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ABSTRACT

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THE ROLE OF PHYSICAL ACTIVITY IN THE TREATMENT OF PATIENTS WITH ARTERIAL HYPERTENSION AND OBESITY

The study aimed to assess the impact of physical activity on the dynamics of neurohumoral and cardio hemodynamic indicators in patients with arterial hypertension and concomitant obesity under different pharmacotherapy regimens.

Materials and Methods: Two hundred patients with stage II arterial hypertension grade 2 and obesity grade I–II were examined using clinical-anamnestic, anthropometric, biochemical, automated immunological analysis, spectrophotometric and instrumental methods. Per the research objective, patients underwent a re-evaluation after six months of prescribed treatment. Non-pharmacological treatment included dietary therapy aimed at weight correction, with recommendations to increase physical activity primarily through brisk or moderately brisk walking for at least 45 minutes daily. Pharmacological treatment followed the European recommendations in 2018, involving dual antihypertensive therapy. The combination included perindopril and amlodipine. Patients who achieved target BP levels within 3 months of treatment continued to receive the prescribed therapy. For the rest of the patients, a third antihypertensive drug, indapamide, was additionally prescribed. In addition to assessing the achievement of target blood pressure levels after 6 months and at interim stages of the study, patients' adherence to recommendations regarding expanding physical activity was evaluated. Patients were divided into groups with sufficient and reduced physical activity accordingly. Statistical analysis of the obtained data was conducted using the SPSS 17 software package (IBM), Microsoft Office Excel-2003. The data are presented as mean values ± standard deviation. Significance was established at the level of $p < 0.05$ in all cases.

Results of the study: Patients who achieved target blood pressure levels on dual antihypertensive therapy and had sufficient physical activity after six months of treatment differed significantly from patients with low physical activity levels. They exhibited lower values of the pulse wave velocity in the carotid artery and abdominal aorta, a considerably higher degree of endothelium-dependent vasodilation, lower values of end-systolic and end-diastolic diameters, left ventricular mass, a lower E/e´ ratio, and showed better dynamics in metabolic and pro-inflammatory indicators. Additionally, they had a higher level of antioxidant protection. Patients who reached the target blood pressure levels on three-component antihypertensive therapy and engaged in sufficient physical activity after six months displayed significant differences compared to patients with low physical activity. These differences included a more substantial reduction in the pulse wave velocity in the carotid artery, an increase in endothelium-dependent vasodilation, lower indicators of carbohydrate metabolism, reduced levels of pro-inflammatory and pro-oxidant activity, lower levels of leptin, and a more pronounced increase in adiponectin.

Conclusions: Physical activity is a crucial factor influencing the treatment outcomes for patients with arterial hypertension and obesity across different antihypertensive therapy regimens. Irrespective of the chosen antihypertensive therapy option, physical activity positively impacted cardiovascular remodelling, the dynamics of metabolic and pro-inflammatory indicators, and the equilibrium within the oxidative stress-antioxidant protection system and activity of the renin-angiotensin-aldosterone system.

Keywords*:* arterial hypertension, obesity, physical activity, antihypertensive therapy.

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РОЛЬ ФІЗИЧНОЇ АКТИВНОСТІ У ЛІКУВАННІ ХВОРИХ НА АРТЕРІАЛЬНУ ГІПЕРТЕНЗІЮ З ОЖИРІННЯМ

Мета роботи – оцінити вплив фізичної активності на динаміку нейрогуморальних та кардіогемодинамічних показників у хворих на артеріальну гіпертензію із супутнім ожирінням в умовах різних схем фармакотерапії.

Матеріали і методи: Клініко-анамнестичні, антропометричні, біохімічні, автоматизовані методи імунологічного аналізу, спектрофотометричні, інструментальні, методи використали для обстеження 200 хворих на АГ II стадії 2 ступеня з ожирінням I-II ступеня. Відповідно до мети дослідження пацієнти були додатково обстежені через 6 місяців після призначеного лікування. В якості немедикаментозного лікування хворим призначали дієтотерапію спрямовану на корекцію маси тіла та рекомендували збільшення фізичної активності переважно за рахунок ходіння швидким або помірно швидким темпом не менше ніж 45 хвилин за добу. Згідно з Європейськими рекомендаціями 2018 року медикаментозне лікування розпочинали з призначення подвійної антигіпертензивної терапії, що включала комбінацію периндоприлу

