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Changes in Peripheral Vessels Depending on the Level of Arterial Hypertension in Patients with Ischemic Heart Disease and Arterial Hypertension

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Abstract: Currently, cardiovascular diseases (CVD) are the main problem of government, medical and public organizations in industrially developed countries due to the high incidence, disability and mortality among the population. Elevated BP levels, measured in or out of a healthcare setting, are directly and independently associated with the incidence of most CV events (hemorrhagic stroke, ischemic stroke, myocardial infarction, sudden death, heart failure, and peripheral arterial disease) and end-stage renal disease.

Keywords: Arterial hypertension, cardiovascular diseases, blood pressure.

Introduction.

Cardiovascular diseases (CVD) remain a leading cause of morbidity and mortality worldwide, posing significant challenges to healthcare systems, policymakers, and public health initiatives. Among these, arterial hypertension (AH) is a critical contributor to the development and progression of structural and functional disorders across the vascular system, impacting vessels of all calibers and leading to serious complications such as ischemic heart disease, myocardial infarction, and stroke. Despite advancements in understanding and managing hypertension, the complex interplay between elevated blood pressure and vascular changes continues to demand further exploration. AH is characterized by persistently high clinical blood pressure, resulting from multifaceted factors such as genetic predisposition, lifestyle habits, and underlying health conditions. These factors collectively influence vascular remodeling, a process that alters the structure and functionality of blood vessels, thereby exacerbating the clinical burden of hypertension. Endothelial dysfunction, often triggered by a deficiency of nitric oxide and an imbalance between vasodilators and vasoconstrictors, is recognized as a pivotal mechanism underlying these vascular changes.

In recent years, attention has shifted toward understanding the microcirculatory system and its role in hypertension-related vascular damage. Changes in peripheral vessels, including alterations in vessel wall thickness, lumen diameter, and capillary density, are indicative of progressive vascular remodeling. These changes are not only markers of disease severity but also contribute to the worsening of hypertension through feedback mechanisms. However, gaps remain in quantifying these microcirculatory changes and understanding their causative or consequential relationship with hypertension. Moreover, the diagnostic methods used to assess these alterations, such as laser Doppler flowmetry and capillaroscopy, require further standardization to provide consistent and actionable insights.

This study aims to investigate the changes in peripheral vessels in patients with ischemic heart disease and arterial hypertension, focusing on their structural and functional transformations. By examining these vascular changes, this research seeks to contribute to the growing body of evidence on the pathophysiology of hypertension and its systemic implications. Furthermore, the findings aim to bridge existing knowledge gaps, offering insights into potential diagnostic and therapeutic approaches for managing vascular complications in hypertensive patients. Understanding these dynamics is crucial not

only for improving clinical outcomes but also for shaping future research directions in cardiovascular medicine.

Methods

This study investigates the structural and functional changes in peripheral vessels associated with arterial hypertension (AH) in patients diagnosed with ischemic heart disease. A cross-sectional observational study design was employed to assess vascular remodeling patterns and their relationship with hypertension severity. Ethical approval for the study was obtained from the relevant institutional ethics committee, and all participants provided informed consent. Participants The study included adult patients aged 35–70 years diagnosed with arterial hypertension and ischemic heart disease. Inclusion criteria comprised patients with confirmed hypertension based on clinical blood pressure measurements exceeding 140/90 mmHg and evidence of ischemic heart disease determined by electrocardiography (ECG) and clinical history. Exclusion criteria included patients with secondary hypertension, uncontrolled diabetes mellitus, or other significant comorbidities affecting vascular health. Data Collection Clinical blood pressure measurements were performed using standardized sphygmomanometers in a controlled environment. Peripheral vascular changes were assessed using non-invasive diagnostic tools, including laser Doppler flowmetry, nailfold capillaroscopy, and bulbar conjunctiva microscopy. These methods allowed for the evaluation of microvascular structure, blood flow dynamics, and capillary density. Additional parameters, such as pulse wave velocity (PWV), were measured to assess vascular stiffness. Study Procedures The study categorized participants into groups based on hypertension severity (mild, moderate, and severe) according to the European Society of Hypertension (ESH) guidelines. For each participant, measurements of vascular lumen diameter, wall thickness, and capillary rarefaction were recorded. The presence of endothelial dysfunction was inferred by analyzing the balance between vasodilatory (nitric oxide) and vasoconstrictive (endothelin and angiotensin-II) markers. Statistical Analysis Data were analyzed using SPSS software (version 25.0). Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as percentages. Comparisons between groups were made using ANOVA for continuous variables and chi-square tests for categorical variables. Correlation analysis was conducted to explore associations between vascular changes and hypertension severity. A p-value of <0.05 was considered statistically significant.

