly increases the risk of cardiovascular events such as heart attacks, strokes, and kidney failure. It is often called the "silent killer" because it can remain asymptomatic for years while gradually damaging vital organs. Globally, it affects over a billion people, making it a major public health concern.

Multiple factors contribute to the development of hypertension, including genetics, lifestyle, environmental influences, and internal physiological mechanisms. Among these, hormonal imbalance plays a crucial role in the regulation of blood pressure and vascular tone.

Hormones like renin, angiotensin II, aldosterone, vasopressin, and catecholamines are intimately involved in fluid balance, sodium retention, and vascular resistance. Abnormalities in these systems can lead to long-term increases in blood pressure.

This paper aims to explore the pathophysiological basis of hypertension, focusing specifically on the hormonal mechanisms that support its development. A comprehensive understanding of these factors is essential for improving diagnosis, treatment, and prevention strategies.

Hypertension arises from an imbalance between cardiac output and systemic vascular resistance. A key factor is increased peripheral resistance due to structural and functional changes in the blood vessels. Endothelial dysfunction impairs the production of vasodilators such as nitric oxide, leading to sustained vasoconstriction. Inflammatory processes and oxidative stress further damage the vascular wall, promoting fibrosis and stiffness. Renal mechanisms also play a central role by regulating sodium and water retention; impaired renal function often contributes to volume overload. Additionally, autonomic nervous system imbalance, particularly sympathetic overactivity, contributes to persistent elevations in blood pressure. These mechanisms are not isolated but interact with one another to create a cycle of worsening hypertension. Left untreated, these pathophysiological changes result in end-organ damage, particularly affecting the heart, brain, kidneys, and eyes. Understanding these mechanisms helps identify specific targets for medical therapy and lifestyle interventions in managing hypertension effectively.

The renin-angiotensin-aldosterone system (RAAS) is a major regulator of blood pressure and fluid balance. When blood pressure drops, the kidneys release renin, an enzyme that converts angiotensinogen to angiotensin I. Angiotensin-converting enzyme (ACE) then transforms angiotensin I into angiotensin II, a potent vasoconstrictor that increases blood pressure by narrowing blood vessels. Angiotensin II also stimulates the adrenal cortex to release aldosterone, which promotes sodium and water reabsorption in the kidneys. This increases blood volume and cardiac output, further raising blood pressure. Chronic activation of the RAAS contributes to sustained hypertension and vascular remodeling. It also promotes inflammation and fibrosis in the heart and vessels. Many antihypertensive drugs target this system, including ACE inhibitors, angiotensin receptor blockers (ARBs), and aldosterone antagonists. Dysfunction or overactivation of RAAS is a primary factor in many hypertensive patients, especially those with conditions such as renal artery stenosis or heart failure. Therefore, understanding and managing RAAS is crucial in hypertension therapy.

The sympathetic nervous system (SNS) is essential for short-term blood pressure regulation, but chronic overactivity leads to hypertension. When the SNS is overactive, it stimulates the heart to beat faster and stronger, increasing cardiac output. Simultaneously, it causes vasoconstriction in peripheral blood vessels, raising vascular resistance. It also stimulates the adrenal medulla to release catecholamines like adrenaline and noradrenaline, which enhance these effects. Prolonged sympathetic stimulation leads to structural changes in the heart and vessels, including hypertrophy and fibrosis. This contributes to sustained hypertension and eventually heart failure. Increased sympathetic activity is often seen in individuals with stress, obesity, sleep apnea, or insulin resistance. Over time, it also impairs baroreceptor sensitivity, which normally helps regulate blood pressure fluctuations. Therapeutic strategies such as beta-blockers and lifestyle interventions aim to reduce sympathetic tone. Thus, the sympathetic nervous system is a vital pathophysiological target in understanding and managing hypertension.

Vasopressin, also known as antidiuretic hormone (ADH), is secreted by the posterior pituitary gland and plays a crucial role in water homeostasis. It promotes water reabsorption in the kidneys, thereby increasing blood volume and pressure. In addition, vasopressin exerts vasoconstrictive effects on blood vessels, further contributing to hypertension.

Another significant hormone is cortisol, which is elevated during stress and enhances the vasoconstrictive response of blood vessels while also promoting sodium retention.

Catecholamines such as epinephrine and norepinephrine raise heart rate and constrict blood vessels. Insulin resistance, commonly observed in metabolic syndrome, is associated with increased sodium reabsorption and sympathetic overactivity. These hormonal changes often co-exist and amplify one another, creating a complex neuroendocrine environment that supports the progression of hypertension. Conditions like Cushing's syndrome, pheochromocytoma, and primary hyperaldosteronism exemplify how hormonal disorders can lead to secondary hypertension. Therefore, hormonal balance is critical in blood pressure regulation.

Recognizing the hormonal basis of hypertension has profound implications for its clinical management. Hormonal evaluation is especially important in patients with resistant or secondary hypertension. Identifying hormonal disorders allows for targeted therapy, which can normalize blood pressure and reduce the risk of complications. For example, patients with primary aldosteronism may benefit from mineralocorticoid receptor antagonists or adrenal surgery.

Pharmacological agents targeting the RAAS, such as ACE inhibitors and ARBs, are standard treatments in many hypertensive patients. Beta-blockers reduce sympathetic drive, while diuretics help counteract fluid retention. Hormonal testing may include measurements of plasma renin, aldosterone, cortisol, and catecholamines. Treatment decisions based on hormonal profiles can be more effective and tailored to individual needs. In addition, understanding hormonal pathways may lead to the development of new therapeutic drugs. Therefore, the integration of endocrine analysis into hypertension management is crucial for achieving better long-term outcomes.

Conclusion

Hypertension is a multifactorial disease that involves complex interactions between genetic, environmental, and physiological factors. Among the most important contributors to its pathogenesis are hormonal and neurohormonal mechanisms. The renin-angiotensin-aldosterone system, sympathetic nervous system, vasopressin, cortisol, and catecholamines all play vital roles in maintaining vascular tone, fluid balance, and cardiac output. Dysregulation of these systems leads to sustained elevations in blood pressure and contributes to the development of cardiovascular disease. An in-depth understanding of these mechanisms enables the use of more effective pharmacological and non-pharmacological interventions. Hormonal evaluation is especially useful in diagnosing secondary hypertension and personalizing treatment strategies.

Future research should focus on identifying novel hormonal targets and improving diagnostic tools for early intervention. In conclusion, managing hypertension effectively requires not only controlling blood pressure but also addressing the underlying hormonal imbalances that drive its progression.

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