UDK: 616.12-008.331.1:616-008.9

EFFECTS OF LOSARTAN ON CARDIOMETABOLIC, HEMOSTATIC, INFLAMMATORY, AND CARDIOVASCULAR PARAMETERS IN PATIENTS WITH METABOLIC SYNDROME

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Abstract: Metabolic syndrome combines hypertension, abdominal obesity, insulin resistance, and vascular risk, creating a cardiometabolic burden. This study evaluated whether losartan could influence hemostatic, inflammatory, metabolic, functional, and psychoemotional abnormalities in patients. Forty-four patients were examined, with 33 randomized to losartan or conventional treatment and followed for six months. Losartan was associated with better blood pressure control, increased exercise tolerance, improved left ventricular function, reduced inflammatory activity, and lower uric acid levels, while metabolic neutrality was maintained. Control patients showed less favorable changes in hemostasis, inflammation, cardiac function, and depressive symptoms.

Keywords: metabolic syndrome; losartan; arterial hypertension; insulin resistance; cardiovascular function

ВЛИЯНИЕ ЛОЗАРТАНА НА КАРДИОМЕТАБОЛИЧЕСКИЕ, ГЕМОСТАТИЧЕСКИЕ, ВОСПАЛИТЕЛЬНЫЕ И СЕРДЕЧНО-СОСУДИСТЫЕ ПОКАЗАТЕЛИ У ПАЦИЕНТОВ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ

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Аннотация: Метаболический синдром объединяет артериальную гипертензию, абдоминальное ожирение, инсулинорезистентность и высокий сосудистый риск, формируя значительную кардиометаболическую нагрузку.

В исследовании оценивалось влияние лозартана на показатели гемостаза, воспаления, метаболизма, функционального и психоэмоционального состояния у пациентов с метаболическим синдромом. Были обследованы 44 пациента, из которых 33 были рандомизированы в группу терапии лозартаном или группу традиционного лечения и наблюдались в течение шести месяцев. Применение лозартана сопровождалось более эффективным контролем артериального давления, повышением толерантности к физической нагрузке, улучшением функции левого желудочка, снижением активности воспалительных процессов и уровня мочевой кислоты при сохранении метаболической нейтральности препарата. В контрольной группе отмечалась менее благоприятная динамика показателей гемостаза, воспаления, сердечной функции и депрессивной симптоматики.

Ключевые слова: метаболический синдром; лозартан; артериальная гипертензия; инсулинорезистентность; сердечно-сосудистая функция.

Introduction: Metabolic syndrome is characterized by interconnected disturbances in blood pressure regulation, adiposity, glucose metabolism, lipid homeostasis, inflammation, and vascular function. Insulin resistance occupies a central position in this pathogenic network and may promote hyperinsulinemia, activation of the renin–angiotensin–aldosterone system, oxidative stress, endothelial dysfunction, thrombogenicity, and progressive cardiovascular remodeling. The coexistence of arterial hypertension and abdominal obesity further increases myocardial workload and may contribute to impaired left ventricular relaxation, hypertrophy, and reduced exercise capacity. Inflammatory mediators, including C-reactive protein, interleukin-6, and tumor necrosis factor-alpha, reflect additional mechanisms linking metabolic abnormalities with cardiovascular injury. The source study emphasized that the effects of angiotensin II receptor blockers on thrombogenic and inflammatory pathways in metabolic syndrome remained insufficiently characterized. Losartan, an angiotensin II receptor blocker, was therefore investigated as a potentially metabolically neutral antihypertensive

capable of influencing several interconnected abnormalities rather than blood pressure alone. Particular attention was directed toward hemostasis, inflammatory markers, insulin resistance, exercise tolerance, ambulatory blood pressure, echocardiographic parameters, and psychoemotional status. A longitudinal comparison with conventional treatment was used to determine whether these effects persisted during six months of observation and whether clinical improvement corresponded with changes in cardiovascular and metabolic indicators. Hypertension severity was considered in relation to hemostatic and inflammatory markers during follow-up.