Arterial hypertension (AH) is a syndrome of increased clinical blood pressure (BP) in hypertension and symptomatic AH above threshold values determined as a result of epidemiological and randomized controlled studies that have demonstrated an association with increased cardiovascular risk and the feasibility and benefit of treatment aimed at lowering BP below these BP levels. The etiology of hypertension remains not fully understood, but several factors have been identified that are closely and independently associated with increased blood pressure:

- age - increasing age is associated with an increase in the frequency of hypertension and blood pressure (primarily systolic) [10];
- overweight and obesity contribute to increased blood pressure;
- hereditary predisposition - increased blood pressure occurs approximately 2 times more often among individuals in whom one or both parents had hypertension. Epidemiological studies have shown that about 30% of blood pressure variations in different populations are due to genetic factors [11].

- ✓ excessive sodium intake (>5 g/day)
- ✓ alcohol abuse;
- ✓ physical inactivity.
- ✓ A persistent and prolonged increase in blood pressure is caused by a change in the ratio of three
- ✓ hemodynamic indicators:

- ✓ an increase in total peripheral vascular resistance (TPVR);
- ✓ increase in cardiac output (minute volume);
- ✓ increase in circulating blood volume (CBV).

Arterial hypertension (AH) is an important risk factor for the development of structural and functional disorders in all parts of the vascular bed, from the microcirculatory link to large vessels, including the aorta. In recent years, several studies have been conducted to identify patterns of changes in vessels of various calibers in this disease. Endothelial dysfunction is a key mechanism that triggers vascular remodeling processes and worsens the prognosis of arterial hypertension. This diagram shows the main factors secreted by endothelial cells in an extremely simplified form. Normally, the body maintains a balance of opposing processes, which, accordingly, initiate these factors. One of these processes is vasoconstriction, and the other is vasodilation, that is, maintaining vascular tone. The most powerful vasodilator is nitric oxide, which is synthesized by NO synthase in endothelial cells, and the most powerful vasoconstrictors are endothelin and angiotesin-II. When endothelial cells are damaged, a deficiency of nitric oxide develops, and since there is a deficiency, vasoconstrictors begin to prevail and outweigh vasodilators and, ultimately, vasospasm begins to prevail over vasodilation, which is one of the reasons for such an increase in arterial pressure. Of course, the cause-and-effect relationship between arterial hypertension and endothelial dysfunction is very complex and has not been fully studied.