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та амлодипіну. Пацієнти, які досягли цільових показників артеріального тиску через 3 місяці лікування, продовжували отримувати призначену терапію. Решті додатково призначався третій антигіпертензивний препарат – індапамід. Крім оцінювання досягнення цільових рівнів артеріального тиску через 6 місяців і на проміжних етапах дослідження оцінювали дотримання пацієнтами рекомендацій щодо розширення фізичної активності, відповідно до цього пацієнтів поділяли на групи з достатньою та зниженою фізичною активністю. Статистичну обробку отриманих даних проводили за допомогою пакета статистичного програмного забезпечення "SPSS 17" (IBM), Microsoft Office Exel-2003. Дані представлені як середні значення ± стандартне відхилення. Значимість встановлена на рівні $p < 0,05$ у всіх випадках.

Результати дослідження. Пацієнти, які досягли цільових показників артеріального тиску на подвійній антигіпертензивній та мали достатню фізичну активність, через 6 місяців лікування відрізнялися від пацієнтів із низьким рівнем фізичної активності достовірно нижчими показниками швидкості пульсової хвилі в сонній артерії та черевній аорті й достовірно вищим ступенем ендотелійзалежної вазодилатації, меншими показниками кінцеводіастолічного та кінцевосистолічного діаметра лівого шлуночка, маси міокарда лівого шлуночка, нижчим співвідношенням E/e′ та мали кращу динаміку метаболічних і прозапальних показників, більший показник антиоксидантного захисту. Пацієнти, які досягали цільових показників артеріального тиску на потрійній антигіпертензивній терапії та мали достатню фізичну активність, через 6 місяців достовірно відрізнялися від пацієнтів із низькою фізичною активністю більшим зниженням швидкості пульсової хвилі в сонній артерії, підвищенням ступеня ендотелійзалежної вазодилатації, нижчими показниками вуглеводного обміну, прозапальної та прооксидантної активності, лептину та більшим підвищенням адипонектину.

Висновки. Фізична активність є важливим фактором покращання динаміки лікування у хворих на артеріальну гіпертензію із супутнім ожирінням в умовах різних схем антигіпертензивної терапії. Незалежно від варіанта антигіпертензивної терапії фізична активність позитивно впливала на показники серцево-судинного ремоделювання, динаміку метаболічних та прозапальних показників, дисбаланс системи оксидативного стресу – антиоксидантного захисту та активність ренін-ангіотензин-альдостеронової системи.

Ключові слова: артеріальна гіпертензія, ожиріння, фізична активність, антигіпертензивна терапія.

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INTRODUCTION / ВСТУП

Obesity is a serious health issue worldwide, primarily because it is closely associated with adverse cardiovascular consequences [1]. A well-established link exists between obesity and high blood pressure (BP), with obesity identified as the cause in 65–78% of cases of primary hypertension [1]. The mechanisms through which obesity induces arterial hypertension (AH) are complex and involve excessive activation of the sympathetic

nervous system, stimulation of the renin-angiotensin-aldosterone system (RAAS), changes in adipose tissue cytokines, insulin resistance, and structural and functional changes in the kidneys [1, 2]. Insulin resistance (IR) is closely related to AH. IR and AH are considered typical representatives of "civilization diseases" emerging in the modern environment due to excessive food consumption and insufficient physical activity [3].

The increase in visceral fat leads to the formation of a pro-inflammatory oxidative environment, contributing to insulin resistance. Normal lipolysis in human adipose tissue is inhibited by insulin, but in individuals with IR, this process is accelerated, leading to increased release of free fatty acids into the bloodstream [4]. IR caused by increased adipose tissue and obesity has systemic consequences for other tissues, such as the kidneys, significantly influencing blood pressure regulation [5]. Insulin resistance at the level of skeletal muscle tissue worsens glucose utilization through its impact on the endothelium, involving vessel dilatation, reduced vessel relaxation, and remodelling, thereby complicating the pathological process.

