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GENETIC DETERMINANTS OF PSEUDORESISTANT ARTERIAL HYPERTENSION IN PATIENTS WITH CONCOMITANT OBESITY

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The article focuses on studying the genetic determinants affecting the development of pseudoresistant arterial hypertension in patients with accompanying obesity. We examined 200 stage II hypertensive patients with class I–II obesity aged 45–55. Treatment is provided following the 2018 European guidelines. If the target level of blood pressure after three months of dual antihypertensive therapy with perindopril and amlodipine was not reached, the thiazide-like diuretic indapamide was additionally prescribed. Diet therapy aimed at lowering blood pressure to target values and correcting body weight was prescribed as a non-drug treatment. Patients were advised to increase physical activity by fast walking for at least 45 minutes daily. Resistant hypertension was diagnosed in 48 patients, while pseudoresistance was detected in 27 patients. It was established that the determinants of pseudoresistant hypertension in patients with concomitant obesity are the genetic polymorphism of IRS-1 and ADIPOQ, relative wall thickness of the left ventricular, HOMA index, body mass index and mean blood pressure. Concurrently, the main predictors are IRS-1 and ADIPOQ polymorphisms.

Key words: genetic polymorphism, arterial hypertension, obesity, resistant hypertension , antihypertensive therapy, logistic regression.

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ГЕНЕТИЧНІ ДЕТЕРМІНАНТИ ПСЕВДОРЕЗИСТЕНТНОЇ АРТЕРІАЛЬНОЇ ГІПЕРТЕНЗІЇ У ХВОРИХ ІЗ СУПУТНІМ ОЖИРІННЯМ

Стаття присвячена вивченню генетичних детермінант, що впливають на розвиток псевдорезистентної артеріальної гіпертензії у хворих із супутнім ожирінням. Було обстежено 200 хворих на артеріальну гіпертензію II стадії з ожирінням I–II класу віком 45–55 років. Лікування призначали відповідно до Європейських настанов 2018 року. Тим хворим, які не досягли цільового рівня артеріального тиску через 3 місяці на подвійний антигіпертензивній терапії з використанням периндоприлу й амлодипіну додатково призначали тіазидоподібний діуретик індапамід. В якості немедикаментозного лікування призначалась дієтотерапія, спрямована на зниження артеріального тиску до цільових значень і корекцію маси тіла. Пацієнтам було рекомендовано збільшити фізичну активність за рахунок ходьби у швидкому темпі не менше 45 хвилин на день. Резистентну артеріальну гіпертензію діагностували у 48 пацієнтів, водночас псевдорезистентна виявлена у 27 хворих. Встановлено, що детермінантами псевдорезистентної артеріальної гіпертензії у пацієнтів із супутнім ожирінням є генетичний поліморфізм IRS-1 та ADIPOQ, відносна товщина стінки лівого шлуночка, індекс HOMA, індекс маси тіла та середній артеріальний тиск. Водночас генетичні поліморфізми є основними предикторами псевдорезистентності.

Ключові слова: генетичний поліморфізм, артеріальна гіпертензія, ожиріння, резистентність, антигіпертензивна терапія, логістична регресія.

The study is a fragment of the research project "Cellular-molecular and neurohumoral echanisms of target organ remodelling, their interconnections, and correction in patients with combined pathology", the state registration No. 0117U006894.

Arterial hypertension (AH) is one of the most common and most powerful risk factors for the development of cardiovascular complications. Despite a significant number of antihypertensive drugs in the doctors' arsenal, cardiovascular mortality continues to increase. It is expected that arterial hypertension will cause about 13 % of deaths in the world in the coming years [2]. Systemic arterial hypertension has become a serious health problem due to the progressive growth of the aging population combined with the increasing prevalence of risk factors such as obesity, salt intake, physical deterioration and lack of physical activity [7, 13]. In addition, according to a number of studies, resistance to antihypertensive therapy (AHT) is associated with an almost threefold increase in the risk of cardiovascular events [1, 8, 12], and its prevalence varies depending on the selected criteria and characteristics of patients and reaches approximately 13 % in adults [3, 10]. It is difficult to estimate the true prevalence of resistant arterial hypertension (RAH), since pseudo-resistant hypertension (PRAH) is determined in most patients, the main reason of which is the insufficient adherence of patients to AH [15]. All factors that determine low adherence to treatment can be divided into the patient related factors and the doctor related factors [5, 15]. In the 2018 European guidelines for the diagnosis and treatment of hypertension, PRAH is defined as being caused, in addition to low adherence to treatment, by the "white coat phenomenon", increased brachial

