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Pharmacotherapy For Arterial Hypertension And IHD With Beta-Blockers

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Abstract: Cardiovascular diseases remain the leading cause of morbidity and mortality worldwide, accounting for approximately one-third of all deaths annually. Hypertension, dyslipidemia, obesity, and metabolic syndrome are among the most significant modifiable risk factors contributing to the development of coronary heart disease and other cardiovascular complications. β-blockers have occupied a central role in cardiovascular pharmacotherapy for more than five decades, particularly in the management of hypertension, ischemic heart disease, and chronic heart failure. This study aimed to evaluate the role of β-blockers in the treatment of hypertension and coronary heart disease, with special attention to their clinical efficacy, indications, and metabolic effects. Recent large-scale trials have questioned the use of traditional β-blockers, such as atenolol, as first-line therapy for primary hypertension prevention due to comparatively weaker effects on central aortic pressure and cardiovascular outcomes. Consequently, international guidelines have limited their use as initial antihypertensive agents. However, β-blockers remain strongly recommended in specific patient groups, including those with angina pectoris, previous myocardial infarction, heart failure, tachyarrhythmias, glaucoma, and pregnancy. Particular emphasis is placed on the role of sympathetic nervous system activation in hypertension and metabolic disorders. While classical β-blockers may adversely affect carbohydrate and lipid metabolism, newer vasodilatory agents such as metoprolol, carvedilol, and nebivolol demonstrate improved metabolic profiles and reduced impact on central hemodynamics. Nebivolol, in particular, shows minimal negative metabolic effects, making it preferable in patients with metabolic syndrome, obesity, and diabetes mellitus.

Keywords: β-blocker, metabolic syndrome, hypertension, coronary heart disease.

Introduction: The relevance of this topic lies in the fact that in the modern world, mortality ranks high, exceeding the birth rate. The second half of the 20th century was characterized by a sharp increase in the prevalence of cardiovascular diseases, primarily those associated with complications of atherosclerosis, which are currently the leading cause of death and disability among the working-age population. Suffice it to say that approximately 12 million people die from cardiovascular diseases annually worldwide, and their share of overall mortality is over 50%; in Uzbekistan, this figure is 48%. All this has generated significant interest in the treatment and prevention of cardiovascular pathology. Ischemic heart disease is one of the most common conditions, which can lead to severe and difficult-to-treat conditions such as myocardial infarction, hypertension, and acute and chronic cerebrovascular accidents. Cardiovascular disease, including ischemic heart disease, cerebrovascular accidents, and peripheral arterial occlusive disease, is the most common cause of morbidity, mortality, and disability in industrialized countries (1, 2, 3). Cardiovascular disease is the leading cause of death worldwide, accounting for approximately one-third of all deaths (17.9 million people annually). The most important modifiable risk factors for cardiovascular disease are hypertension, excess body weight, and dyslipidemia. Hypertension is widespread (approximately 40% worldwide) and accounts for approximately 9.4 million deaths annually (5, 6, 7). Dyslipidemia, particularly elevated total cholesterol and low-density lipoprotein cholesterol

levels, is known to increase the risk of cardiovascular disease and cardiovascular mortality. The causes of death are diseases, various accidents, and natural disasters. (3,5,7) Among diseases, cardiovascular diseases rank first, namely, coronary heart disease, arterial hypertension, cardiomyopathy, rheumatic heart disease, infectious myocarditis, and congenital heart defects.(1,4,8) Treatment of these diseases requires properly selected medications that will work promptly, quickly, and without side effects. One such medication is beta-blockers. For more than 50 years, β-blockers have established a strong position in pharmacotherapy, primarily in the treatment of cardiovascular diseases (2,3,6). Their high efficacy has been proven in all forms of coronary artery disease (stable and unstable angina), hypertension, and chronic heart failure (CHF) (1,7,10).

