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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

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**ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ
ТБИЛИСИ - НЬЮ-ЙОРК**

GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board and The International Academy of Sciences, Education, Industry and Arts (U.S.A.) since 1994. **GMN** carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

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995 (32) 253-70-58

Fax: 995 (32) 253-70-58

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NINITEX INTERNATIONAL, INC.

3 PINE DRIVE SOUTH

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3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

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3. სტატიაში საჭიროა გამუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანორმილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოსალები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტ-სურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედეგის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ურნალის დასახელება, გამოცემის ადგილი, წელი, ურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფრჩილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცეზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტშე მუშაობა და შეჯრება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდიდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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CORRELATION BETWEEN THE LEVELS OF ADIPOSE-DERIVED HORMONE AND CARDIOMETABOLIC MARKERS IN PATIENTS WITH HYPERTENSION AND OBESITY

Goncharuk O., Matyukha L.

P. Shupyk National Healthcare University of Ukraine, Kyiv, Ukraine

The WHO declared obesity to be a global pandemic in 2014. The worldwide number of overweight individuals makes up 2.1 billion, which is about 30% of the total population. This tendency keeps on rising. More than 3 million people die yearly from obesity. Since 1975, the incidence of obesity has tripled in all parts of the world [1]. Countries with advanced economies, such as the United States and the United Kingdom, are considered to have the highest obesity levels, but still they are ranked 12th and 36th by obesity rates, with 36.2% and 27.8%, respectively. Actually, obesity is the most typical for island countries – Samoa, Tokelau – nearly 74%, Nauru – 61% and the Cook Islands – 55.9%. Countries that almost don't face the problem of obesity are India (3.9%), Bangladesh (3.6%) and Vietnam (2.1%). Ukraine is ranked 76th among 210 countries by obesity rates – about 24.1% of the population have this condition and almost 38% are overweight [2,3]. The discovery concerning endocrine functions of adipose tissue has led to the interest in studying its role in the development of cardiovascular and metabolic diseases. It is known currently that adipose tissue not only responds to afferent signals originating from the autonomic and central nervous system, but also secretes itself factors having crucial endocrine functions. Examples may include leptin, adiponectin, ghrelin, resistin and other cytokines - tumor necrosis factor alpha (TNF α), interleukin-6 (IL-6), complement cascade components, plasminogen activator inhibitor-1, proteins of the renin-angiotensin system (I and II), cytochrome P450 and others. Adipose tissue plays a significant part in the metabolism of steroid, sex hormones and stress-responsive glucocorticoids (cortisol and cortisone), secretes enzymes essential for activation, interconversion and inactivation [4-9]. It is also a known fact that adipocytes have numerous receptors, responsible for their sensitivity to humoral factors, enabling adipose tissue to interact with endocrine, cardiovascular, immune and nervous systems. Before the discovery of leptin, the research involving mice, holding recessive mutations linked to obesity (ob) and diabetes (db), predicted the existence of a circulating endocrine hormone capable of transmitting information relating to energy sufficiency of fat depots from periphery to the CNS. These predictions were later verified by the discovery of the leptin-encoding gene (Lepob) in 1994 [10-12].

Leptin (from the Greek *leptos* – thin) is a 16-kDa polypeptide containing 167 amino acids with a structural configuration similar to cytokines. Leptin has anorexigenic effects, metabolic costs and controls carbohydrate and fat metabolism [13-14]. A correlation between leptin levels and values in systolic dysfunction and heart failure has also been proved [15-16]. The matter of a correlation between the hormone leptin and the values of morphometry, anthropometry, lipid, purine, calcium profiles and other metabolic indicators in patients with hypertension and obesity remains under-explored.

The aim of our study was to analyze the relationship between leptin levels and morphometric, anthropometric, biochemical parameters in patients with hypertension and obesity and in group with healthy individuals.

Material and methods. The research was conducted within the period between 2017 and 2019 at the clinical bases belong-