Reducing body mass index (BMI), consistently increasing physical activity, psychofunctional self-control, behavioural psychotherapy, and healthy eating are currently the main principles in combating obesity and preventing cardiovascular diseases [6]. Despite the extensive research conducted in this direction over the past two decades, it is challenging to identify the physiological and behavioural factors that regulate energy balance [7]. The global cardiology community recommends physical exercise as a cornerstone of non-pharmacological therapy for hypertension. Maria LM Rêgo and colleagues have described the benefits of physical exercises in lowering BP levels and cardiovascular risk factors, improving physical fitness, body composition, quality of life, and reducing mortality risk. The authors found that physical exercises alleviate sympathetic hyperactivity, positively affecting AH and cognitive functions. Despite the broad spectrum of advantages, adherence to physical exercises is challenging among hypertensive individuals [8].

Weight reduction is a primary goal in the treatment of obesity-related hypertension, but few achieve success solely through non-pharmacological treatment. Additionally, there are limited available medications that safely and effectively ensure adequate long-term weight loss [1]. In addition to weight reduction strategies, patients with AH and obesity should receive specific recommendations for treating high BP.

The study aimed to assess the impact of physical activity on the dynamics of neurohumoral and cardio hemodynamic indicators in patients with arterial hypertension and concomitant obesity under different pharmacotherapy regimens.

Materials and methods

Clinical and anamnestic, anthropometric, biochemical, automated methods of immunological analysis, spectrophotometric, instrumental, and statistical methods were used for the examination of two hundred patients with stage II AH grade 2 and obesity grade I–II (BMI 30–34.9 kg/m2 and 35.0–39.9 kg/m2, respectively) and abdominal obesity according to IDF criteria (2005: waist circumference > 94 cm for men and > 80 cm for women), absence of proteinuria (only microalbuminuria is allowed), age 45–55 years. Patients with secondary AH, stage III AH grade 3, obesity grade III, oncology, rheumatic diseases, reduced glomerular filtration rate and proteinemia, acute inflammatory processes, acute coronary syndrome, severe rhythm and conduction disorders were excluded from the study.

Clinical and anamnestic methods with office measurement, home blood pressure monitoring, and anthropometric methods were used to assess clinical manifestations of AH, study etiological factors of the disease, determine the degree of obesity, and diagnose abdominal obesity. The difference between SBP (systolic blood pressure) and DBP (diastolic blood pressure) is evaluated as pulse BP (blood pressure). The formula calculated the average BP:

Average BP = 0.42 × (SBP – DBP) + DBP.

Fasting glycemia, glycosylated hemoglobin (HbA1c), and glucose tolerance tests were evaluated to determine the degree of carbohydrate metabolism disorders. Insulin resistance was determined according to the HOMA model:

HOMA-IR = Blood glucose [mmol/L] × Insulin [μU/mL] / 22.5.

The lipid profile was studied using Olvex diagnosticum kits. Total cholesterol, triglycerides, HDL (high-density lipoprotein), and LDL (low-density lipoprotein) were determined. The functional state of adipose tissue was assessed by leptin and adiponectin indicators (leptin was determined in blood serum using Leptin ELISA kits (DRG Diagnostics, Germany, to determine the

adiponectin levels, the Avi Bion Human Adiponectin (Acrp30) Elisa Kit (Ani Biotech Oy Orgenium Laboratories Busines Unit, Finland)used), the state of pro-inflammatory activity – by the levels of interleukin-6 (IL-6) and C-reactive protein (CRP), the activity of the renin-angiotensin-aldosterone system and the phenotype of AH (low-renin, high-renin) were assessed by the content of aldosterone, by plasma renin activity (PRA) and their ratio (ARR). The intensity of lipid peroxidation was evaluated using spectrophotometry: by the levels of malondialdehyde (MDA) and diene conjugates (DC) – prooxidant activity, by the index of total antioxidant protection – antioxidant capacity.

The morphofunctional properties of the heart and blood vessels were evaluated on the "IMAGIC Agile" ultrasound scanner (manufactured by "Kontron Medical", France). The volumes of left and right atria (LAV and RAV, respectively), end-systolic and end-diastolic diameters (LVESD and LVEDD, respectively) of the left ventricle (LV), diameters of LA and aorta (LAD and AD, respectively) were evaluated. The thickness of the posterior wall of the LV and the thickness of the interventricular septum in the systole (TPWs and TIVSs, respectively) and diastole (TPWd and TIVSd, respectively) were measured. The relative wall thickness of the LV (RWT) was calculated by the formula:

RWT = (TPWd + TIVSd) / LVEDD

The ejection fraction (EF) was calculated by the formula:

EF = (EDV – ESV) / EDV,

where ESV and EDV are the end-systolic and end-diastolic LV volumes, respectively.