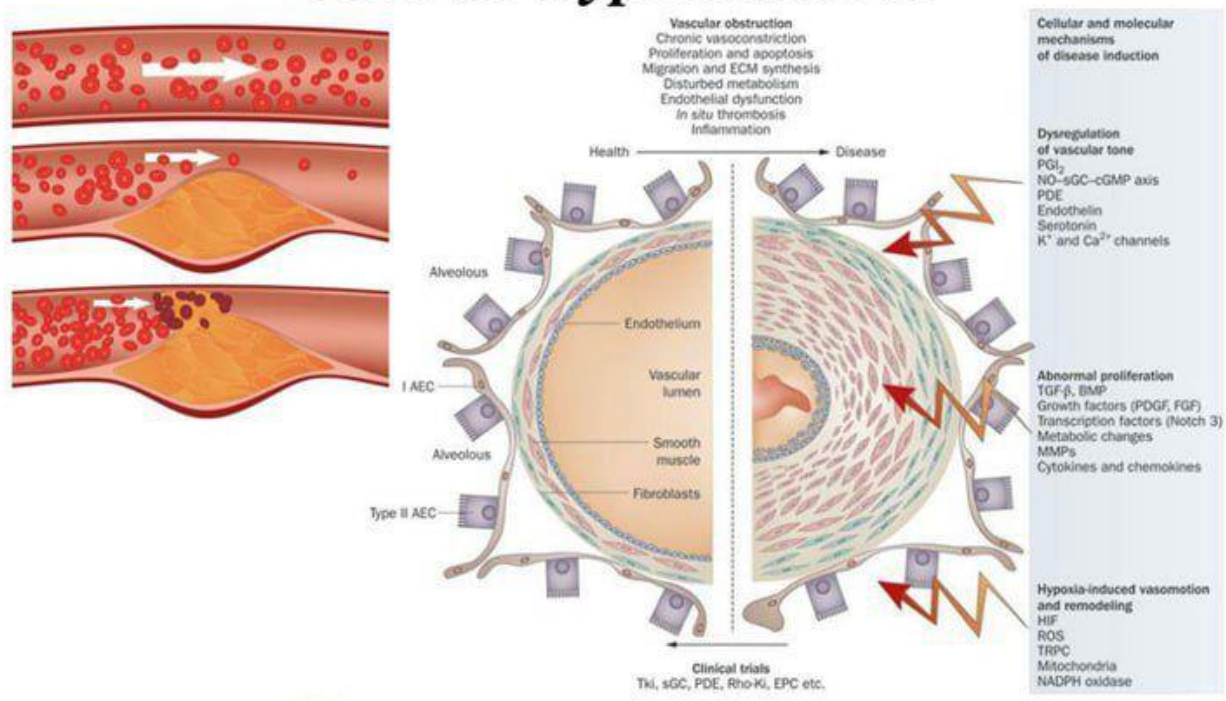
Researchers conditionally distinguish several types of vascular remodeling in hypertension [1].

The first type (wall hypertrophy) is characterized by an increase in the wall thickness/vascular lumen ratio in response to a prolonged increase in blood pressure, either due to hypertrophy of the muscular and subendothelial layers of the vascular wall, or due to the restructuring of its cellular and non-cellular elements. It is observed predominantly in muscular arteries (femoral, brachial, radial, small resistance vessels).

In the second type of remodeling (dilation and general enlargement of the vessel with the formation of aneurysms), due to a consistently increased blood flow rate or a decrease in the cellular composition of the vascular wall and active proteolysis of matrix components, a pronounced increase in the internal and external diameters is observed vessel with a slight change in wall thickness, disorganization of the cellular and non-cellular components of the vascular wall, a decrease in the wall thickness/vascular lumen ratio. This variant is typical for elastic vessels (aorta, carotid artery). The third variant of vascular remodeling (rarefaction of the vascular network) is formed with a long-term decrease in blood flow and consists of a decrease in the thickness of the vessel wall, as well as its internal and external diameters, complete anatomical closure of the vessel. It is observed in the vessels of the microcirculatory link.

At the initial stage of the formation of hypertension, cardiac output increases, while the microvascular network reacts differently to the increase in blood pressure, having, however, one compensatory-adaptive goal - to reduce the degree of load on the capillary wall.

Pathophysiology of vascular tone. Arterial hypertension.



In individuals with high reserve capacity, a reduction in resistance in the microvascular bed and pressure on the capillary wall is possible due to the opening of non-functioning capillaries, as a result of which the volumetric blood flow rate increases. However, with prolonged exposure to high cardiac output on the capillary network, a decrease in the lumen of the resistive vessels occurs, and in some cases, their lumen is completely closed due to the contraction of the precapillary sphincters, which dampen the pressure on the walls of the capillaries. This reaction of muscular vessels in response to an increase in blood pressure is called the “Bayliss effect”, first described in 1902. In this case, a decrease in the cross-sectional area of the lumen of small vessels is accompanied by an increase in vascular reactivity.