ing to the Department of Family Medicine and Outpatient Care Shupyk National Healthcare University of Ukraine. The main group was comprised of 64 individuals with hypertension and obesity/excess weight aged 25-63. Patients diagnosed with stage I-II hypertension, BP over 140/80 mmHg or lower, in case of chronic usage of antihypertensive drug, BMI over 24.9, waist circumference of 80 cm and higher in women and 94 cm and higher in men. The control group was comprised of 21 relatively healthy individuals (7 men and 24 women) with the BMI of 18-24.9 kg/m². All patients observed were comparable by age with predominance of women. Essential hypertension was verified in accordance with the guidelines by the Ukrainian Association of Cardiology [17]. Obesity was diagnosed according to the WHO classification [18]. All patients underwent an in-depth clinical, anthropometric, morphometric and lab examination, including blood pressure, height, weight and waist circumference, as well as the detection of fasting glucose, total cholesterol, HDL, LDL and VLDL levels, atherogenic index, uric acid, serum hormone leptin and ionized calcium (Ca²⁺) levels and body fat percentage measured by a bioimpedancemetry (BIM) method. A BIM test was performed using a body composition analyzer – OMRON Body Composition Monitor BF212 (Japan), 2012. Measurements were taken while standing, barefoot, pre-treating a surface with ethanol 96%, preliminarily bringing height, sex and age parameters into the program. The accuracy of measuring the body fat percentage was up to 0.1% [19]. The firmware measured the percentage of adipose tissue and weight. To characterize a body weight a body mass index (BMI) was used, calculated by the formula: BMI (kg/m²) = body weight (kg)/height (m)². A body weight was considered normal in case of a BMI < 25.0 kg/m², excess – from 25.0 to 29.9 kg/m², I degree obesity – from 30.0 to 34.9 kg/m², II degree obesity – from 35.0 to 39.9 kg/m², III degree obesity > 40.0 kg/m². The type of adipose tissue distribution was determined according to a waist circumference (WC). A WC measurement > 94 cm in men and > 80 cm in women was considered the distinctive feature of abdominal obesity [20]. A waist circumference was measured using a standardized medical tape measure supposed to identify the risk for metabolic syndrome by the WHO, and height – by means of a floor-mounted stadiometer RP-2000. A biochemical analysis was conducted using Olympus Aubso automated analyzer (France). The criteria for non-inclusion of patients in the study were patients with stage III hypertension, stable and unstable angina, previous myocardial infarction, pre-existing cerebrovascular accident, ejection fraction lower than 50%, systemic infections, pregnancy, insulin-dependent diabetes, renal failure, decompensated cirrhosis (elevation of transaminases to more than 3 times, total bilirubin – more than 2 times), thyroid gland hyper- and hypofunction, cancer. The study was carried out in accordance with the national norm of bioethics and statements of Helsinki declaration (1975, version of 2013) for medical research involving human subjects, including research on identifiable human material and data. The study was approved by the Bioethics Committee for Experimental and Clinical Research Shupyk National Healthcare University of Ukraine, Kyiv, Ukraine. All patient signed an informed consent to participate in the study.

Statistical data analysis was performed using SPSS Statistics ver. 23.0.0 and StatPlus ver.7.3.3.0. The results base and data preparation for mathematical processing were performed in MS Excel 2007. A database check, when it comes to patient distribution, was performed using the Kolmogorov-Smirnov test and the Shapiro-Wilk W-test. The probability of a difference between comparison groups was identified using the Mann-Whitney U test for non-parametric statistics and the Student t-test for parametric ones. A correlation between parameters was estimated using the Pearson's correlation coefficient. A correlation between parameters was analyzed using a multivariate multiple regression model. Multiple regression analysis was used to analyze a correlation between the dependent variable of plasma leptin and body fat mass (BFM), BMI, age, SBP, DBP, cholesterol, TG, HDL, LDL, VLDL, atherogenic index, uric acid, ionized calcium, glucose, glycated Hb, as independent variables. The acknowledged probability of error $p < 0.05$ with the confidence interval (CI) of 95%, $p < 0.01$ with a CI accounting for 99% were considered significant for all calculations.

Results and discussion. In accordance with the findings the majority of those examined in both groups were made up by women – 65.6% (aged 40.25 ± 17.53) in the core group and 63.6% among controls (aged 37.5 ± 7.14). The average weight in the core group with obesity and hypertension was 34.1% higher than in the group consisting of healthy individuals (88.62 ± 21.01 kg – core group, 58.35 ± 16.02 kg - control group). Anthropometric and lab findings are represented in the Table 1.