Study Objective: To study the role of β-blockers in the treatment of hypertension and coronary artery disease. Materials and Methods: β-blockers in the treatment of hypertension. In recent years, discussions have continued about the possibility of using β-blockers in the treatment of hypertension for primary prevention. Over the past few years, a number of large studies have been completed, such as LIFE and ASCOT, which examined the treatment of hypertension with β-blockers, namely atenolol and its analogs. The findings of the CAFE trials within the ASCOT framework were worse in terms of impact on prognosis than therapy using drugs from other groups (ACE inhibitors, calcium channel blockers, angiotensin receptor blockers) (8,9). These and other similar results of the studies conducted served as the basis for excluding β-blockers as first-line drugs in the treatment of hypertension. In addition, as was shown in sub-studies, therapy based on taking atenolol was significantly inferior to a therapy regimen based on taking the calcium antagonist amlodipine in the ability to reduce central aortic pressure. It was central aortic pressure that was independently associated with the risk of adverse outcomes in multivariate analysis. Despite the exclusion of β-blockers as first-line drugs from hypertension treatment regimens, the British guidelines, as revised in 2011, still allow the use of β-blockers in certain groups of patients:

• In patients with angina pectoris;

• In patients who have had a myocardial infarction;

• In patients with signs of heart failure;

• In patients with tachyarrhythmias;

• In patients with glaucoma (this is a new indication not available for other groups of antihypertensive drugs);

• In pregnant women.

Activation of the sympathetic nervous system is one of the leading pathogenetic mechanisms for increased blood pressure (BP). Moreover, an increase in heart rate often accompanies metabolic disorders such as carbohydrate metabolism disorders, diabetes mellitus, and metabolic syndrome, and reflects the constant sympathetic stimulation characteristic of these conditions. Tachycardia can be not only a marker but also a predictor of diabetes mellitus and metabolic syndrome. This relationship is mediated by the effects of activation of all three β-adrenergic receptor subtypes, as well as stimulation of alpha-adrenergic receptors, causing arteriolar spasm. A large study showed that the use of β-blockers for the treatment of arterial hypertension is associated with a reduction in the risk of all major cardiovascular events and a decrease in heart rate. All these data prompt consideration of the use of β-blockers in patients with tachycardia. The second significant limitation of the use of β-blockers was their side effects, primarily metabolic ones. The ability to influence carbohydrate and lipid metabolism, increasing blood glucose and lipid levels, provided grounds in the 2007 Recommendations of the European Society of Cardiology to limit the use of β-blockers, especially in combination with thiazide diuretics in patients with diabetes mellitus and metabolic syndrome. However, the 2013 guidelines presented at the European Society of Hypertension Congress in June 2013 included caveats regarding vasodilatory beta-blockers. These medications include metoprolol, carvedilol, and nebivolol. These medications can reduce central aortic pressure and arterial stiffness. Furthermore, it is noted that nebivolol has virtually no adverse metabolic effects.

Thus, this drug can be considered preferable for patients with metabolic syndrome, obesity, and diabetes. Beta-blockers used in the treatment of coronary heart disease block β1-adrenergic receptors in the heart and conduction system, reducing atrioventricular conduction and automatism. This results in decreased heart rate, stroke volume, and

cardiac output, as well as myocardial oxygen demand, and lower blood pressure. The hypotensive effect is also associated with a decrease in renin formation under the influence of β-blockers. Beta-blockers have an antiarrhythmic effect, which is explained by blocking β1-adrenergic receptors in the cardiac conduction system, reducing sympathetic influences on the heart, and retaining potassium in intracellular membranes. They also have an antianginal effect, reducing myocardial oxygen demand, have a positive effect on coronary circulation, improve blood supply to ischemic areas, and reduce the sensitivity of the heart muscle to sympathetic influences.