Left ventricular mass (LVM) was determined according to the Dereveux method:

$LVM = 1{,}04 \times [(TIVSd + TPWd + LVEDD)^3 – (LVEDD)^3] – 13{,}6.$

The LV myocardial mass index (LVMI) was calculated as the ratio of the LV myocardial mass (LVM) to the surface area of the body (S):

LVMI = LVM / S

Where S was calculated according to the Du Bois formula:

$S = 0{,}007184 \times H^{0.725} \times W^{0.425}$,

Where H – height [cm], W – body weight [kg]

LV diastolic function was evaluated based on the results of pulmonary artery blood flow and transmitral diastolic blood flow in pulsed and tissue Doppler modes with the determination of: maximum early LV filling rate in spectral mode (E), maximum late (atrial) filling speed (A), ratio of maximal rates of early and late filling of LV at spectral mode (E/A), time of isovolumic relaxation of LV (IVRT), time of deceleration early diastolic flow rate (DT), maximum early LV filling rate at tissue mode (e′), mean pulmonary artery pressure (AP) by Kitabatake, ratio of E and e′ (E/e′). For studying endothelial function, the degree of endothelium-dependent vasodilation (EDVD) in reactive hyperemia was determined in all patients according to the method of Celermajer D.S. in the modification of the method by Ivanova O.V. [9, 10]. We measured the intima media thickness (CIMT) of the carotid artery according to the generally accepted method. The pulse wave velocity (PWV) in the carotid artery (cPWV) was determined by the W-Track method; determination of the PWV in the abdominal aorta (aPWV) was performed using a phased sensor.

Patients are recommended a diet aimed at decreasing BP to target values, correcting body weight, and increasing physical activity to at least 45 minutes a day, mainly by walking at a fast or moderately fast pace. Pharmacological treatment was prescribed according to the European guidelines for managing patients with AH 2018 [11]. The primary target indicators were blood pressure values < 140/90 mm Hg; in the case of good treatment tolerability, a further decrease to < 130/80 mm Hg is recommended. According to the purpose of the study, patients were additionally examined 3 and 6 months after the prescribed treatment. BP level control, compliance with recommendations for non-drug and drug therapy, as well as drug tolerance were carried out every 2–4 weeks.

The data obtained was processed using the statistical software package "SPSS 17" (IBM), Microsoft Office Excel 2003. Data are presented as means ± standard deviation. Significance was set at $p < 0.05$ in all cases. The Ethics Committee approved the research protocol. All participants were informed about the purpose of the study and signed a written consent form.

Research results

After three months of dual antihypertensive therapy (AHT), which included a combination of the angiotensin-converting enzyme inhibitor perindopril arginine (5–10 mg/day) and the calcium channel blocker amlodipine (5–10 mg/day), 102 patients (51%) achieved BP targets and continued to receive prescribed therapy. At the same time, 79 had sufficient physical activity, and 29 – were

partially reduced. The second re-examination of patients was carried out six months after the prescribed therapy started, during which, in addition to BP levels, anthropometric, biochemical and cardio-hemodynamic parameters were assessed. The results of the dynamics of indicators in the groups of patients with low and sufficient physical activity showed that six months after treatment, differences in the studied indicators were noted for different degrees of physical activity. In addition to the fact that patients with sufficient physical activity had a significantly lower weight (p=0.000) and a lower BMI (p=0.000), they had better dynamics of indicators of the state of the vascular wall, which was confirmed by a significantly lower cPWV (p=0.001) and aPWV (p=0.032), as well as a considerably higher degree of EDVD (p=0.000) compared to patients with low physical activity. Differences in indicators of the structural and functional state of the myocardium were also established in the studied groups: patients with sufficient physical activity had significantly lower LVEDD (p=0.033), LVESD (p=0.009) and LVM (p=0.015), which characterize the degree of heart remodelling, as well as significantly lower than the ratio E/e′ (p=0.033), which is considered as an integral indicator of diastolic function. After the treatment, patients with sufficient physical activity had a significantly higher level of antiatherogenic HDL (p=0.037), a lower blood glucose level (p=0.015), lower pro-inflammatory activity (the differences in IL-6 and CRP were p<0.001 for both indicators) and more pronounced antioxidant protection (p=0.000) compared to patients with low physical activity. In addition, with sufficient physical activity in hypertensive patients with obesity, the imbalance of adipokines was less pronounced than with low physical activity, which was manifested by significantly higher levels of adiponectin (p=0.000) and lower levels of leptin (p=0.000). It was established that without differences in aldosterone and PRA indicators, patients with sufficient physical activity had a significantly lower ARR (p=0.025) than patients with low physical activity.