artery calcification, especially in the elderly, inaccuracy of blood pressure measurements when using a smaller cuff, clinical inertia of doctors, leading to the prescription of antihypertensive drugs in inadequate doses or irrational combinations, obesity and a number of other reasons [15]. At the same time, if the multifactorial etiology of AH itself has been proven, the mechanisms of its development have been thoroughly studied, and the hereditary predisposition has been confirmed, then the issue of the influence of molecular genetic factors on the development of resistance to AHT requires detailed and further research.

The purpose of the study was to establish the genetic determinants of pseudoresistant arterial hypertension in patients with concomitant obesity.

Materials and Methods. We examined 200 stage II hypertensive patients with class I–II obesity aged 45–55, who provided informed written consent to participate in the study. Patients office blood pressure was measured according to the generally accepted method (in accordance with the 2018 ESC/ESH), and home blood pressure monitoring was performed to exclude white coat hypertension. In the absence of both office control and blood pressure control at home, "uncontrolled persistent hypertension" was diagnosed. Mean BP was calculated by the formula: Mean BP=0.42×(SBP–DBP)+DBP. Anthropometric examination was used to assess the degree of obesity and diagnose abdominal obesity height, body weight, body mass index (BMI=kg/m^2 where kg is a person's weight in kilograms and m^2 is their height in metres squared), waist circumference, thigh volume, index "waist-thigh" were determined. The degree of carbohydrate metabolism disorders was assessed by determining fasting glucose, glycosylated hemoglobin (HbA1c) and performing an oral glucose tolerance test. Serum insulin concentration was determined using Insulin ELISA kits ("DRG Diagnostics", Germany).The HOMA index was calculated by the formula: HOMA-IR=blood glucose (mmol/L)×blood insulin (μU/mL)/22.5. HOMA-IR values of 2.77 or more were regarded as the insulin resistance (IR) presence. The state of proinflammatory activity was assessed by the levels of interleukin-6 (IL-6) and C-reactive protein (CRP). Serum levels of CRP and IL-6 were determined by enzyme-linked immunosorbent assay. The activity of RAAS was assessed by the content of aldosterone and plasma renin activity. The functional state of adipose tissue was assessed by leptin and adiponectin parameters. Leptin was determined in blood serum using Leptin ELISA kits (DRG Diagnostics, Germany). The Avi Bion Human Adiponectin (Acrp30) Elisa Kit test system (Ani Biotech Oy Orgenium Laboratories Business Unit, Finland) was used to determine adiponectin levels. The intensity of lipid peroxidation was evaluated by indexs of prooxidant activity – levels of malonic dialdehyde (MDA) and diene conjugates, antioxidant capacity by the index of general antioxidant protection.

Genomic DNA was extracted from peripheral blood leukocytes using "DNA express blood" kit. Identification of the G276T polymorphism of the ADIPOQ gene and the G972R polymorphism of the IRS-1 gene was performed by the polymerase chain reaction followed by restriction analysis using sequences of specific primers (direct – 5'GGCCTCTTTCATCACAGACC-3' and reverse – 5'AGATGCAGCAAAGCCAAAGT-3' for identification of G972T polymorphism; for identification of G972T polymorphism direct – 5'AGTCTGGCTACTTGTCTGGC-3, reverse – 5'ATGAGTTGTCCCCGTCAGA-3'). The BsmI enzyme was used to cleave the polymerase chain reaction products at genotyping the G276T polymorphism of the ADIPOQ gene, and the amplification products were incubated with AluI restriction enzyme when genotyping the G972R gene of the IRS-1 gene. The hydrolysis products were isolated in 3 % agarose gel and visualized. Three genotypes of the IRS-1 gene (G/G, G/R and R/R) and three genotypes of the ADIPOQ gene (G/G, G/T, T/T) were identified.

