

## ARTERIAL HYPERTENSION AND ARTERIAL WALL STIFFNESS IN CLINICAL PRACTICE: LITERATURE REVIEW

Bazarova Sabina<sup>1</sup>

Makhmatmuradova Nargiza<sup>2</sup>

*Samarkand State Medical University*

### KEYWORDS

arterial hypertension, arterial stiffness, pulse blood pressure, smoking cessation.

### ABSTRACT

Arterial stiffness, as a marker of subclinical target organ damage in patients with hypertension, is an important and independent predictor of mortality and cardiovascular morbidity. The review examines factors contributing to increased vascular wall stiffness with a focus on smoking, pathogenesis of increased arterial stiffness with aging, and the effect of arterial stiffness on increased systolic and pulse pressure.

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Arterial hypertension (AH) still occupies a leading place in the structure of morbidity and mortality from cardiovascular diseases (CVD). The prognosis of patients with hypertension primarily depends on the blood pressure level (AH) and target organ damage (TOD). One of the criteria for subclinical vascular TOD is arterial stiffness [1]. For a long time, it was believed that hypertension leads to thickening and rigidity of the central arteries (i.e., rigidity is a trace of increased blood pressure), while more recent evidence suggests that rigidity may precede hypertension (i.e., rigidity is the cause). Currently, evidence has been obtained for the possibility of vascular stiffness being both a cause and a consequence of hypertension [2]. Hypertension and vascular stiffness increase with age and are closely related to aging processes. High blood pressure can cause vascular damage and fragmentation of elastin, which contributes to increased stiffness. On the other hand, aortic rigidity increases systolic pressure. BP (SAH), which leads to an increase in pulse pressure HELL. Hypertension and aging can have an additive effect, as evidenced by the presence of stiffer arteries in elderly patients with hypertension, compared with patients with normal blood pressure levels of the corresponding age [3]. Both aging and hypertension are associated with structural, mechanical, and functional changes in the vascular wall, characterized by increased arterial

<sup>1</sup> 1st-year Master's degree student at Samarkand State Medical University

<sup>2</sup> Scientific Supervisor: PhD, Assistant of the Department of Internal Medicine №4 of SamSMU

stiffness, decreased elasticity, impaired extensibility, and endothelial dysfunction. and increased vascular tone [4]. It should be noted that the risk of developing cardiovascular events and increased vascular stiffness increases with age in both normotensive patients and patients with hypertension [5-6].

### **Research methodology**

The search for literary sources was carried out in the following electronic libraries: elibrary.ru, Pubmed/MEDLINE. The key words for searching literary sources were: arterial stiffness, pulse blood pressure, smoking, smoking cessation.

### **Results**

#### **Causes of increased vascular stiffness**

The reason for the age-related increase in vascular stiffness is a change in the extracellular matrix of blood vessels as a result of an increase in collagen content and a decrease in elastin content, a violation of the structure of collagen and elastin, calcium deposition, endothelial dysfunction and a change in the number of vascular smooth muscle cells [7]. The main mediator of increased vascular stiffness is the destruction of elastin under the action of matrix metalloproteinases and serum elastase [8]. In addition, increased salt intake [9] and calcium deposition in the aorta [10] affect vascular stiffness. The effect of calcium deposition in the aorta on vascular rigidity was most pronounced in patients resistant to antihypertensive therapy, suggesting that arterial stiffness not only contributes to the development of isolated systolic hypertension, but may also be involved in resistance to hypertension treatment [9]. The stiffness of the aorta increases with an increase in the number of plaques, therefore, vessel rigidity is closely related to atherosclerosis [11]. However, increased vascular stiffness is also a sign of aging even in the absence of atherosclerosis [12]. Many different factors are involved in the mechanisms of development of increased vascular stiffness associated with atherosclerosis: hypercholesterolemia, overweight/morbid obesity, increased oxidative stress [7, 13, 14]. Aging leads to many changes in the cardiovascular system and is a powerful predictor of adverse cardiovascular events. A distinctive feature of this process is the increased stiffness of the central vessels due to fragmentation of elastin fibers and their replacement with tougher collagen, which leads to an earlier return of the reflected pulse wave, an increase in the direct wave and, consequently, an increase in central SAH, an increase in PAH, an increase in cardiac load conditions and a deterioration in perfusion of vital organs [7]. In most age groups, the cardiovascular risk for men is higher than for women, and balance is achieved only when women reach postmenopause [15]. There are gender differences in the temporal dynamics of arterial stiffness and the associated risk of CVD, which disproportionately increases in postmenopausal women. In postmenopausal women, the association between arterial rigidity and mortality is almost 2 times higher than in men. Understanding the mechanisms that determine sex differences in vascular stiffness, it can help in the development of new sex-specific treatments to reduce the risk of CVD [16].

#### **Hypertension, vascular stiffness, and smoking**

The relationship between smoking and hypertension lies in the commonality of such pathophysiological mechanisms as activation of the sympathetic nervous system, oxidative

stress, and vasopressor effects associated with an increase in inflammatory markers. Smoking also leads to endothelial dysfunction, vascular damage, and increased arterial stiffness, which contributes to the development of hypertension [17,18]. The effect of smoking on arterial elasticity leads to the progression of vascular stiffness of CVD. It is known that the presence of smoking in the general population accelerates vascular aging and contributes to the formation of the syndrome. early vascular aging, and the risk of detecting increased arterial rigidity in smokers is 2 times higher than in non-smokers [19]. Studies on the effect of smoking cessation on arterial stiffness are important. A meta-analysis including thirteen studies showed that smoking cessation significantly reduces arterial stiffness, and this effect was significantly stronger in healthy subjects. These data are important in the prevention of cardiac diseases, since smoking cessation partially reduces or even eliminates the effect of this factor on arterial rigidity. Some weight gain in some patients after smoking cessation does not increase the risk of CVD does not reduce the benefits of smoking cessation in reducing mortality. Consequently, the combination of hypertension and smoking increases vascular stiffness, leads to higher blood pressure in these patients and earlier POM, which affects cardiovascular risk and the risk of death from all causes [20].

**Conclusion:** Arterial stiffness is one of the criteria for lesions of target organs in patients with hypertension and determines the presence of a high risk of cardiovascular complications. With age, there is an increase in the prevalence of hypertension and an increase in vascular stiffness. Arterial stiffness is affected by many risk factors, primarily smoking. The clinical significance of increased vascular stiffness is due to the effect on SAH and especially on decline, since with an increase in PAH, the cardiovascular risk increases significantly.

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