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BETTA BLOCKER ASSIGNMENT FOR PATIENTS WITH METABOLIC DISORDERS

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Annotation: A metabolic disorder is a disorder that negatively alters the body's processing and distribution of macronutrients, such as proteins, fats, and carbohydrates. Metabolic disorders can happen when abnormal chemical reactions in the body alter the normal metabolic process. It can also be defined as inherited single gene anomaly, most of which are autosomal recessive.

Key words: Betta blockers, Metabolic disorder, hypertension, sympathetic nervous system, hyperinsulinemia, myocardial infarction, nebivalol.

Increased mortality in patients with metabolic syndrome (MS) and type 2 diabetes is associated with three main risk factors: hypertension, hyperglycemia and hyperlipidemia. An important role in the pathogenesis of hypertension in MS and type 2 diabetes is played by an increase in the activity of the sympathetic nervous system (SNS), which is caused by hyperinsulinemia and hyperleptinemia. Both insulin and leptin, acting at the level of the hypothalamic nuclei, cause activation of a number of sympathetic nerves (renal, adrenal, visceral) and an increase in the concentration of catecholamines in plasma [3]. Patients with MS and type 2 diabetes with hypertension develop left ventricular hypertrophy much earlier, which is an important risk factor for sudden death due to asystole and the development of severe cardiac arrhythmias (LDC). LDC in patients with type 2 diabetes occurs in 37.7%, beta-blockers in this case can serve as a means not only of antiarrhythmic therapy, but also of preventing the development of LDC and asystole.

The main cause of death in almost 50% of patients with type 2 diabetes is myocardial infarction (MI). The results of numerous studies on the use of highly selective beta-blockers as a secondary prevention of recurrent MI and postinfarction angina pectoris have shown a 25% reduction in mortality and a 29% reduction in cases of recurrent MI. Moreover, in patients with type 2 diabetes, there was an almost 3 times greater decrease in mortality compared with patients without diabetes (37% and 13%, respectively) as a result of taking selective beta-blockers [5]. A significant decrease in mortality among patients with diabetes who underwent it was noted in the ISIS-1 study (The First International Study of Infarct Survival) [6], MIAMI (The Metoprolol In Acute Myocardial Infarction) [7] and The Goteborg Metoprolol Trial [8]. In The Bezafibrate Infarction Prevention Study (BIP), beta-blockers therapy was accompanied by a significant decrease in overall mortality and mortality from cardiovascular complications in patients with diabetes, primarily in those who had MI [5]. All patients suffering from type 2 diabetes and who have suffered AMI or unstable angina pectoris are indicated to be prescribed selective beta-blockers as a standard means of secondary prevention of coronary heart disease.

Nebivolol is one of the modern highly selective drugs. Its peculiarity is not only its exceptionally high $\beta 1$ selectivity, but also its vasodilating effect [9-12]. The efficacy and safety of nebivolol therapy was demonstrated in the SENIORS study [9]. As a result of experimental studies, it was found that even the drug prescribed in relatively high doses does not lose its $\beta 1$ selectivity due to the ratio of $\beta 1$ - and $\beta 2$ -blocking activity [13, 14]. NO, penetrating into platelets, prevents their

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aggregation, that is, prevents the formation of thrombosis. NO prevents the development of inflammation in the damaged endothelial wall of the vessel, thus slowing the progression of atherosclerosis. NO reduces the growth of smooth muscle cells, thereby reducing the progression of chronic heart failure. NO also has multiple physiological effects. As a neurotransmitter of the central nervous system, NO is able to improve memory, possibly thereby preventing the progression of Alzheimer's disease. NO has antioxidant properties, neutralizing free radicals. In the respiratory system, it is a vaso- and bronchodilator. In the gastrointestinal tract, it stimulates normal intestinal peristalsis. In the urogenital system, NO is involved in the regulation of bladder functions. In addition, being an important mediator, it participates in the mechanism of erection in men. NO improves blood circulation of skeletal muscles, thereby increasing the flow of nutrients and oxygen to them and removing excess lactic acid. This is especially important for patients with type 2 diabetes and obesity.

Our studies indicate the effectiveness and safety of the use of Nebilet (nebivolol) in patients with MS and hypertension. Nebilet not only significantly reduced blood pressure during the day with a single dose, without disturbing its daily rhythm, but also had a positive effect on carbohydrate and lipid metabolism, which distinguishes it from all known drugs of this group. The ability of nebivolol to stimulate the synthesis of NO by the vascular endothelium led to a significant improvement in the state of brain perfusion detected by scintigraphy. An effective decrease in blood pressure against the background of taking a Non-ticket was accompanied by an improvement in HRV indicators (an increase in the initially reduced SVVR). The lack of improvement in HRV indicators or their deterioration during antihypertensive therapy is a factor that should be taken into account when selecting therapy. HRV dynamics better reflects the dynamics of the patient's well-being than the dynamics of blood pressure. Against the background of Nonbilet therapy, an average daily shortening of the initially elongated value of the QT interval was noted, which is also an important predictor of the effectiveness of antihypertensive therapy. At the same time, we did not detect a negative effect of nebivolol on the level of thyroid hormones, whereas the level of aldosterone in the patients we studied decreased significantly. Similar data on aldosterone were obtained by foreign researchers in patients with hypertension [15].