Morphofunctional properties of the heart and blood vessels were evaluated on an ultrasound scanner "IMAGIC Agile" (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) 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, where TPWd – thickness of the posterior wall of the left ventricle in diastole, TIVSd – thickness of the interventricular septum (diastole), LVEDD – end-diastolic diameters.

Statistical methods were used for mathematical data processing: variation statistics, factor analysis, correlation analysis, ROC-analysis, logistic regression method. The statistical processing of the obtained data was carried out using the package of statistical software "SPSS 17" (IBM), Microsoft Office Exel-2003. The data are presented as mean values±standard deviation. Significance was set at a p value of <0.05 in all cases. The study protocol was approved by the Ethics Committee. All participants were informed about the aim of the study and signed a written consent form.

Results of the study and their discussion. According to 2018 ESC/ESH Clinical Practice Guidelines for the Management of Arterial Hypertension, treatment started with the prescription of dual

antihypertensive therapy (AHT). Patients received angiotensin-converting enzyme inhibitor (ACEI) perindopril and calcium antagonist amlodipine. The primary target of blood pressure levels was <140/90 mm Hg. with a subsequent decrease in blood pressure <130/80 mm Hg. with good tolerability of treatment.

A diet therapy aimed at blood pressure lowering to target levels and weight correction was prescribed as a non-drug treatment. Patients were advised to increase physical activity, mainly by walking at a brisk pace for at least 45 minutes per day. Physical activity considered sufficient in case of the increase of the mean intensity of aerobic exercise not least than 300 minutes per week, and patients who did not adhere to it – patients with reduced physical activity

Initially, three months after the start of treatment, the number of patients who reached or did not reach the target BP levels was assessed. Those patients who reached the target blood pressure levels with dual AHT, there were 102 of them, continued prescribed therapy. Among them, 73 people had sufficient physical activity, and 29 – partially reduced physical activity. Thiazide-like diuretic indapamide was additionally prescribed to 98 patients who had not reached the BP target after 3 months. If the target blood pressure levels were not reached one month after indapamide administration (with sufficient daily doses of antihypertensive drugs), such patients considered as patients with resistant hypertension (48 people), and they were additionally prescribed the fourth antihypertensive drug spironolactone. Among the 48 patients who did not achieve the target blood pressure levels, 27 patients had pseudoresistant hypertension (caused by insufficient compliance with the doctor's instructions to ensure a sufficient level of physical activity), and 21 patients had true resistance (failure to achieve the target blood pressure levels with prescribed triple antihypertensive therapy and sufficient physical activity).

The second re-examination of patients was performed six months after the prescribed therapy, during which, in addition to blood pressure levels, anthropometric, biochemical and cardiohemodynamic parameters were assessed.

According to ESC/ESH 2018 criteria, 27 patients were diagnosed pseudo-resistance to AHT. The use of logistic regression in such patients at the stage of the initial examination showed that the model of pseudo-resistance included indices: genetic polymorphism IRS-1 (regression coefficient (CR) 4.13), genetic polymorphism ADIPOQ (CR 2.34), HOMA index (CR 1.07), MDA (CR 0.41) and mean BP (CR 0.38) ($p<0.05$ for all indexs). The influence of these indices on the formation of pseudo-resistance was confirmed by the odds ratio (OR) and 95 % confidence intervals (CI) and the area under the ROC curve (0.982). For IRS-1: OR – 62.43, 95 % CI – 7.79–500.19; for ADIPOQ: OR – 10.37, 95 % CI – 1.47–73.20; for HOMA index: OR – 2.91, 95 % CI – 1.08–7.83; for MDA: OR – 1.51, 95 % CI – 1.01–2.27; for mean BP: OR – 0.68, 95 % CI – 0.48–0.99.

Given the above data, the model of pseudoresistance hypertension to AHT in patients with concomitant obesity in the pre-treatment phase is as follows:

$$y=\exp(b_0+1.07x_1+0.38x_2+4.13x_3+2.34x_4+0.41x_5)/[1+\exp(b_0+1.07x_1+0.38x_2+4.13x_3+2.34x_4+0.41x_5)],$$

where b_0=-15.43 – constant; x_1 – HOMA index; x_2 – mean BP; x_3 – genetic polymorphism IRS-1; x_4 – genetic polymorphism ADIPOQ; x_5 – MDA.

