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PROBLEMS OF SYSTEMIC ARTERIAL HYPERTENSION IN RHEUMATOLOGICAL DISEASES

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Abstract
Cardiovascular complications, caused by the development of arterial hypertension and early atherosclerotic damage to blood vessels, are the leading cause of death in rheumatic diseases (RD). The results of the conducted studies convincingly demonstrate the role of the inflammatory process in the occurrence and progression of hypertension in RD. In RD with arterial hypertension, there was hyperactivation of the sympathoadrenal system with an increase in the level of vasoactive amines (adrenaline and noradrenaline) and an increase in the plasma renin level. In RD, the indicators of daily blood pressure monitoring clearly correlated with kidney damage and the activity of the inflammatory process.

Keywords: Arterial hypertension, rheumatoid arthritis, systemic lupus erythematosus.

Introduction

The main methods for detecting hypertension (HT) are “office” measurement of blood pressure (BP) and 24-hour blood pressure monitoring (ABPM), with target organ damage being an important independent predictor of cardiovascular mortality. Markers of asymptomatic target organ damage include albuminuria, increased pulse wave velocity, left ventricular hypertrophy, and changes in the intima-media thickness of the carotid arteries. According to literature, patients with “masked” hypertension have a higher left ventricular myocardial mass index compared to patients with normotension based on “office” measurements or ABPM data. The relationship between systolic BP (SBP), diastolic BP (DBP), target organ damage, and associated clinical conditions is better reflected by ABPM [1].

Cardiovascular complications, caused by the development of arterial hypertension and early atherosclerotic damage to blood vessels, are the leading cause of death in RD. Epidemiological studies on the prevalence of hypertension (HT) in the population and among patients with rheumatoid arthritis (RA) are difficult to compare. Such comparisons are valid only when studies are conducted according to a unified protocol, which assumes a uniform strategy for selecting the population for examination, standardized methods for measuring blood pressure (BP), unified criteria for assessing measurement results, a system for external and internal data quality control, a data rejection computer program, and mathematical-statistical analysis. Therefore, any epidemiological comparisons are conditional. Since the prevalence of HT depends on gender and significantly increases with age, we selected from the few large epidemiological studies on the

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prevalence of HT in the population those that took gender and age into account. The results of the study show that, in general, the prevalence of HT among RA patients is higher than in the population, accounting for 45.24% among RA patients over the age of 18. As in the general population, a significant increase in the incidence of HT with age is observed in RA patients. The age distribution of HT frequency in RA patients has gender-specific features. HT in RA patients is associated with the female gender, and the prevalence of HT in women with RA aged 40-59 years significantly exceeds its population level [2].

Rheumatoid arthritis remains one of the most common joint diseases, and the analysis of literature data indicates a high frequency of cardiovascular pathology in these patients. A high prevalence of hypertension (59.6%) has been observed in patients with rheumatoid arthritis (RA). In most patients (59.7%), the diagnosis of hypertension occurs against the background of already existing RA. There is an increase in the number of cases of hypertension with the duration of RA and the activity of the disease. The high prevalence of hypertension in RA patients requires regular monitoring of blood pressure in all RA patients for the earliest possible diagnosis and timely correction of hypertension. In RA patients, the frequency of regular antihypertensive therapy is lower than in patients with hypertension, which may be related to underestimating the risk of developing cardiovascular complications [3].

The obtained data confirm the opinion that in rheumatoid arthritis (RA), the most common type of hypertension is characterized by significant variability, predominantly elevated systolic blood pressure (SBP), accompanied by tachycardia. When patients were classified based on the degree of nocturnal blood pressure reduction, it was found that patients with optimal nocturnal SBP reduction (dippers) are more common in the group with low disease activity, while the type with insufficient nocturnal SBP reduction (non-dippers) is more frequently seen in patients with high disease activity [4].

RA patients with masked arterial hypertension (MAH) and RA patients with hypertension (HT) had significantly higher blood pressure levels during the night, despite having comparable average daily blood pressure levels, than RA patients without hypertension, individuals in the comparison group, and the control group. This can be explained by the lack of adequate antihypertensive therapy in RA patients, especially in the presence of masked hypertension. In our study, almost one in three patients with hypertension, regardless of the presence of joint pathology, had high nocturnal variability of systolic blood pressure (SBP), while in RA patients without hypertension, nocturnal SBP variability was significantly less frequent [5].

The frequency of “masked” hypertension according to the results of 24-hour blood pressure monitoring (ABPM) is 11%. Most of these patients had high normal or normal blood pressure during office measurements. Most RA patients with hypertension have “uncontrolled masked hypertension” (office blood pressure is controlled, but values obtained from ABPM remain elevated), with high blood pressure not only during the day but also at night, indicating inadequate antihypertensive therapy and increasing the likelihood of cardiovascular complications. The 24-hour blood pressure profile in RA patients with hypertension is characterized by a high frequency of “non-dippers”, who are typically older, have lipid metabolism disorders, worse health assessments from RA patients, and higher pain intensity, which requires special attention in treating these patients [6].

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Another rheumatological disease with fluctuating arterial blood pressure is systemic lupus erythematosus.

Systemic lupus erythematosus (SLE) is an autoimmune rheumatic disease characterized by the hyperproduction of autoantibodies to various components of the nucleus and the formation of immune complexes that cause immunoinflammatory damage to internal organs [8]. Modern diagnostic and treatment methods have significantly increased the life expectancy of such patients, however, the mortality rate among SLE patients remains high. Cardiovascular complications, caused by the development of arterial hypertension (HT) and early atherosclerotic damage to blood vessels, are the leading cause of death in rheumatic diseases [9,11]. HT is found significantly more often in SLE patients than in the general population, resulting in a much higher risk of developing cardiovascular complications: acute myocardial infarction is 10 times more common, stroke 8 times more common; overall mortality from cardiovascular diseases is 17 times higher in these patients [11,12]. Kidney damage plays a significant role in the development of HT, leading to the activation of the renin-angiotensin-aldosterone system (RAAS). The initiating component of the RAAS is the enzyme renin, which, by participating in the formation of angiotensin I, produces a number of non-hemodynamic effects that cause remodeling of the myocardium, vascular wall, and kidneys [9]. In SLE, an increase in the renin activity level in the blood plasma is characteristic, and this is associated with a worse long-term prognosis in terms of developing cardiovascular complications [7,15].

The traditional use of angiotensin-converting enzyme inhibitors (ACE inhibitors) or angiotensin II receptor antagonists (ARBs) does not always achieve complete blockade of the renin-angiotensin-aldosterone system (RAAS). Studies have shown that the inhibition of angiotensin II formation and the suppression of its effects stimulate compensatory increases in renin activity in the blood plasma and its secretion [13]. This phenomenon is known as the “escape phenomenon”—the “escape phenomenon” of the antihypertensive and organ-protective action of RAAS blockers [9]. The introduction of the direct renin inhibitor aliskiren has made it possible to target the initiating enzyme of RAAS, blocking both the hemodynamic and non-hemodynamic effects of renin. In recent years, the efficacy of aliskiren in the treatment of essential and secondary hypertension in patients with chronic kidney diseases has been studied in numerous clinical trials. The results of these studies show not only a significant antihypertensive effect of the direct renin inhibitor but also its organ-protective effect, which is comparable to that of antihypertensive drugs from other classes (ACE inhibitors, diuretics, ARBs) [10,14,16,17], or even exceeds it. However, the possibility of using aliskiren as an antihypertensive therapy in patients with systemic inflammatory diseases has not been studied previously.

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