References:

- 1. Kannel W.B., McGee D.L. Diabetes and cardiovascular risk factors: the Framingham Study // Circulation. 1979. Vol. 59. № 1. P. 8–13.
- 2. Haffner S., Lehto S., Ronnemaa T., Pyorala K., Laakso M. Mortality from coronary heart disease in subject with type 2 diabetes and nondiabetic subjects with and without prior myocardial infarction // N. Engl. J. Med. 1998. Vol. 339. № 4. P. 229–234.
- 3. Pickup J.C., Crook M.A. Is type II diabetes mellitus a disease of innate immune system? // Diabetologia. 1998. Vol. 41. № 10. P. 1241–1248.
- 4. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in Type 2 diabetes: UKPDS 38 // Br Med J. 1998. Vol. 317. № 7160. P. 703–713.

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- 5. Jonas M., Reicher-Reiss H., Boyko V., Shotan A., Mandelzweig L., Goldbourt U., Behar S. Usefulness of beta-blocker therapy in patients with non-insulin-dependent diabetes mellitus and coronary artery disease. Bezafibrate Infarction Prevention (BIP) Study Group // Am. J. Cardiol. 1996. Vol. 77. № 15. P. 1273–1277.
- 6. Randomised trial of intravenous atenolol among 16 027 cases of suspected acute myocardial infarction: ISIS-1. First International Study of Infarct Survival Collaborative Group // Lancet. 1986. Vol. 2. № 8498. P. 57–66.
- 7. The MIAMI Trial Research Group. Metoprolol in acute myocardial infarction (MIAMI). A randomized placebo-controlled international trial // Eur. Heart J. 1985. Vol. 6. № 3. P. 199–226.
- 8. Hjalmarson A., Elmfeldt D., Herlitz J., Holmberg S., Málek I., Nyberg G., Rydén L., Swedberg K., Vedin A., Waagstein F., Waldenström A., Waldenström J., Wedel H., Wilhelmsen L., Wilhelmsson C. Effect on mortality of metoprolol in acute myocardial infarction. A double blind randomized trial // Lancet. 1981. Vol. 2. № 8251. P. 823–827.
- 9. Cleophas T.J. Experimental evidence of selective antagonistic action of Nebivolol on β1-adrenergic receptors // J. Clin. Med. 1998. Vol. 2. P. 1–8.
- 10. Van Nueten J.M. et al. In vitro pharmacological profile of R065824, a potent and selective β1-adrenergic antagonist // JRF Preclinical Research Report R065834/8. November 1985.
- 11. Flather M.D., Shibata M.C., Coats A.J., Van Veldhuisen D.J., Parkhomenko A., Borbola J., Cohen-Solal A., Toman J., Bohm M., Anker S.D., Thompson S.G., Poole-Wilson P.A. Randomized trial to determine the effect of nebivolol on mortality and cardiovascular hospital admission in elderly patient with heart failure (SENIORS) // Eur Heart J. 2005. Vol. 26. № 3. P. 215–225.
- 12. Cockcroft J.R., Chowienczyk P.J., Brett S.E., Chen C.P., Dupont A.G., Van Nueten L., Wooding S.J., Ritter J.M. Nebivolol vasodilates human forearm vasculature: evidence of Larginine/NO-dependent mechanism // J. Pharmacol. Exp. Ther. 1995. Vol. 274. № 3. P. 1067–1071.
- 13. Van de Water A., Janssens W., Van Neuten J., Xhonneux R., De Cree J., Verhaegen H., Reneman R.S., Janssen P.A. Pharmacological and hemodynamic profile of Nebivolol, a chemically novel, potent, and selective beta 1-adrenergic antagonist // J Cardiovasc Pharm. 1988. Vol. 11. № 5. P. 552–563.
- 14. Janssens W.J., Geyskens D., Proost F. Comparison of the β-adrenergic antagonistic effects of Nebivolol, carvedilol, celiprolol and bisoprolol on isolated right atria and trachea of the guineapig. Preclinical Research Report. June 1996.
- 15.Chan T.Y., Woo K.S., Nicholls M.G. The application of nebivolol in essential hypertension: a double-blind, randomized, placebo-controlled study // Int. J. Cardiol. 1992. Vol. 35. № 3. P. 387–395.