Volume 3, Issue 12, December 2025 ISSN (E): 2938-3765

THE IMPORTANCE OF RISK FACTORS IN THE DEVELOPMENT OF ARTERIOSCLEROSIS IN UNCONTROLLED ARTERIAL HYPERTENSION

Шукурова Дилафруз Юсуфовна
Тошкент давлат тиббиёт университети. Ўзбекистон, 100109,
Тошкент, Олмазор тумани, Фаробий кўчаси 2, тел: +99878 1507825
Орсид: 0009-0007-5598-8537

Abstract
The article discusses the effect of uncontrolled arterial hypertension on the development of arteriosclerosis and analyzes the main risk factors. It is noted that a prolonged increase in blood pressure leads to morphological changes in the vascular wall, endothelial dysfunction, lipid metabolism disorders and an increase in blood viscosity. In addition, factors such as age, heredity, unbalanced diet, lack of physical activity and stress accelerate the development of arteriosclerosis. The paper emphasizes the importance of blood pressure control and risk factor correction for the prevention of cardiovascular disease.

Keywords: Arterial hypertension, arteriosclerosis, risk factors, endothelial dysfunction, lipid metabolism disorders, vascular system, prevention, control of blood pressure.

Introduction

Relevance

It is known that both modifiable and non-modifiable risk factors play a significant role in the pathogenesis of arterial hypertension (AH) and in target-organ damage. In particular, among the non-modifiable risk factors such as sex and age, studies have shown that cardiovascular diseases (CVD) and complications occur earlier in men with AH compared to women.

The Aim of the Study:

The aim is to identify the main risk factors influencing the development of arteriosclerosis in uncontrolled arterial hypertension, to analyze their pathogenetic role, and to determine preventive and effective control measures.

With increasing age, not only the arteries but also the heart, brain, kidneys, and retina undergo physiological changes; however, when modifiable risk factors are added to this process, they lead to subclinical damage of target organs, which has been substantiated in studies [1]. At the same time, research has proven that reducing modifiable risk factors in the development of cardiovascular diseases (CVD)—such as eliminating excess weight and obesity, increasing the consumption of fruits and vegetables, limiting excessive salt intake to less than 5 g/day as recommended by WHO, increasing physical activity, reducing tobacco use, and decreasing alcohol consumption—can lower arterial blood pressure (BP) and reduce overall risk [2].

Obesity, especially abdominal obesity, is highly prevalent and is considered one of the components of metabolic syndrome. In obesity, increased activity of the sympathoadrenal and renin–angiotensin systems, the development of insulin resistance, and hyperinsulinemia lead not only to elevated blood pressure but also to target-organ damage, including the development of arteriosclerosis in the arteries [3]. Another component of metabolic syndrome—hypertriglyceridemia—has been identified as a factor that increases not only the risk of arteriosclerosis but also cardiovascular risk [4]. Based on the above, the aim of the study was defined as investigating the impact of common risk factors on arterial damage—particularly the development of arteriosclerosis—in patients with uncontrolled arterial hypertension.

Materials and Methods of the Study

Our study included 143 patients with uncontrolled arterial hypertension (grades 1–3). The average age of all participants was 63.1 ± 8.9 years. All individuals enrolled in the study underwent physical, functional, and laboratory examinations according to the research protocol.

To assess the clinical condition, the following cardiovascular risk factors were evaluated: systolic and diastolic blood pressure (SBP and DBP), smoking status, body mass index (BMI = weight (kg) / height² (m²)), blood lipid profile—including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C)—serum glucose level, salt taste sensitivity test results, urinary electrolyte levels (Na, K, Cl), and microalbuminuria (MAU), all determined using biochemical methods.

Among functional tests, the following were performed: electrocardiography, echocardiography, and carotid ultrasound of the brachiocephalic arteries to measure carotid intima–media thickness (IMT). Left ventricular hypertrophy (LVH) was diagnosed based on left ventricular myocardial mass indexed to body surface area. The arteriosclerosis process was assessed by pulse wave velocity (PWV) using applanation tonometry on the "Sphygmocor" device. In both patients and healthy individuals, the threshold of salt taste sensitivity was determined using the Henkin test.

