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PATHOPHYSIOLOGICAL MECHANISMS OF ARTERIAL HYPERTENSION DEVELOPMENT IN OBESITY

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Abstract: Obesity is a major risk factor for the development of arterial hypertension. Excess body weight is associated with complex alterations in metabolism, hormonal regulation, and vascular function that contribute to elevated blood pressure. Key pathophysiological mechanisms include activation of the sympathetic nervous system, dysregulation of the renin-angiotensin-aldosterone system, endothelial dysfunction, chronic inflammation, and sodium metabolism disturbances. These changes lead to increased vascular resistance, fluid retention, and vascular remodeling. Understanding these mechanisms is crucial for early diagnosis, prevention, and effective management of hypertension in obese patients.

Keywords: Obesity; arterial hypertension; sympathetic nervous system; renin-angiotensin-aldosterone system; endothelial dysfunction.

Introduction

Obesity has become a global health challenge and is closely linked to the development of arterial hypertension (AH). Individuals with excess body weight are at a significantly higher risk of elevated blood pressure, which increases the likelihood of cardiovascular complications. Adipose tissue is not only an energy storage depot but also an active endocrine organ that secretes adipokines, cytokines, and other bioactive molecules. These substances influence vascular tone, kidney function, and neurohormonal regulation of blood pressure. Understanding the pathophysiological mechanisms underlying hypertension in obesity is essential for developing preventive measures and therapeutic strategies to control blood pressure and reduce cardiovascular risk.

Relevance

The prevalence of obesity continues to rise globally, leading to a parallel increase in arterial hypertension cases. Elevated blood pressure in obese individuals is a major risk factor for cardiovascular diseases, stroke, and chronic kidney disease. Investigating the pathophysiological mechanisms of hypertension in obesity allows for early detection of abnormalities, optimization of pharmacological therapy, and development of preventive interventions aimed at weight reduction and improvement of vascular function.

Main Part

Obesity is associated with chronic activation of the sympathetic nervous system (SNS). Excess adipose tissue releases leptin and other adipokines that stimulate hypothalamic centers

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regulating sympathetic activity. SNS activation increases heart rate, cardiac output, and arteriolar constriction. These changes raise peripheral vascular resistance, contributing to the development of hypertension. Prolonged SNS stimulation also promotes vascular smooth muscle hypertrophy and enhances vascular reactivity to vasoconstrictor agents. Adipose tissue contributes to the activation of the renin-angiotensin-aldosterone system (RAAS). Adipokines stimulate renal renin secretion, increasing angiotensin II formation. Angiotensin II causes vasoconstriction, promotes aldosterone release, and enhances sodium and water retention in the kidneys. These effects increase circulating blood volume and elevate blood pressure. Chronic RAAS activation also induces hypertrophy of vascular smooth muscle cells and contributes to the progression of hypertension.

Obesity impairs endothelial function, which is essential for regulating vascular tone. Excess adiposity increases the production of proinflammatory cytokines and oxidative stress, reducing nitric oxide synthesis—a key vasodilator. Nitric oxide deficiency leads to vasoconstriction, increased peripheral resistance, and hypertension development. Endothelial dysfunction also diminishes the anticoagulant and antiproliferative properties of the endothelium, aggravating vascular changes. Low-grade chronic inflammation is a hallmark of obesity. Adipose tissue secretes proinflammatory cytokines, such as interleukins and tumor necrosis factor- α , which activate immune cells in the vascular wall and kidneys. Inflammation increases vascular stiffness, disrupts sodium and water regulation, and enhances sympathetic activity. The combined effect of inflammation and endothelial dysfunction creates a favorable environment for the progression of hypertension.

Obesity is associated with increased renal sodium reabsorption, mediated by RAAS activation and sympathetic stimulation. Sodium and water retention expand circulating blood volume, elevate cardiac preload, and contribute to increased arterial pressure. Additionally, obesity is linked to altered renal perfusion and enhanced vascular sensitivity to angiotensin II and norepinephrine, further exacerbating hypertension. Chronic hypertension in obesity induces structural changes in blood vessels, including smooth muscle cell hypertrophy, intimal thickening, and reduced arterial elasticity. These alterations increase peripheral vascular resistance, creating a “vicious cycle” in which elevated blood pressure further promotes vascular remodeling. Vascular remodeling is a key factor in the transition from functional hypertension to organic damage of blood vessels and target organs.

Conclusion

Arterial hypertension in obesity develops through complex pathophysiological mechanisms, including sympathetic nervous system activation, renin-angiotensin-aldosterone system dysregulation, endothelial dysfunction, chronic inflammation, sodium retention, and vascular remodeling. These mechanisms act synergistically, increasing peripheral vascular resistance, circulating blood volume, and maintaining elevated blood pressure. Understanding these processes is essential for developing preventive and therapeutic strategies aimed at weight reduction, blood pressure normalization, and prevention of cardiovascular complications in obese patients.

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