Three months after the start of the prescribed therapy, 98 out of 200 patients did not achieve target blood pressure levels with dual AHT. They were additionally prescribed a third antihypertensive drug – the thiazide-like diuretic indapamide (2.5 mg/day). At this stage, patients were recommended a fixed combination of perindopril arginine, amlodipine, and indapamide to improve adherence. Among patients who required an additional prescription of a third antihypertensive drug, groups with low and sufficient physical activity were also selected (49 people in each group). Patients who achieved BP targets on three-component AHT and had adequate physical activity after six months significantly differed from patients with low physical activity by a more significant decrease in cPWV (p=0.008), an increase in the degree of EDVD (p=0.003). Among the indicators of heart remodelling, the only difference was established according to TIVSs (significantly lower than sufficient physical activity, p=0.019) (Table 1).

With sufficient physical activity, significantly more pronounced dynamics of insulin, HOMA-IR and HbA1c levels were noted compared to patients with reduced physical activity. It was confirmed by substantially lower insulin, HOMA-IR and HbA1c values in the group with sufficient physical activity after the treatment (p<0.001 for all indicators). Enough physical activity also affected the dynamics of indicators of oxidative stress – antioxidant protection, which was confirmed by significantly lower levels of DC (p=0.001) and higher levels of the overall antioxidant protection indicator (p=0.000). Positive dynamics of pro-inflammatory response indicators were noted in patients with sufficient physical activity, confirmed by significantly lower levels of IL-6 and CRP (p<0.001 for both indicators) compared to patients with insufficient physical activity. With sufficient physical activity, the leptin level decreased to a greater extent, and the adiponectin level increased (p<0.001 for both indicators) compared to low physical activity, confirming the decrease in the severity of adipokine imbalance (Table 2).

Regardless of the option of antihypertensive therapy, significantly (p=0.000) lower weight and BMI were noted in patients with sufficient physical activity after six months of complex treatment. At the same time, patients with adequate physical activity had better vascular remodelling (CIMT, CIMT bifurcation, aPWV) and heart remodelling (TPWd, EF, RWT, E/e′) values. Physical activity positively affected the levels of lipid indicators and carbohydrate metabolism, the antioxidant defence system, pro-inflammatory activity and the adipokines balance (Table 3).

Table 1 – Comparative assessment of cardiovascular remodelling indicators in groups after treatment using three-component AHT

Indicators	Low physical activity, n = 49	Sufficient physical activity, n = 49	p
CIMT [mm]	0,92 ± 0,08	0,89 ± 0,09	0.103
CIMT bifurcation [mm]	1,32 ± 0,14	1,23 ± 0,13	0.002
cPWV [m/s]	8,25 ± 1,08	7,70 ± 0,93	0.008
aPWV [m/s]	7,95 ± 0,94	7,80 ± 1,13	0.490
EDVD (%)	7,59 ± 1,00	8,25 ± 1,13	0.003
TIVSd [cm]	1,14 ± 0,10	1,12 ± 0,13	0.379
TIVSs [cm]	1,37 ± 0,13	1,31 ± 0,13	0.019
TPWd [cm]	1,14 ± 0,14	1,11 ± 0,16	0.440
TPWs [cm]	1,57 ± 0,33	1,54 ± 0,41	0.710
LVEDD[cm]	5,02 ± 0,29	5,12 ± 0,43	0.177
LVESD[cm]	3,19 ± 0,22	3,22 ± 0,33	0.597
LVEF (%)	65,91 ± 2,34	66,73 ± 2,97	0.134
LVM [g]	261,25 ± 50,10	266,27 ± 81,30	0.714
LVMI [g/m2]	124,55 ± 24,81	128,33 ± 39,52	0.572
RWT	0,46 ± 0,05	0,44 ± 0,05	0.072
Mean pulmonary AP [mm Hg] by Kitabatake	15,84 ± 2,77	16,16 ± 3,59	0.628
E [cm / s]	68,19 ± 8,57	69,36 ± 8,23	0.492
A [cm / s]	74,54 ± 9,51	75,52 ± 9,08	0.602
E/A	0,92 ± 0,14	0,93 ± 0,13	0.947
DT [s]	0,15 ± 0,09	0,16 ± 0,15	0.647
IVRT [s]	0,11 ± 0,05	0,11 ± 0,04	0.492
e′ [cm / s]	11,88 ± 2,16	12,56 ± 2,43	0.143
E/e′	5,89 ± 1,09	5,70 ± 1,22	0.438

