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Influence of a Short-Action Inhibitor on the Course of Chronic Heart Failure

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Abstract . During the chronic heart failure occurs an intensified production of aldosterone, which results in the retention of natrium and water. The inhibitors ACF dilate renal vessels and increase renal blood flow that contributes to the amplification of diuresis and natriuresis. In addition, these drugs decrease aggregate peripheral vascular resistance, arterial pressure and increase cardiac output. This article demonstrates the impact of inhibitors on the improvement of microcirculation and decline of platelets' aggregation in patients with chronic heart failure.

Key words: Captopril, chronic heart failure, microcirculation, aggregation of platelets.

Introduction: Increased activity of the renin-angiotensin-aldosterone system has been found in a large number of clinical and experimental studies in patients with chronic circulatory heart failure [1]. Angiotensin produced in the liver under the influence of renin forms hemodynamically and clinically inactive decapeptide angiotensin I, which after the cleavage of two amino acids, under the influence of angiotensin converting factor, produced mainly in the lungs, is converted into angiotensin II, stimulating the production of aldosterone by adrenal cortex. Angiotensin II is a powerful vasoconstrictor, increasing blood pressure and venous return. It also decreases renal blood flow and causes sodium and water retention in the body, and stimulates antidiuretic hormone production [1, 2, 5]. Based on the above, it is obvious that it is advisable to block the action of A-II in patients with severe chronic circulatory insufficiency, especially when the renin-angiotensin aldosterone system is activated.In addition, ACE is an enzyme, kininase II, which inactivates bradykinin and breaks it down to inactive peptides. Bradykinin is a biologically active substance, a peripheral vasodilator with a predominant effect on the venous knee of the vascular bed [2, 7]. Bradykinin also has the ability to dilate renal vessels, increase renal blood flow, cause its redistribution and increase diuresis and natriuresis [3, 4]. ACE blockade can slow down the destruction of bradykinin in the body, and thus potentiate the positive effect of the latter on the hemodynamics of patients with chronic circulatory insufficiency. Among the agents that reduce the activity of the renin-angiotensin-aldosterone system, the specific ACE blocker - captopril - is of the greatest interest for clinicians [1, 6].

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Purpose of the study: to study the state of peripheral circulation, microcirculation (tissue oxygen regime) and platelet aggregation in patients with chronic heart failure and evaluate the effect of captopril on these parameters.

Material and methods: 58 patients with heart failure (20 females, 38 males), aged 16 to 66 years were examined. The mean age was 46.8±2.8 years. The causes of decompensation were: CHD (35 cases), dilated cardiomyopathy (18 cases), mitral insufficiency of rheumatic etiology (5 cases). Captopril was used in a dose of 25 mg. We performed acute pharmacological test, during which we measured peripheral vascular tone with polarographic study of oxygen tension in tissues on P-7e device. Microcirculation and platelet aggregation were studied using a nonphylometric method on a Chrono-Log aggregometer (USA). Captopril was further administered for 30 days at 25 mg x 4 times daily, after which the study was repeated.

Results and discussion: When used in adequate doses for each patient, captopril decreased the total peripheral vascular resistance by 40% due to arteriolodilatation, A/D - by 20-30%, and the cardiac output increased by 15-30% due to the reduction of afterload. The oxygen tension in peripheral tissues increased significantly. This index remained significantly increased (+34,6±3,0%) even during the course of drug administration. In acute test with captopril there was also decreased platelet aggregation. However, there was a significant increase in platelet aggregation during the course of treatment (Table 1).

Changes in platelet aggregation in patients with heart failureduring captopril treatment

Index	Baseline	Acute test	Course intake
T1, % - absolute value of change in %	72,6±3,7	61,6±3,2x -15,0±2,1	70,8±2,8 -3,1±1,4 +13,2±1,7
T2, % - absolute value of change in %	79,8±1,5	72,5 ±2,3x -9,3 ±1,8	76,9±1,4 -3,9±1,6 +6,0±1,5
DT- absolute value of change in %	10,1±2,1	17.3±1.9x	8,8±1,8
Patients with preserved aggregation reserve	5(27,8%)	13(72.2%) xx	4(22,2%)

Long-term use (up to 30 days) of captopril at a dose up to 100 mg/day was associated with evident improvement in the form of decreased dyspnea, palpitations, decreased wheezing in the lungs, and decreased liver function. I.e. there was not only improvement of central and peripheral hemodynamics, but also of microcirculation, tissue oxygen regime and platelet aggregation activity. Evaluation of the effects The estimation of drug effect on this "distal" (by its location, but very important) link of cardiovascular system in patients with cardiac insufficiency is of great importance for the efficacy control of the performed treatment.

Conclusions .

- ACE inhibitor captopril is a "mild" vasodilator with a balanced effect on both vascular knees that improves tissue oxygenation.

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- 2. In acute use, captopril has an antiaggregative effect due to stimulation of prostacyclin synthesis and disruption of bradykinin inactivation.
- During a course of use of ACE inhibitor, captopril losing its antiaggregative effect, increases its diuretic activity due to improvement of renal hemodynamics, as well as blockade of aldosterone synthesis.

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