Exclusion Criteria

Patients with ischemic heart disease, chronic heart failure, complex cardiac arrhythmias, peripheral arterial disease, cerebrovascular complications (history of transient ischemic attack or acute cerebrovascular accident), diabetes mellitus, and severe comorbid conditions were not included in the study.

Statistical Analysis

Statistical processing of the results was performed using Microsoft Office Excel 2007 and Statistics 6.0 for Windows. The χ^2 test was used to evaluate the significance of differences between qualitative variables. The reliability of correlations was assessed using Spearman's correlation analysis. For all types of analyses, $p<0.05$ was considered statistically significant.

Obtained Results

Screening and physical examination of patients with arterial hypertension revealed that 42% of patients were not under follow-up at their local outpatient clinic and did not take antihypertensive

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

medications. Among those surveyed, 28% were aware of their cardiovascular risk factors, 87% had a hereditary predisposition to cardiovascular diseases, 67% showed low physical activity, almost all patients with hypertension were overweight (BMI >25 kg/m²), and 67% of them were diagnosed with obesity. The salt taste sensitivity threshold was elevated in nearly three-quarters of the patients, indicating a tendency toward salt-sensitive hypertension in the examined hypertensive population (Figures 1 and 2).

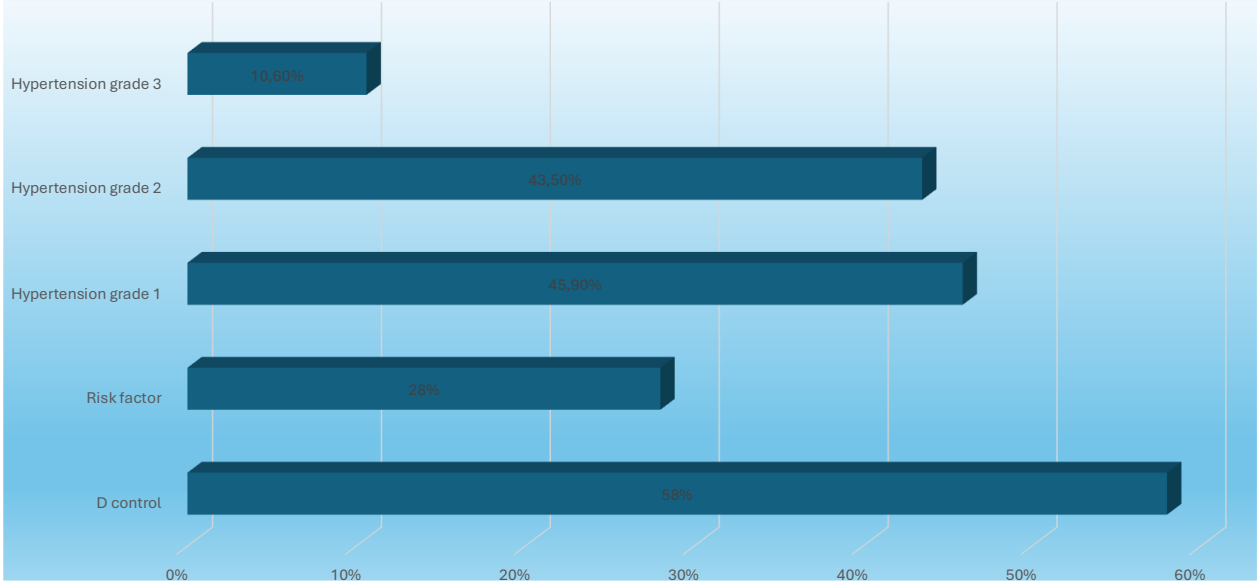

Figure 1. Hypertension grade among patients, their follow-up status (dispensary observation), and awareness of risk factors (RF).