Notes: $p < 0,05$: Significance of differences between groups with sufficient and low physical activity.
A – maximum late (atrial) filling speed; AP – artery pressure; DT – time of deceleration early diastolic flow rate; E – filling rate in spectral mode; e' – maximum early LV filling rate at tissue mode; E/A – ratio of maximal rates of early and late filling of LV at spectral mode; E/e' – ratio of E and e'; IVRT – time of isovolumic relaxation of LV; EDVD – endothelium-dependent vasodilatation; EF – ejection fraction; CA – carotid artery; IMT – intima-media thickness; LVM – left ventricular mass; LVMI – left ventricular mass index; PWV – pulse wave velocity (cPWV – carotid artery, aPWV – abdominal aorta); TIVSd – thickness of the interventricular septum (diastole); TIVSs – thickness of the interventricular septum (systole); TPWd – thickness of the posterior wall of the left ventricle in diastole; TPWs – the thickness of the posterior wall of the left ventricle in systole; LVEDD – end-diastolic diameters; LVESD – end-systolic diameters; RWT – relative wall thickness

Discussion. Scientists widely recommend lifestyle modification as a first-line approach for treating high blood pressure. Still, its effect on patients who are potential candidates for the development of resistant AH and already developed resistance to antihypertensive therapy has yet to be thoroughly studied. The evidence presented in a comprehensive analysis of multicenter studies suggests that lifestyle changes, such as increased physical activity, dietary modification, and weight control, can contribute to reducing both clinical and ambulatory blood pressure levels and improving biomarkers associated with cardiovascular risk. The recent discoveries affirm the effectiveness of lifestyle modification when combined with optimized drug therapy in lowering blood pressure and enhancing cardiovascular risk markers. It holds even for patients with resistant hypertension [12–14]. Our study affirmed the positive influence of heightened physical activity on the dynamics of clinical, cardiodynamic, and neurohumoral indicators in individuals with arterial hypertension and concomitant obesity.

Table 2 – Comparative evaluation of biochemical indicators in groups after treatment using three-component AHT

Indicators	**Low physical activity, n = 49**	**Sufficient physical activity, n = 49**	**p**
Total cholesterol [mmol/L]	5,84 ± 0,43	5,76 ± 0,46	0.395
Triglycerides [mmol/L]	2,04 ± 0,45	1,78 ± 0,34	0.002
LDL cholesterol [mmol/L]	4,78 ± 0,51	4,69 ± 0,42	0.348
HDL cholesterol,[mmol/L]	1,04 ± 0,09	1,07 ± 0,11	0.151
Blood glucose [mmol/L]	4,72 ± 0,30	4,69 ± 0,27	0.545
Insulin [μU/mL]	16,65 ± 2,95	12,88 ± 4,08	0.000
HOMA-IR	3,47 ± 0,64	2,69 ± 0,85	0.000
HbA1c (%)	4,87 ± 0,32	4,63 ± 0,27	0.000
Overall antioxidant protection [mmol/L]	1,16 ± 0,05	1,23 ± 0,06	0.000
MDA [nmol/mL]	31,56 ± 3,11	32,13 ± 1,75	0.266
DC [nmol/mL]	30,32 ± 3,10	27,95 ± 3,64	0.001
IL-6 [pg/mL]	111,34 ± 6,91	106,58 ± 6,16	0.001
CRP [mg/L]	4,82 ± 0,79	4,28 ± 0,61	0.000
Aldosteron [ng/dl]	15,35 ± 1,70	15,03 ± 1,55	0.331
PRA , ng/ml/hour	2,52 ± 0,48	2,51 ± 0,44	0.910
ARR	6,39 ± 1,65	6,23 ± 1,49	0.622
Adiponectin [ng/mL]	7,03 ± 0,75	8,34 ± 0,91	0.000
Leptin [ng/mL]	13,96 ± 2,21	11,58 ± 1,40	0.000