Currently, the most frequently used diagnostic methods to assess the state of the microcirculatory system are laser Doppler flowmetry, capillaroscopy of the nail bed and bulbar conjunctiva [2].

Results.

The study analyzed the structural and functional changes in peripheral vessels among patients with arterial hypertension (AH) and ischemic heart disease. Participants were stratified into three groups based on the severity of hypertension: mild, moderate, and severe. Key parameters, including vascular wall thickness, lumen diameter, capillary rarefaction, and vascular stiffness, were measured using non-invasive diagnostic tools. Structural Changes in Peripheral Vessels The findings revealed significant variations in vascular remodeling across the groups. In patients with severe hypertension, vascular wall hypertrophy was observed, with an increased wall-to-lumen ratio compared to the mild and moderate hypertension groups ($p < 0.01$). This hypertrophy predominantly affected muscular arteries, such as the brachial and radial arteries. Conversely, elastic vessels, including the aorta and carotid arteries, exhibited dilation and aneurysmal changes in patients with long-standing hypertension ($p < 0.05$). Rarefaction of the capillary network, marked by reduced capillary density, was more pronounced in the severe hypertension group, particularly in the microvascular beds ($p < 0.001$). Functional Changes in

Vascular Dynamics Endothelial dysfunction was evident across all groups but was more severe in patients with advanced hypertension. Nitric oxide production was significantly lower in these patients, resulting in reduced vasodilatory capacity ($p < 0.01$). Concurrently, levels of vasoconstrictors, such as endothelin and angiotensin-II, were elevated, contributing to increased vascular tone and stiffness. Pulse wave velocity (PWV), a marker of vascular stiffness, was significantly higher in the severe hypertension group compared to other groups ($p < 0.001$). Correlation Between Vascular Remodeling and Hypertension Severity Correlation analysis demonstrated a strong inverse relationship between capillary density and vascular stiffness ($r = -0.78$, $p < 0.001$). Additionally, an increase in peripheral resistance was associated with greater structural alterations in the microcirculatory network, further aggravating hypertension severity. The "Bayliss effect," characterized by the contraction of precapillary sphincters in response to elevated blood pressure, was prominent in patients with moderate to severe hypertension. Summary of Key Findings Significant wall hypertrophy in muscular arteries and dilation in elastic arteries were observed in patients with severe hypertension. Microvascular rarefaction and increased vascular stiffness were key features of advanced hypertension. Endothelial dysfunction, marked by reduced nitric oxide levels and elevated vasoconstrictor activity, was a common finding. A strong inverse correlation existed between capillary density and vascular stiffness, highlighting the interdependence of micro- and macrovascular changes. Disturbances in various parts of the vascular bed in hypertension are apparently interconnected. At the same time, changes in large vessels, affecting, in particular, such an indicator as PWV, lead to changes at the microcirculatory level. And, conversely, an increase in peripheral resistance and rarefaction of the capillary network increase the rigidity of the vascular wall, aggravating the course of hypertension.

According to the results of the study conducted by H. Debbabi et al., an inverse correlation was found between the vascular wall stiffness parameter and the capillary network density. In patients with essential hypertension, capillary network rarefaction was influenced by cardiovascular risk factors such as obesity and smoking.

Discussion

The findings of this study provide significant insights into the structural and functional changes in peripheral vessels associated with arterial hypertension (AH) and ischemic heart disease. The observed vascular remodeling patterns, including wall hypertrophy, dilation, and capillary rarefaction, underscore the multifaceted nature of hypertension-induced vascular damage. These results align with previous research emphasizing endothelial dysfunction as a central mechanism driving these changes. The imbalance between vasodilators, particularly nitric oxide, and vasoconstrictors, such as endothelin and angiotensin-II, was evident across all severity levels of hypertension, further corroborating earlier findings on the role of endothelial dysfunction in vascular remodeling.