A negative correlation between ionized calcium levels and age ($r=-0.26$ $p=0.007$), uric acid ($r=-0.28$ $p=0.004$), positive correlation with HDL ($r=0.29$ $p=0.003$) were detected in a correlation

analysis in the core group, a negative correlation between ionized calcium and body fat mass turned out to be weak ($r=-0.21$ $p=0.03$). A weak positive correlation between uric acid levels and age ($r=0.25$ $p=0.009$), triglycerides ($r=0.28$ $p=0.004$), VLDL ($r=0.28$ $p=0.004$) and atherogenic index ($r=0.35$ $p=0.0002$) was obtained; negative – between uric acid levels and HDL ($r=-0.49$ $p=0.002$). A strong positive correlation between glucose levels and SBP ($r=0.34$ $p=0.0004$), weak positive correlation between glucose and leptin ($r=0.21$ $p=0.03$), glycated hemoglobin and SBP ($r=0.29$ $p=0.003$), between glucose levels and age ($r=0.23$ $p=0.01$) and weak negative between glucose measurements and HDL ($r=0.23$ $p=0.02$), glycated Hb and VLDL ($r=-0.3$ $p=0.001$) were obtained when it came to metabolism. A weak negative correlation was found between glycated hemoglobin and leptin ($r=-0.21$ $p=0.03$), however, a strong one – between glycated hemoglobin and glucose ($r=0.85$ $p=0.000001$). A strong positive correlation between a BMI value and a waist circumference ($r=0.92$ $p=0.00001$), body weight ($r=0.88$ $p=0.000001$) and BFM ($r=0.65$ $p=0.000001$) was also detected. A strong positive correlation was also reported between BFM and a body weight, BFM and a waist circumference ($r=0.47$, $r=0.57$ $p<0.05$). A strong positive correlation was detected between a waist circumference and a patient's weight ($r=0.7$ $p=0.00001$). A weak positive correlation was recognized between SBP and a waist circumference ($r=0.24$ $p=0.01$), no correlation was found between SBP and age ($r=0.02$ $p=0.03$). A strong positive correlation was found between the levels of DBP and SBP ($r=0.8$ $p=0.00001$), moderate positive – between DBP and a waist circumference ($r=0.45$ $p=0.001$), weak positive – between DBP and weight ($r=0.36$ $p=0.0001$) and BFM ($r=0.26$ $p=0.008$). A strong positive correlation between LDL and cholesterol ($r=0.7$ $p=0.00001$), VLDL

Table 1. Clinical, morphometric, anthropometric and lab values in the group of healthy individuals and patients with hypertension and obesity/excess weight

Parameters	Patients with hypertension and obesity/excess weight (n=64)	Group of healthy individuals (n=21)
Men, n (%)	22 (34.38)	7 (34.33)
Women, n (%)	42 (65.63)	14 (66.67)
SBP, mmHg	$151.88 \pm 13.52^*$	120.47 ± 0.47
DBP, mmHg	$101.47 \pm 5.72^*$	80.47 ± 0.47
Weight, kg	$88.62 \pm 21.01^*$	58.35 ± 16.02
BMI	$32.13 \pm 6.97^*$	21.14 ± 2.83
BFM, %	$40.57 \pm 8.52^*$	29.3 ± 1.73
Waist circumference, cm	$109.33 \pm 18.82^*$	69.75 ± 6.84
Glucose, mmol/L	4.6 ± 0.7	4.0 ± 0.11
Total cholesterol, mmol/L	$5.6 \pm 1.01^{**}$	4.67 ± 0.84
Triglycerides, mmol/L	$1.41 \pm 0.8^*$	0.78 ± 0.5
HDL, mmol/L	$1.45 \pm 0.3^{**}$	1.73 ± 0.1
LDL, mmol/L	$3.5 \pm 0.95^*$	3.6 ± 0.32
VLDL, mmol/L	$0.64 \pm 0.36^*$	0.34 ± 0.03
Atherogenic index	$3.97 \pm 0.89^*$	2.49 ± 0.2
Uric acid, μ mol/L	$288.23 \pm 74.04^{***}$	164.42 ± 84.48
Ionized calcium, mmol/L	$1.13 \pm 0.5^*$	1.844 ± 0.94
Leptin, ng/ml	$14.47 \pm 10.1^{***}$	4.2 ± 1.76

notes: * - The difference between groups is significant at the significance level $p < 0.05$; ** - $p < 0.01$, *** - $p < 0.001$

and triglycerides ($r=0.9$ $p=0.000001$) was detected when it came to lipid metabolism. A weak negative correlation – between cholesterol and body weight ($r=-0.36$ $p=0.0002$), weak positive – between cholesterol and age ($r=0.25$ $p=0.009$), cholesterol and BMI ($r=0.27$ $p=0.006$); as well as a moderate correlation – between HDL and triglycerides ($r=-0.4$ $p=0.00004$).

