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TABLE OF CONTENTS

AGRICULTURAL SCIENCES

BIOLOGICAL SCIENCES

MEDICAL SCIENCES

THE ROLE OF ENDOTHELIAL DYSFUNCTION BIOMARKERS IN THE TREATMENT STRATEGY FOR PATIENTS WITH HYPERTENSION

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Introduction.

Hypertension (HT), defined as a chronic elevation of systemic blood pressure, serves as a primary modifiable risk factor for cardiovascular disease and a leading cause of global morbidity and mortality. The substantial burden of this condition poses a critical public health challenge, particularly in Ukraine. This situation highlights the urgent necessity for advanced prognostic tools to optimize disease management [1, 2].

Endothelial dysfunction (ED) is a fundamental pathophysiological process driving the onset and progression of numerous disorders, particularly cardiovascular diseases. Its presence highlights the universal albeit nonspecific role of ED in the pathogenesis of various pathological conditions. The impaired bioavailability of nitric oxide (NO) is considered the principal manifestation of ED and is central to understanding its underlying mechanisms. Indeed, compromised endothelial function is a primary mechanism of cardiovascular pathology. ED often arises from systemic inflammation, even in the absence of traditional atherogenic factors, significantly influencing the risk of atherosclerosis development and progression [3, 4].

Endothelial dysfunction plays a central role in the pathogenesis and progression of arterial hypertension and its associated cardiovascular complications.

Crucially, ED often precedes the clinical onset of hypertension, rendering it a vital target for early detection and intervention. This condition is characterized by reduced NO bioavailability, leading to impaired vasodilatation and dominant vasoconstriction. Consequently, a pro-hypertensive environment is established, defined by elevated vascular resistance, inflammatory pathway activation, and vascular remodelling. Therefore, identifying specific ED biomarkers is essential for developing personalized prevention strategies [5, 6].

The aim of the work is to analyze role of endothelial dysfunction biomarkers in the treatment strategy for patients with hypertension.

Materials and Methods. We performed a retrospective analysis of publications from 2015–2025 using PubMed, Scopus, and Web of Science. The search focused on endothelial dysfunction biomarkers in hypertension management, utilizing keywords such as 'hypertension', 'endothelial dysfunction', 'biomarker', and 'hypertensive crisis'. Selection criteria included publication recency, journal reputation, and citation metrics.

Results. Modern science defines the endothelium not simply as a structural barrier, but as a massive, metabolically active organ weighing 1.5–1.8 kg with a surface area of roughly 6000–7000 m^2. Acting through endocrine, paracrine, and autocrine pathways, it serves as a critical regulator of vascular homeostasis. Therefore, endothelial dysfunction is correctly understood as a systemic failure rather than a local injury. This perspective underscores the prognostic value of ED biomarkers in managing patient health and preventing complications [7, 8].

The relationship between endothelial dysfunction and hypertension is a bidirectional, self-perpetuating vicious cycle. Elevated blood pressure induces vascular inflammation that exacerbates ED, which conversely sustains hypertension. Therefore, effective management requires a multifaceted strategy that not only controls blood pressure but also targets endothelial restoration to break this pathological loop and prevent complications [9, 10].

The healthy endothelium serves as a master regulator of vascular homeostasis and blood pressure, exerting vital antioxidant, anti-inflammatory, and antithrombotic

effects. However, the transition to endothelial dysfunction involves complex pathophysiological mechanisms. These processes are clinically reflected by distinct alterations, both upregulation and downregulation – in specific biomarkers [11, 12].

Endothelial dysfunction is characterized by an imbalance of vasoactive substances: a critical reduction in the vasodilator NO and a corresponding increase in vasoconstrictor endothelin-1. Synthesized by endothelial NO-synthase (eNOS) from L-arginine, NO provides vital anti-atherosclerotic protection by promoting vasodilation, inhibiting cell proliferation, and preventing adhesion [13, 14].

Endothelial dysfunction biomarkers are promising tools for monitoring treatment efficacy and tailoring therapeutic strategies in hypertension. Changes in their levels reflect the therapeutic response, as exemplified by I. M. Fushtey et al.'s findings: successful treatment correlated with decreased endothelin-1 levels and increased nitric oxide metabolites (NO_3+NO_2) concentrations [15, 16].

Biomarkers of endothelial dysfunction are categorized as soluble or cellular indicators. Their ability to reflect acute pathophysiological changes at the vascular wall level makes them crucial for improving risk stratification in patients with uncomplicated hypertensive crises. Below, we detail the key ED biomarkers applicable in this acute setting [17, 18].

Nitric oxide is central to vascular tone and homeostasis, making reduced bioavailability its defining feature in ED. Since NO is an unstable molecule with a short half-life, direct clinical measurement is impractical. Consequently, the assessment of endogenous NO relies on quantifying its stable breakdown products: nitrates and nitrites [19, 20].

Soluble cell adhesion molecules (sCAMs), including sICAM-1, sVCAM-1, and E-selectin, are shed proteins that signal endothelial activation and injury. Elevated levels, frequently detected in arterial hypertension, are critical because sCAMs facilitate the recruitment of inflammatory cells to the vascular wall, driving vascular inflammation. Their measurement thus provides valuable insight for risk assessment and therapeutic monitoring [21, 22].

Von Willebrand factor (vWF), a large multimeric glycoprotein synthesized

primarily by endothelial cells, is central to hemostasis by mediating platelet adhesion. Elevated vWF levels are a widely recognized biomarker of endothelial injury and dysfunction in patients with hypertension and cardiovascular disease. Furthermore, vWF acts as an independent predictor of adverse cardiovascular events in hypertensive patients [23, 24].

The hypertensive crisis represents a common and acute complication of hypertension, characterized by an abrupt and significant elevation in blood pressure. HC is categorized into two distinct clinical presentations: hypertensive urgency, defined by severe blood pressure elevation in the absence of target organ damage, and hypertensive emergency, which involves acute target organ damage. This fundamental clinical distinction dictates fundamentally different approaches to both emergency stabilization and ongoing care [25, 26].

Endothelial dysfunction biomarkers may offer significant prognostic value for predicting complications in the management of HT. This utility is particularly crucial during a hypertensive crisis. The precise role of specific biomarkers in reliably predicting complications and improving short-term risk stratification during uncomplicated hypertensive crisis remains to be established [27].

In conclusion, endothelial dysfunction biomarkers offer a promising strategy for precise risk stratification of complications during a hypertensive crisis. Their ability to detect subclinical target organ damage is critical for timely intervention, thus preventing irreversible injury. Beyond prognostication, these biomarkers are vital for optimizing and personalizing treatment strategies in patients with arterial hypertension, ultimately contributing to improved clinical outcomes.

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