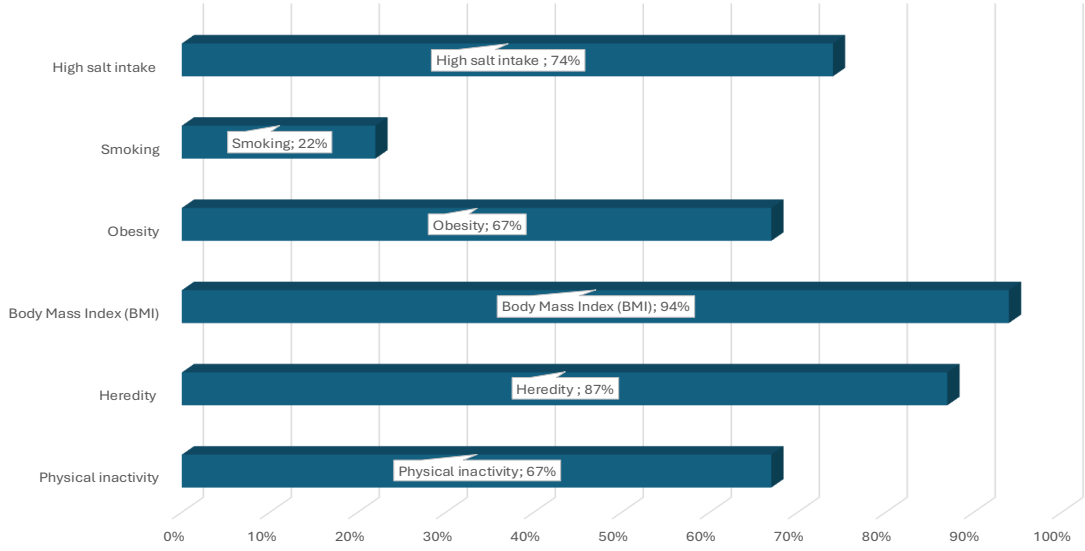

Figure 2. Prevalence of identified risk factors in patients with arterial hypertension.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Patients included in the study were assessed as having a high cardiovascular (CV) risk stratification based on the detected target-organ damage, and they were recommended lifestyle modification and combination antihypertensive pharmacotherapy. In more than 70% of patients, echocardiographic findings typical of hypertensive heart disease—left ventricular hypertrophy (LVH) and left ventricular diastolic dysfunction (LVDD)—were identified, along with carotid artery remodeling characterized by increased intima–media thickness (IMT). Microalbuminuria (MAU), an indicator of hypertensive nephrosclerosis, was diagnosed in 20% of the patients (Figure 3). The clinical characteristics of the examined patients are presented in Table 1.

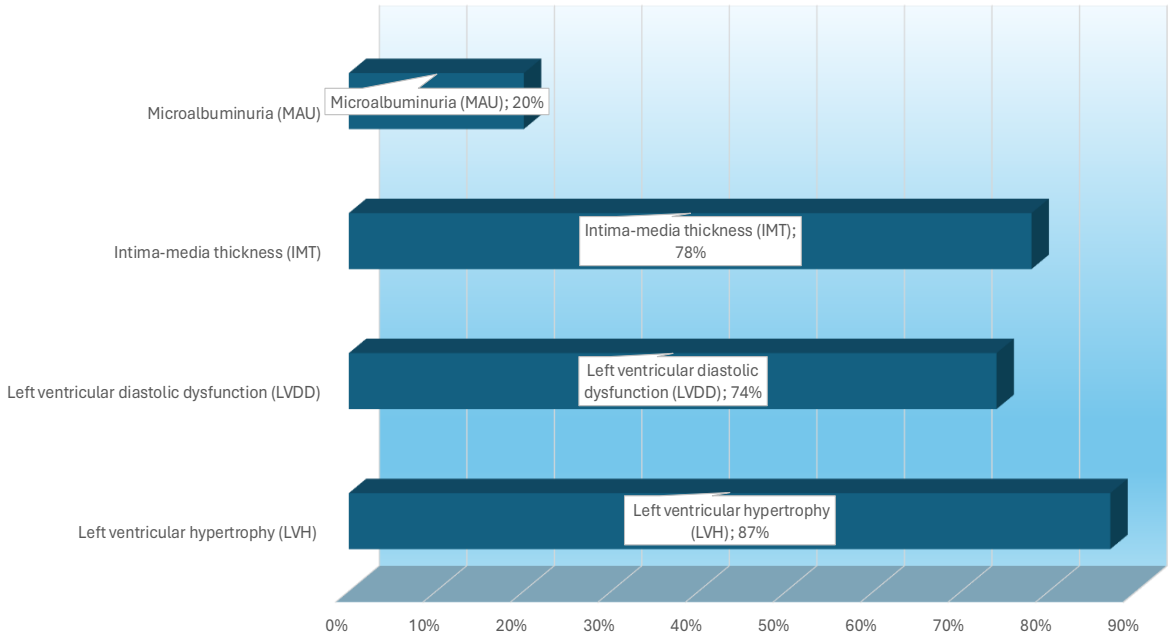

Figure 3. Prevalence of target-organ damage in patients with arterial hypertension.