After treatment, the model of pseudoresistance included some of the indices that impact the pre-treatment phase (genetic polymorphism IRS-1 (CR 5.52); genetic polymorphism ADIPOQ (CR 4.52); HOMA index (CR 4.31); mean BP (CR 2.09)), as well as new indices: RWT (CR 26.34); BMI (CR 2.19), ($p<0.05$ for all indexs)). At the same time, the greatest influence on the formation of this model was exerted by indices such as LV RWT, genetic polymorphisms of IRS-1 and ADIPOQ, the HOMA index, which was confirmed by the largest values of regression coefficients. The influence of these indices on the formation of pseudoresistance was confirmed by OR and 95 % CI and the area under the ROC curve (0.994). All these parameters confirmed their influence on the formation of pseudo-resistance in hypertensive patients with obesity. For genetic polymorphism IRS-1: OR – 250.44, 95 % CI – 4.33–14495.68; for genetic polymorphism ADIPOQ: OR – 91.99, 95 % CI – 2.25–3757.83; for HOMA index: OR – 74.17, 95 % CI – 3,30–1669.18; for mean BP: OR – 8.07, 95 % CI – 1.05 –61,95; for RWT: OR – 274E+009, CI – 3,25–23.1E+021;for BMI: OR – 8.97, CI – 1.38–58.27. Thus, the model of pseudo-resistant arterial hypertension in obese patients looks as follows:

$$y=\exp(b_0+4.31x_1+2.091x_2+26.34x_3+5.52x_4+4.52x_5+2.19x_6)/[1+\exp(b_0+4.31x_1+2.091x_2+26.34x_3+5.52x_4+4.52x_5+2.19x_6)],$$

where b_0=-281.74 – constant; x_1– HOMA; x_2 – mean BP; x_3 – RWT; x_4 – genetic polymorphism IRS-1; x_5 – genetic polymorphism ADIPOQ; x_6 – BMI.

It is widely known and scientifically proven that patients with resistant hypertension have a significantly increased risk of target organ damage, adverse clinical events, and all-cause mortality [14]. One of the conditions that contributes to the increased prevalence of true resistant hypertension is pseudoresistant hypertension. Approximately 50% of patients diagnosed with resistant hypertension

actually have pseudoresistance [6, 9]. PRAH is often caused by the patient's low adherence to therapy. Therefore, there has been growing interest in the bioanalytical assessment of adherence to antihypertensive medications, the study of potential determinants of non-compliance in the treatment regimen of resistant hypertension, and the evaluation of objective screening of antihypertensive drugs in serum [9]. To rule out pseudo-resistance to therapy, in addition to clarifying the etiology of hypertension (to exclude secondary causes), several approaches are employed. These include 24-hour blood pressure monitoring, home blood pressure measurements, adjustments to medication therapy, and lifestyle modifications [11, 13, 15]. Lifestyle modification is widely recommended as a first-line approach for managing high blood pressure, but its impact on patients with resistant hypertension has not been extensively studied. Fernando Ribeiro and colleagues conducted a comprehensive analysis of multicenter studies and presented data suggesting that lifestyle changes, including increased physical activity, dietary modification, and weight control, may contribute to reducing clinical and ambulatory blood pressure and improving cardiovascular risk biomarkers. These new findings confirm the efficacy of lifestyle modifications in combination with optimized drug therapy for lowering blood pressure and improving cardiovascular risk markers in patients with resistant hypertension [11, 14].

Arterial hypertension is a multifactorial condition and is often associated with obesity and excess body weight, which also increases the risk of developing resistance to antihypertensive therapy [15]. In our study, using logistic regression with ROC analysis, we identified the main determinants of PRAH formation. It was found that genetic polymorphisms in IRS-1 and ADIPOQ, the HOMA index, mean blood pressure, and the marker of oxidative activity, malonic dialdehyde, should already draw the doctor's attention during the initial examination regarding the possible development of PRAH in patients with obesity. In other words, these determinants should be considered early diagnostic markers for the formation of pseudoresistance in patients with hypertension and concurrent obesity. It's worth noting that when studying the processes of genetic information realization, it's important to take into account the modifying influence of the environment, which often plays a crucial role in genotype realization [4].

