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Results of Studying the Effect of Iapf, Omeprazole and Sitotec on Gastric Mucosa Pol Indices

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Annotation: Angiotensin-converting enzyme inhibitors (ACEIs) are baseline drugs for the treatment of arterial hypertension (AH).Many original articles, reviews, and meta-analyses have been devoted to ACEIs and their effect on pathophysiological mechanisms and clinical efficacy, which unequivocally prove the positive role of these drugs in reducing overall mortality, the number of fatal and nonfatal cardiovascular events, and in preventing pathologic remodeling of certain organs and systems.

Keywords: i-APP, omeprazole, sitotec, oxidative stress, nitric oxide, stomach.

Introduction. In the pathogenesis of NSAID gastropathy, the state of lipid peroxidation (POL) in the gastric mucosa is of no small importance. Recently, there has been evidence that ACE inhibitors have a corrective effect on the processes of SEX and NO formation in various organs. This circumstance served as the basis for studying the effectiveness of some ACE inhibitors in indomethacin-induced gastropathy in animals. The ACE group includes substances with a similar chemical structure that have a similar property: to suppress the activity of one of the enzymes of the renin-angiotensin-aldosterone system (RAAS) – angiotensin converting enzyme (dipeptidyl carboxypeptidase, kinase II). Somewhat simplistically, the process of regulating blood pressure with the participation of RAAS looks like this (Figure): angiotensinogen is synthesized in the liver, which is cleaved by renin in blood plasma, turning it into a weakly active decapeptide – angiotensin I; the latter, under the influence of ACE (synthesized in the lungs), turns into angiotensin II; further conversion of angiotensin II to angiotensin III occurs under the influence of angiotensinase, which is accompanied by a decrease in vasoconstrictor activity. Angiotensin II has a wide range of biological effects, in particular, vasoconstrictor (stronger and longer than norepinephrine); stimulates aldosterone synthesis, activating sodium reabsorption in the kidneys and, accordingly, passive reabsorption of water; It stimulates the synthesis of antidiuretic hormone, which increases the synthesis of aquaporin channels in the collecting glomeruli of the kidney, hence the reabsorption of water. Due to peripheral vasoconstriction, an increase in the volume of circulating blood, blood pressure increases. Ace inhibitors, by competitively inhibiting the activity of angiotensin converting enzyme, reduce the activity of RAAS, while simultaneously increasing the level of bradykinin, which is inactivated by the same ACE . In addition, in the heart muscle, along with the constriction of the coronary arteries, angiotensin II causes a positive inotropic effect (directly or as a result of presynaptic release of nor-adrenaline), which may contribute to the development of arrhythmias in ischemic conditions.

The purpose of the study: to study the results of studying the effects of ACE, omeprazole and sitotek on the parameters of sex in the gastric mucosa

Materials and methods of research. Experimental studies were carried out on 78 male rats of a mixed population weighing 160-200 g, which were on a regular vivarium diet. The animals were divided into 13 groups of 6 individuals each.

Results of the study: the results of studying the effect of ACE inhibitors, omeprazole and sitotek on the parameters of SEX in the gastric mucosa in indomethacin gastropathy in animals with ERA are presented. By reducing the formation of angiotensin II not only in blood plasma, but, for example, in the heart, ACE inhibitors prevent the progression of left ventricular dilation (remodeling) and cause the reverse development of myocardial hypertrophy (PREAMI study).

ACE inhibitors can improve endothelial function, reduce platelet aggregation, and suppress many aspects of atherogenesis, so these drugs have anti-ischemic potential. The use of ramipril (efficacy confirmed in the HOPE study) and perindopril (efficacy confirmed in the EUROPA study) is recommended for the treatment of coronary heart disease.

Drugs of this group increase the volumetric velocity of coronary blood flow and reduce the tension of the ventricular walls. They can have an antiarrhythmic effect associated with the effect on trophic processes in the myocardium, an increase in the content of potassium and magnesium ions in the blood, and a decrease in the content of adrenaline.