Note: LVH – left ventricular hypertrophy; LVDD – left ventricular diastolic dysfunction; IMT – carotid intima–media thickness; MAU – microalbuminuria.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Table 1. Clinical, functional, and biochemical parameters of the examined patients.

Indicators	Patients with hypertension n=143 M±SD (Me [Q1; Q3])
Patient age, y.	55.68±9.8 (49 [58; 73])
Duration of hypertension, y.	10.7±6.6 (5 [10; 44])
Hypertension grade, %: Grade 1 Grade 2 Grade 3	 45.9% 43.5% 10.6%
BMI (kg/m²)	32.3±5.07 (22.8 [32; 46])
SBP (mm Hg)	171.06±17.7 (160 [170; 240])
DBP (mm Hg)	100.06±8.7 (100 [100; 140])
Mean BP (mm Hg)	122.9±9.9 (120 [123; 173])
PWV>10 m/s	10.9±2.4 (9.4 [10.8; 22.8])
LVMI, g/m²	136.3±34.1 (133.0 [113.3; 276.2])
IMT, mm	1.027±0.19 (1 [0.9; 1.5])
Glucose, mmol/L	6.14±2.29 (5 [5.4; 16.5])
Creatinine, µmol/L	94.5±22.2 (79 [92; 207])
GFR <60 ml/min/1.73 m2	70.25±17.1 (57.15 [67.4; 120.68])
Uric acid, mg/dL	6.25±1.65 (5.1 [6.2; 11.2])
Total cholesterol, mg/dL	205±47.8 (169 [205; 364])
TG, mg/dL	166.8±83.2 (105.25 [151; 407])
LDL-C, mg/dL	122.02±43.6 (100 [123; 274])
HDL-C, mg/dL	44.26±11.1 (37 [43; 97])
MAU	38.08±42.9(9.02 [22.8; 251.2])

Note: AH – arterial hypertension; BMI – body mass index; SBP – systolic blood pressure; DBP – diastolic blood pressure; PWV – pulse wave velocity; LVMI – left ventricular myocardial mass index; IMT – intima–media thickness; GFR – glomerular filtration rate; TG – triglycerides; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; MAU – microalbuminuria.

As a result of studying the relationship between risk factors and target-organ damage with the progression of arteriosclerosis, the influence of risk factors and markers of target-organ damage on pulse wave velocity (PWV), an indicator of early vascular aging, was analyzed.

In our study, significant correlations were identified between risk factors and PWV, a predictor of vascular aging, in 143 patients with arterial hypertension. Specifically, moderate and statistically significant correlations were observed between PWV and the following variables: patient age ($r = 0.383$, $p = 0.0001$), smoking ($r = 0.44$, $p = 0.021$), systolic blood pressure (SBP) ($r = 0.15$, $p = 0.046$), serum glucose level ($r = 0.2$, $p = 0.008$), carotid intima–media thickness (IMT) ($r = 0.3$, $p = 0.0001$), and microalbuminuria (MAU) ($r = 0.4$, $p = 0.034$).

Discussion

Pulse wave velocity (PWV) plays a major role in the phenomenon of arterial stiffness, including the most elastic artery—the aorta. The pulse wave results from the summation of two waves: the

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

forward wave, directed from the aorta toward distal parts of the arterial tree, and the reflected wave, which travels backward from the aortic bifurcation opposing the forward wave. Due to its high elasticity, the aorta can dampen the stroke volume and transfer part of the energy generated by ventricular contraction into the diastolic phase. This leads to a reduction in systolic blood pressure and an increase in diastolic blood pressure in the aorta, thereby decreasing the damaging effect of pulse waves on cerebral, cardiac, and renal vessels and improving tissue perfusion [5].