Conclusions

1. Genetic markers play a leading role in the formation of pseudoresistant arterial hypertension in patients with accompanying obesity.

2. It has been established that the main determinants of pseudoresistant hypertension in patients with concomitant hypertension are the genetic polymorphism of IRS-1 and ADIPOQ, RWT LV, HOMA index, BMI, and mean blood pressure.

3. For the purpose of early diagnosis of pseudoresistant hypertension in patients with concomitant hypertension, it is necessary to determine the genetic polymorphism of IRS-1 and ADIPOQ, HOMA index, mean BP and MDA.

4. The application of the model of pseudoresistant hypertension at the stage of primary examination of patients with hypertension and concomitant hypertension provides an opportunity to develop a comprehensive treatment program for such patients in advance.

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USING LASER PHOTODYNAMIC THERAPY IN THE TREATMENT OF AUTOIMMUNE THYROIDITIS

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The purpose of the study was to comparatively assess the effectiveness of treating patients with a diffuse form of autoimmune thyroiditis using laser photodynamic therapy and traditional treatment. The work was based on examination and treatment data of 90 patients in whom the concentrations of a number of hormones were examined: TSH, FT3, FT4, and Anti-TPO. In each group, the relative values of the analyzed indices, their mean error (m), and the reliability of intergroup differences (according to the χ^2 criterion) were calculated. Conservative therapy in patients of the control group brought a positive result only in 32 (64 %) patients, while in 18 patients out of 50 (36 %), signs of subclinical hypothyroidism, as well as structural changes in the thyroid gland, persisted in later dates (≥21 days). In the main group, hormone levels clearly improved, approaching normal values, indicating the absence of hypothyroidism signs in this group.

Key words: autoimmune thyroiditis, photodynamic therapy, photoditazine.

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ЗАСТОСУВАННЯ ЛАЗЕРНОЇ ФОТОДИНАМІЧНОЇ ТЕРАПІЇ ПРИ ЛІКУВАННІ АУТОІМУННОГО ТИРЕОЇДИТУ

Метою дослідження було проведення порівняльної оцінки ефективності лікування пацієнтів з дифузною формою аутоімунного тиреоїдиту із застосуванням лазерної фотодинамічної терапії та традиційним методом лікування. Робота ґрунтувалася на даних обстеження та лікування 90 пацієнтів, у яких було досліджено концентрацію низки гормонів: ТТГ, св. Т3, св. Т4 та АТ-ТПО. У кожній групі хворих розраховувалися відносні значення аналізованих показників, їхня середня помилка (m), 95 % довірчий інтервал (±2m) та достовірність міжгрупових відмінностей (за критерієм χ^2). Консервативна терапія у хворих контрольної групи принесла позитивний результат тільки у 32 (64 %) пацієнтів, у той час як приблизно у третини 18 (36 %) з 50 пацієнтів ознаки субклінічного гіпотиреозу, також, як і структурні зміни в щитовидній залозі, зберігалися і у пізніші терміни (21 днів і більше). У хворих основної групи показники гормонів явно покращали і наблизилися до показників нормальних цифр, що свідчить про відсутність у хворих цієї групи ознак гіпотиреозу.

Ключові слова: аутоімунний тиреоїдит, фотодинамічна терапія, фотодитазин.

The problem's urgency is due to the continuous growth of morbidity and insufficient effectiveness of existing treatment methods. The frequency of autoimmune thyroiditis reaches 25–35 % among all thyroid diseases and, after diabetes mellitus, ranks second among endocrinological diseases. Autoimmune thyroiditis (AIT) mainly affects the population aged 25 to 65 years [12, 14]. Long-term courses of conservative therapy have a temporary positive effect, but following the cessation of therapy, 40–60 % of patients have a relapse of autoimmune thyroiditis since the drugs help alleviate the symptoms but do not solve the real problem – the elimination of autoimmune inflammation [8, 10].