Since angiotensin II activates the sympathetic centers of the medulla oblongata, enhancing sympathetic effects on the heart and blood vessels, and also stimulates the transmission of impulses in sympathetic nerve endings and autonomic ganglia, stimulates the secretion of adrenaline in the adrenal medulla, the use of ACE inhibitors reduces the influence of the sympathetic nervous system on vascular tone.

ACE inhibitors have no effect on cerebral circulation. Blood flow in the vessels of the brain is maintained at a sufficient level and against the background of a decrease in blood pressure (PROGRESS). Drugs of this group do not affect the metabolism of lipids, uric acid, and prevent the negative effect of diuretics on the electrolyte balance. ACE inhibitors do not cause an increase in blood pressure and an increase in afterload immediately after their withdrawal, i.e. they are not characterized by withdrawal syndrome.

ACE inhibitors have a pronounced nephroprotective effect, since they have a beneficial effect on intrarenal hemodynamics. With prolonged use, these drugs act on two main factors of the progression of renal insufficiency: intraclubular hypertension and tubulointerstitial fibrosis (angiotensin II has adverse intracranial hemodynamic effects, as well as proliferative and profibrogenic effects). In addition, by reducing systemic blood pressure and reducing proteinuria, ACE inhibitors act on two other factors in the progression of kidney damage. ERA indicators of GENDER practically do not change. Indomethacin significantly accelerates the processes of oxidative stress. In animals of this group, the content of POL, MDA and CHL products was 130.4 and 179.0% higher than the control values, respectively. Catalase activity decreased by more than 2.5 times, and SOD by more than 2 times. Ace inhibitors, omeprazole and sitotec have an antioxidant effect on the gastric mucosa. In animals with enalapril, compared with the indicators of the GERA+H2O group, the content of MDA decreased by 38.5%, and CL by 47.7%. Catalase activity increased by 79.0%, SOD by 42.8%. Almost the same antioxidant effect was observed in rats treated with lisinopril. However, a more pronounced effect was observed with captopril treatment. In this group, the content of MDA and CL decreased by 47.7 and 44.4%, respectively, catalase activity increased by 103.0%, SOD by 66.7%. The antioxidant effect was also recorded during treatment with omeprazole. In rats of this group, the content of POL products and the activity of AOS enzymes significantly differed from those in the untreated group. When treated with sitotec, the antioxidant effect was comparable to that in the lisinopril group. When omeprazole is combined with other drugs, their antioxidant effect is potentiated. In the omeprazole group with enalapril, the content of MDA and CL decreased by 49.5% and 53.2%, respectively, compared with the group without treatment. There was also a more pronounced increase in catalase activity by 103.5% and SOD by 60.6%. In the omeprazole group with lisinopril, the content of MDA, CL and catalase activity did not differ significantly from those in the omeprazole group with enalapril. Only the activity of SOD was significantly high. In animals treated with omeprazole with captopril, a decrease in the content of POL products and an increase in the activity of AOS enzymes was significant. The studied indicators significantly differed from the control. The content of MDA and CL decreased by 67.1% and 59.9%, catalase activity increased by 126.0%, SOD by 90.7%. In the omeprazole group with sitotec, the content of MDA and CL decreased by 60.9% and 60.8%, respectively, and the activity of catalase and SOD increased by 136.6% and 87.0%.

Conclusions: Thus, the choice of a drug for the treatment of nonsteroidal anti-inflammatory drugs (NSAIDs)-induced gastropathies in patients with rheumatological diseases presents significant difficulties, primarily due to the need for anti-ulcer treatment against the background of continued NSAID therapy. In many patients admitted to a rheumatology hospital due to an exacerbation of the

underlying disease, when ulcers or erosions of the upper gastrointestinal tract are detected, NSAIDs cannot be canceled even temporarily, since this can lead to a significant deterioration and an increase in joint syndrome.

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