Based on data from studies conducted by the European Society of Cardiology (ESC), the role of β-blockers in coronary artery disease was examined. In contrast to the drug treatment regimen for arterial hypertension discussed above, β-blockers clearly occupy a leading position in the pharmacotherapy of patients with all forms of coronary artery disease, including stable angina. This primarily applies to their use in patients after myocardial infarction. However, while the duration of β-blocker use after an acute coronary event was previously unlimited, this position is currently being revised, and the duration of β-blocker use is determined by additional conditions. For example, the latest edition of the American guidelines for the diagnosis and treatment of chronic ischemic heart disease (2012) recommends continuous use of β-blockers for three years in all patients who have experienced acute coronary syndrome. Indications for continued β-blocker use in such patients are determined individually by the physician. A high level of evidence recommends the use of β-blockers in cases of low left ventricular ejection fraction and clinical manifestations of chronic heart failure. If left ventricular function is preserved and there are no other indications for β-blocker use (hypertension, angina, cardiac arrhythmias, etc.), β-blocker therapy may be discontinued. The beneficial clinical effects of beta-blockers in coronary artery disease are achieved through their direct effects on the heart by reducing heart rate, contractility, atrioventricular conduction, and ectopic activity. They can also increase perfusion of ischemic areas by prolonging diastole and increasing vascular resistance in non-ischemic areas. In patients who have had a myocardial infarction, beta-blockers contribute to a 30% reduction in the risk of cardiovascular death and myocardial infarction. Thus, it is presumed that beta-blockers may also have a cardioprotective effect in patients with stable coronary artery disease, but this has not yet been confirmed by placebo-controlled clinical trials. The most commonly used drugs in Europe for the treatment of coronary heart disease are those that selectively block β-receptors (such as metoprolol, bisoprolol, atenolol or nebivolol), as well as the non-selective α1-, β-adrenergic blocker carvedilol.

RESULTS AND DISCUSSION

The largest study available to date has shown that beta-blocker therapy reduces the risk of stroke by 13%, but they are still inferior to other classes of antihypertensive drugs in their ability to prevent cerebrovascular complications. However, with respect to coronary events and heart failure, beta-blockers were no worse than other classes, and in patients with a history of coronary heart disease, they were better than other classes. However, these data apparently do not apply to all drugs in this group. Some studies have compared the effect of atenolol on the risk of cardiovascular complications in hypertension and the effect of other beta-blockers on the risk of adverse outcomes compared with antihypertensive drugs of other classes. It turns out that while atenolol is inferior to other antihypertensive drugs in reducing stroke risk among the white population, other beta-blockers are comparable in efficacy to other therapies. A recent retrospective analysis showed that the use of beta-blockers is not associated with a reduced risk of cardiovascular events in either patients with risk factors for coronary artery disease or in patients with a history of myocardial infarction. It should also be noted that most studies of the effect of beta-blockers in patients after myocardial infarction were conducted before the introduction of other secondary prevention agents, such as statins and ACE inhibitors, into clinical practice. Therefore, uncertainty remains regarding the prophylactic efficacy of beta-blockers in patients with coronary artery disease.

CONCLUSION

The current position regarding the use of beta-blockers has changed. To study the role of beta-blockers in arterial hypertension, data from the LIFE and ASCOT

trials were used. The conclusion was reached that prescribing drugs in this group as monotherapy for uncomplicated arterial hypertension is inappropriate. However, in patients with high and very high cardiovascular risk due to coronary heart disease and heart failure, or insufficiently treated arterial hypertension, the role of beta-blockers remains important. When choosing beta-blockers, preference should be given to more highly selective ones with minimal intrinsic sympathomimetic activity and high receptor binding strength. Such drugs include modern representatives of beta-blockers, which provide a persistent hypotensive effect and are metabolically neutral: nebivolol, carvedilol, bisoprolol, and betaxolol. In the treatment of coronary heart disease, studies by the European Society of Cardiology (ESC) were reviewed, concluding that beta-blockers are more effective, and all of them reduce the risk of cardiac complications in chronic heart failure. Studies show that beta-blockers can be first-line antianginal drugs in patients with coronary heart disease without contraindications. It should also be remembered that modern generations of beta-blockers can be partially excreted by the kidneys, while others are metabolized in the liver, making the latter preferred for treating patients with kidney disease. In chronic stable coronary heart disease, in the absence of the above-mentioned symptoms, beta-blockers are prescribed as first-line drugs for the treatment of angina (myocardial ischemia), in combination with other medications if necessary.

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