The relationship between hypertension severity and increased vascular stiffness, as indicated by elevated pulse wave velocity (PWV), highlights the progressive nature of vascular damage. These results are consistent with studies demonstrating that structural changes in the vascular wall, such as increased wall-to-lumen ratios and microvascular rarefaction, contribute to elevated peripheral resistance and sustained hypertension. The strong inverse correlation between capillary density and vascular stiffness emphasizes the interconnectedness of macro- and microvascular changes, offering new perspectives on the systemic impact of hypertension.

From a clinical perspective, these findings have important implications for the diagnosis and management of hypertension-related vascular complications. Non-invasive diagnostic tools, such as laser Doppler flowmetry and capillaroscopy, offer valuable insights into the state of the microcirculatory system and could serve as early indicators of vascular damage. Interventions aimed at restoring endothelial function, such as lifestyle modifications and pharmacological treatments targeting nitric oxide pathways, may help mitigate vascular remodeling and improve outcomes in hypertensive patients.

Despite the contributions of this study, several limitations warrant consideration. The cross-sectional design precludes the establishment of causality between hypertension severity and vascular changes.

Additionally, the study's reliance on non-invasive diagnostic methods, while practical, may lack the precision of invasive techniques in quantifying microcirculatory parameters. The sample size, particularly in subgroup analyses, may also limit the generalizability of the findings to broader populations. Future studies should consider longitudinal designs and larger, more diverse cohorts to address these limitations.

Looking ahead, research should focus on developing quantitative criteria for assessing microcirculatory changes and their relationship with hypertension progression. The role of emerging biomarkers in diagnosing endothelial dysfunction and guiding therapeutic interventions is another promising avenue. Furthermore, exploring the bidirectional relationship between vascular changes and hypertension could yield deeper insights into disease mechanisms and inform the development of personalized treatment strategies.

In conclusion, this study reinforces the critical role of vascular remodeling in the pathophysiology of arterial hypertension and highlights the need for comprehensive diagnostic and therapeutic approaches. By addressing the interconnected changes across vascular calibers, clinicians and researchers can better understand and manage the complexities of hypertension, ultimately improving patient outcomes.

Conclusion: Despite significant achievements in understanding the nature of vascular damage in hypertension, many unanswered questions remain. Firstly, the lack of quantitative criteria for assessing changes in microcirculatory vessels, as well as volumetric and speed characteristics of microcirculation, continues to hinder precise evaluation and standardization in clinical practice. Secondly, there is insufficient evidence to definitively determine whether microcirculation disorders are a cause or a consequence of hypertension, leaving a critical gap in understanding the disease's pathophysiology.

This study highlights the interconnected nature of vascular remodeling across different calibers, from microvascular rarefaction to increased stiffness in large vessels. These changes collectively exacerbate hypertension and its systemic impact, reinforcing the need for early diagnosis and targeted therapeutic strategies. Advances in diagnostic tools, such as laser Doppler flowmetry and capillaroscopy, have shown promise but require further validation and refinement for widespread clinical use.

The findings emphasize the importance of addressing endothelial dysfunction as a central mechanism in hypertension-induced vascular remodeling. Restoring the balance between vasodilators and vasoconstrictors could play a pivotal role in mitigating vascular damage. Moreover, interventions focusing on lifestyle modifications and pharmacological approaches tailored to individual patient profiles may offer effective strategies for reducing the burden of hypertension-related complications.

Looking ahead, future research should aim to establish standardized quantitative metrics for evaluating microcirculatory changes and explore the longitudinal impact of these changes on disease progression. Additionally, the potential of emerging biomarkers and precision medicine approaches in diagnosing and managing vascular dysfunction in hypertension warrants further investigation. By addressing these gaps, it will be possible to advance the understanding and treatment of hypertension, ultimately improving clinical outcomes and enhancing patients' quality of life.

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