Aim of the study: To evaluate the effects of losartan on hemostatic, inflammatory, metabolic, cardiovascular functional, and psychoemotional parameters in patients with metabolic syndrome and arterial hypertension.

Materials and methods. The study included 44 patients with metabolic syndrome; biochemical and clinical-instrumental assessments were available for 33 participants, while psychoemotional status was assessed in all 44 patients. Thirty-three patients were randomized into a main group receiving losartan and a control group receiving conventional treatment. The main group comprised 20 patients and the control group 13 patients. Losartan was initiated at 50 mg once daily, with an increase to 100 mg once daily when target blood pressure was not achieved. Assessments were performed at baseline, after one month, and after six months. Venous blood samples were collected using vacuum tubes. Biochemical measurements included glucose, lipids, insulin, uric acid, C-reactive protein, interleukin-6, cortisol, and tumor necrosis factor-alpha. Hemostatic parameters were measured in citrated plasma by clotting methods. Insulin resistance was estimated using the HOMA index. Cardiovascular functional status was assessed by cardiopulmonary exercise testing, ambulatory blood pressure monitoring, and transthoracic echocardiography. Exercise testing used a continuously increasing stepwise protocol. Echocardiography evaluated left ventricular dimensions, volumes, ejection fraction, myocardial mass, diastolic function, and regional

contractility. Psychoemotional status was assessed with HADS and the Zung depression scale. Statistical analysis used variational methods and Student's t-test. Laboratory analyses used automated biochemical, immunoturbidimetric, chemiluminescent, and immunoenzymatic techniques. Ambulatory pressure recordings were obtained at predefined daytime and nighttime intervals. The protocol integrated laboratory, functional, imaging, and questionnaire-based measures to characterize treatment response across multiple cardiometabolic domains during the six-month observation period and follow-up.

Results. At baseline, the treatment groups were generally comparable in age, sex, and obesity severity. After one month, the proportion of patients with elevated C-reactive protein decreased in the losartan group from 50% to 25%, whereas it increased in the control group from 38% to 70%. Baseline fibrinogen was higher in the treatment group, and its concentration remained relatively stable during follow-up, while the control group showed a tendency toward progression. Tumor necrosis factor-alpha remained elevated in both groups but was consistently higher in the losartan group. Glucose and insulin concentrations remained within laboratory reference limits; however, HOMA values indicated persistent insulin resistance in both groups. Losartan produced a significant reduction in uric acid, from 399.0 ± 17.1 to 318.1 ± 48.5 μmol/L after six months, while the control group showed a tendency toward increase. Exercise capacity improved with losartan: anaerobic threshold increased from 95.0 ± 7.8 to 112.5 ± 12.0 W. Maximum oxygen consumption also increased, reaching 18.8 ± 0.5 ml/kg/min after one month. Echocardiographic assessment demonstrated regression of left ventricular hypertrophy, decreasing from 68.8% to 37.5% after six months, together with reduced diastolic dysfunction. In the control group, regional contractility abnormalities increased from 50% to 66.7%. Depression prevalence among the overall metabolic syndrome cohort was 47.7%; patients receiving losartan demonstrated lower depressive symptom scores throughout follow-up. Overall, intervention produced favorable cardiovascular trajectories.

Conclusions: Losartan demonstrated a multidimensional beneficial effect in patients with metabolic syndrome and arterial hypertension. Its use was associated with improved ambulatory blood pressure control, greater exercise tolerance, reduced C-reactive protein prevalence, lower uric acid concentration, and favorable changes in left ventricular structure and function. The drug did not produce unfavorable changes in glucose or lipid metabolism, supporting its metabolically neutral profile. In contrast, conventional treatment was accompanied by less favorable trajectories of inflammatory, hemostatic, and cardiovascular parameters. Psychoemotional findings also favored losartan. These observations support consideration of losartan when hypertension coexists with metabolic syndrome, although larger controlled studies are required.

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