Notes: p<0,05: Significance of differences between groups with sufficient and low physical activity.
BMI – body mass index; HbA1c – glycated hemoglobin; HDL – high density lipoprotein; LDL – low-density lipoprotein; CRP – C-reactive protein; IL-6 – interleukin 6; HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; MDA – malonic dialdehyde; DC – diene conjugates; PRA – plasma renin activity; ARR – aldosterone-renin ratio

Table 3 – Comparative assessment of indicators after treatment in groups of patients depending on physical activity and regardless of the AHT variant

Indicators	Low physical activity, n = 78	Sufficient physical activity, n = 122	p
Weight [kg]	98,13 ± 8,58	91,43 ± 9,18	0.000
BMI [kg/m2]	33,95 ± 2,04	31,52 ± 2,41	0.000
Waist circumference [cm]	100,10 ± 6,28	99,49 ± 7,06	0.534
Hip [cm]	106,36 ± 12,54	108,54 ± 6,87	0.114
Waist-to-hip ratio	1,03 ± 0,84	0,92 ± 0,10	0.156
SBP [mm Hg]	126,68 ± 1,68	126,52 ± 1,71	0.508
DBP [mm Hg]	77,72 ± 1,03	77,57 ± 1,03	0.308
Heart rate [bpm]	68,63 ± 3,59	69,03 ± 3,17	0.404
Pulse BP [mm Hg]	48,96 ± 1,94	48,95 ± 1,94	0.970
Average BP [mm Hg]	98,28 ± 0,94	98,12 ± 0,96	0.257
CIMT [mm]	0,90 ± 0,08	0,87 ± 0,08	0.006
CIMT bifurcation [mm]	1,32 ± 0,14	1,24 ± 0,13	0.000
cPWV [m/s]	8,24 ± 1,10	7,58 ± 0,89	0.000
aPWV [m/s]	7,96 ± 0,96	7,63 ± 1,04	0.025
EDVD (%)	7,39 ± 1,02	8,22 ± 1,22	0.000
Total cholesterol [mmol/L]	5,79 ± 0,47	5,70 ± 0,45	0.205
Triglycerides [mmol/L]	1,96 ± 0,41	1,84 ± 0,38	0,053
LDL cholesterol [mmol/L]	4,64 ± 0,58	4,57 ± 0,56	0.398
HDL cholesterol, [mmol/L]	1,04 ± 0,09	1,07 ± 0,11	0.011
Blood glucose [mmol/L]	4,74 ± 0,29	4,65 ± 0,26	0.029
Insulin [μU/mL]	11,82 ± 4,07	12,82 ± 4,55	0.116
HOMA-IR	2,47 ± 0,83	2,64 ± 0,94	0.194
HbA1c (%)	4,88 ± 0,32	4,73 ± 0,37	0.004
Overall antioxidant protection [mmol/L]	1,18 ± 0,07	1,28 ± 0,08	0.000
MDA [nmol/mL]	30,83 ± 3,46	31,05 ± 2,63	0.598
DC [nmol/mL]	27,33 ± 3,22	28,02 ± 3,48	0.076
IL-6 [pg/mL]	110,83 ± 6,52	105,36 ± 6,42	0.000
CRP [mg/L]	4,73 ± 0,80	4,05 ± 0,59	0.000
Aldosteron [ng/dl]	14,77 ± 2,79	14,15 ± 3,01	0.146
PRA , ng/ml/hour	2,30 ± 0,77	2,25 ± 0,71	0.682
ARR	7,54 ± 3,68	7,05 ± 2,66	0.272
Adiponectin [ng/mL]	6,86 ± 0,69	8,13 ± 0,87	0.000
Leptin [ng/mL]	13,63 ± 2,23	10,76 ± 1,68	0.000
TIVSd [cm]	1,14 ± 0,10	1,09 ± 0,12	0.001
TIVSs [cm]	1,36 ± 0,14	1,29 ± 0,14	0.001
TPWd [cm]	1,13 ± 0,12	1,09 ± 0,14	0.033
TPWs [cm]	1,56 ± 0,31	1,50 ± 0,35	0.157
LVEDD[cm]	5,02 ± 0,30	4,97 ± 0,37	0.374
LVESD[cm]	3,18 ± 0,21	3,11 ± 0,29	0.064
EDV [mL]	119,85 ± 17,03	117,75 ± 21,58	0.469
ESV [mL]	40,63 ± 7,06	38,73 ± 9,22	0.122
EF (%)	66,11 ± 2,87	67,28 ± 3,08	0.008