In cardiovascular diseases, the phenomenon of aortic stiffness alters this process. The reflected pulse wave, instead of being dampened at the aortic bifurcation, is not attenuated and adds to the primary pulse wave. As a result, systolic blood pressure in the aorta rises, the central systolic–diastolic pressure difference increases, and central pulse pressure—an established predictor of cardiovascular complications—also increases.

Arterial stiffness is assessed non-invasively using applanation tonometry based on the measurement of pulse wave velocity (PWV). In this method, the transit time between two pulse waves recorded at different segments of major arteries separated by a known distance (D) is measured. Specifically, the time from the initial upstroke of the first wave (at the carotid artery) to the upstroke of the second, more distal wave (at the femoral artery) is recorded, yielding the carotid–femoral pulse wave velocity. Measurement of PWV is a simple, reproducible method applied in clinical practice and is considered the "gold standard" for evaluating aortic stiffness. In applanation tonometry, a PWV value greater than 10 m/s indicates target-organ damage and is suggestive of the development of arteriosclerosis [6].

As noted, early aging of elastic arteries, i.e., arteriosclerosis, reflects target-organ (vascular) damage in arterial hypertension, and its close association with systolic blood pressure (SBP), pulse pressure, various risk factors, and markers of target-organ damage has been documented in the literature [7]. Diagnosis of early vascular aging and arteriolosclerosis using applanation tonometry, which provides quantitative indices, is primarily conducted in large specialized centers. Although assessing vascular age, vascular remodeling, treatment-induced changes, and the degree of target-organ damage provides high diagnostic value for clinicians, it is not included in routine examinations at the primary care level, such as in family or multi-specialty outpatient clinics.

Consequently, at present, it is recommended that primary care physicians perform risk stratification for all hypertensive patients. When three or more risk factors and evidence of target-organ damage are identified, patients should be classified as having high or very high cardiovascular risk. In such cases, the use of antihypertensive agents providing vasoprotection (vascular protection) is advised, taking into account the progression of arterial damage and arteriosclerosis. Our results indicate that controlling modifiable risk factors through both pharmacological and non-pharmacological measures can influence the progression of arteriosclerosis, as reflected in the observed significant correlations.

The results of our study demonstrated that in 143 patients with grades 1–3 uncontrolled arterial hypertension, modifiable risk factors and target-organ damage were highly prevalent. Although non-modifiable risk factors such as patient age cannot be influenced, modifiable risk factors—such as smoking, elevated systolic blood pressure, fasting glucose levels, and markers of target-organ damage including carotid intima–media thickness (IMT) and microalbuminuria (MAU)—can be favorably affected. Promoting a healthy lifestyle and implementing combination antihypertensive

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

therapy to achieve target blood pressure levels can positively influence the progression of arteriosclerosis.

References

1. Kurbanov, R.D., Khamidullaeva, G.A. Arterial Hypertension. Monograph. Tashkent, 2017. 356 p.
2. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. European Heart Journal. 2018; 39(33):3021– 104. DOI: 10.1093/eurheartj/ehy339.
3. Kurbanov R.D., Srozhidinova N.Z. Metabolic syndrome. Methodical manual. Tashkent 2016
4. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison, Himmelfarb C et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/, APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2018; 71(6):1269–324.
5. Tursunova NB, Nizamov UI, Kurbanov RD, Khamidullaeva GA, Abdullaeva GJ, Shek AB. //The relationship between the parameters of blood pressure variability and arterial wall stiffness in patients with arterial hypertension. International Journal of Biomedicine 2019, V 9, Issue 3: 197-201
6. Lu Y, Pechlaner R, Cai J, Yuan H, Huang Z, Yang G, et al. Trajectories of Age-Related Arterial Stiffness in Chinese Men and Women. J Am Coll Cardiol 2020; 75:870–880
7. Agbaje AO, Barker AR, Tuomainen TP. Effects of arterial stiffness and carotid intima-media thickness progression on the risk of overweight/obesity and elevated blood pressure/hypertension: a cross-lagged cohort study. Hypertension (Dallas, Tex: 1979) 2022; 79:159–169.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5