Cont. Table 3

Indicators	Low physical activity, n = 78	Sufficient physical activity, n = 122	p
LVM [g]	260,70 ± 52,83	243,58 ± 70,47	0.067
LVMI [g/m2]	125,23 ± 26,84	120,08 ± 33,27	0.252
RWT	0,45 ± 0,04	0,44 ± 0,04	0.006
LAD [mm]	37,50 ± 3,32	37,20 ± 3,04	0.512
AD [mm]	32,30 ± 1,07	32,42 ± 1,63	0.578
Mean pulmonary AP [mm Hg] by Kitabatake	15,87 ± 2,73	15,40 ± 3,28	0.298
RAV [mL]	37,07 ± 3,91	37,74 ± 5,00	0.063
LAV [mL]	46,23 ± 4,14	46,65 ± 4,73	0.080
e´[cm/s]	11,89 ± 2,04	12,01 ± 2,74	0.073
E [cm/s]	69,27 ± 8,51	69,37 ± 11,86	0.948
A [cm/s]	75,42 ± 9,53	75,39 ± 10,93	0.986
E/A	0,93 ± 0,13	0,94 ± 0,19	0.725
DT [s]	0,15 ± 0,08	0,14 ± 0,10	0.736
IVRT [s]	0,11 ± 0,02	0,11 ± 0,02	0.765
E/e´	5,96 ± 1,03	5,63 ± 1,13	0.042

Notes: $p < 0,05$: Significance of differences between groups with sufficient and low physical activity.
BP – blood pressure; DBP – diastolic blood pressure; SBP – systolic blood pressure; A – maximum late (atrial) filling speed; AP – artery pressure; DT – time of deceleration early diastolic flow rate; E – filling rate in spectral mode; e – maximum early LV filling rate at tissue mode; E/A – ratio of maximal rates of early and late filling of LV at spectral mode; E/e – ratio of E and e; IVRT – time of isovolumic relaxation of LV; EDVD – endothelium-dependent vasodilatation; EF – ejection fraction; CA – carotid artery; IMT – intima-media thickness; LVM – left ventricular mass; LVMI – left ventricular mass index; PWV – pulse wave velocity (cPWV – carotid artery, aPWV – abdominal aorta); RAV – right atrial volume; LAV – left atrial volume; TIVSd – thickness of the interventricular septum (diastole); TIVSs – thickness of the interventricular septum (systole); TPWd – thickness of the posterior wall of the left ventricle in diastole; TPWs – the thickness of the posterior wall of the left ventricle in systole; LVEDD – end–diastolic diameters; LVESD – end-systolic diameters; EDV – end-diastolic volume; ESD – end-systolic volume; RWT – relative wall thickness; LAD – left atrial diameter; AD – aortic diameter; BMI – body mass index; HbA1c – glycated hemoglobin; HDL – high density lipoprotein; LDL – low-density lipoprotein; CRP – C-reactive protein; IL-6 – interleukin 6; HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; MDA – malonic dialdehyde; DC – diene conjugates; PRA – plasma renin activity; ARR – aldosterone-renin ratio

CONCLUSIONS / ВИСНОВКИ

Physical activity plays a crucial role in enhancing treatment outcomes for patients with AH and obesity under various antihypertensive therapy regimens.

Irrespective of the chosen antihypertensive approach, engaging in physical activity has shown positive effects on cardiovascular remodelling, metabolic and pro-inflammatory indicators, as well as the balance within the oxidative stress-antioxidant protection system and the renin-angiotensin-aldosterone system.

For patients with hypertension and obesity, it is strongly recommended to incorporate increased physical activity into their routine. One such activity is brisk or moderately paced walking for a minimum of 45 minutes per day, aiming to improve the overall effectiveness of the treatment.

CONFLICT OF INTEREST / КОНФЛІКТ ІНТЕРЕСІВ

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS / ВКЛАД АВТОРІВ

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