A strong positive correlation in the group with hypertension and obesity between plasma leptin levels and total cholesterol ($r=0.40$, $p=0.00004$), strong negative correlation between leptin and HDL ($r=-0.43$, $p=0.0005$), uric acid ($r=0.32$ $p=0.00092$) and ionized calcium levels ($r=-0.35$ $p=0.00027$) were traced in accordance with the resulting data of a correlation analysis, although correlations observed between leptin and age ($r=0.22$ $p=0.02$), leptin and glycated hemoglobin ($r=-0.21$ $p=0.03$) were weak. A correlation analysis between the level of the hormone leptin and lab markers is represented in the Table 2.

When analyzing measurements in the group of healthy individuals, the following correlations were observed: a strong positive correlation between BMI, waist circumference and BFM ($r=0.85$, $r=0.86$, $p<0.05$), between age and a waist circumference $r=0.67$

($p=0.001$), BFM $r=0.7$ ($p=0.005$), BMI $r=0.95$ ($p<0.05$). Hormone leptin levels in the group of healthy individuals correlated with a waist circumference ($r=0.78$, $p=0.005$), BFM, BMI and age ($r=0.92$, $r=0.94$, $r=0.81$, $p<0.05$), consequently, a strong positive correlation of leptin was also observed with uric acid levels ($r=0.94$) and ionized calcium ($r=0.91$) at $p<0.05$, while a strong negative – between leptin and HDL ($r=-0.76$, $p=0.00008$).

Based on the results, we can state the high importance of an adipose-derived hormones – leptin and obesity factor in the development of atherosclerotic, metabolic complications, and disorders of calcium and purine metabolism, resulting in negative impact on the duration and quality of patient's life.

In order to further analyze and determine the intensity of the effect of leptin measurements on risks for cardiometabolic complications we conducted a regression analysis using a linear regression between hormone leptin levels, clinical, lab, anthropometric and morphometric indicators. Variables that had significant association with leptin in univariate analysis were entered in multivariable analysis. The results are represented in the Table 3, 4, 5.

Table 2. Pearson's correlation coefficients for lab parameters

Parameters	Pearson's correlation coefficient
Age**	0.22
Total cholesterol***	0.40
HDL***	-0.43
LDL – **	0.4
Uric acid ***	0.32
Ionized Ca ²⁺ ***	0.35

notes: * A correlation is significant at $p<0.05$; ** $p<0.01$, *** $p<0.001$

Table 3. Multiple linear regression analysis involving hormone leptin^a levels and clinical parameters

	Beta-coefficient	Standard error	Upper limit 95% CI	Lower limit 95% CI	T-statistics	P
Waist circumference	-0,11	0,12	-0,32	0,18	-0,54	0,58
BFM	-0,23**	0,11	-0,51	-0,07	-2,61	0,01**
BMI	0,21	0,36	-0,37	1,07	0,95	0,34
Age	0,12	0,07	-0,02	0,25	1,67	0,09
SBP	0,03	0,09	-0,17	0,21	0,24	0,8
DBP	-0,02	0,17	-0,37	0,32	-0,15	0,87
Cholesterol	-0,04	1,06	-2,58	1,65	-0,43	0,66
Triglycerides	75,54**	151,63	691,18	1 294,47	6,54	0,04**
HDL	-1,25***	10,01	-63,97	-24,14	-4,4	0,00003***
LDL	0,46	3,79	-2,32	12,77	1,37	0,17
VLDL	-75,44*	333,34	-2 839,66	-1 513,41	-6,52	0,05*
Atherogenic index	-1,11**	5,33	-23,80	-2,56	-2,46	0,01**
Uric acid	0,03	0,01	-0,01	0,02	0,42	0,67
Ionized calcium	-0,48***	1,63	-12,77	-6,27	-5,83	0,001***
Glucose	-0,03	2,08	-4,61	3,68	-0,22	0,82
Glycated Hb	-0,21	1,21	-4,28	0,55	-1,53	0,12

notes: ^a - Dependent variable: leptin; * - coefficient significant at $p<0.05$; ** $p<0.01$, *** $p<0.001$

Table 4. Correlation coefficients of the hormone leptin and the clinical indicators studied: BFM, BMI, age, SBP, DBP, cholesterol, TG, HDL, LDL, VLDL, atherogenic index, uric acid, ionized calcium, glucose, glycated Hb

R	R squared	Adjusted R squared	Predicted R squared
0,83	0,70	0,64	0,55

Table 5. Model validity indicators

	d.f.	SS (sum of squares)	MS	F	P
Regression	16	7 028.54	439.28	12.11	0.000002
Residual	48	2 974.18	36.27		
Total	64	10 002.73			

notes: SS – sum of squares, DF – degrees of freedom, F – F statistics (F criterion)

Taking into consideration this regression model, we are getting 6 indicators with a significant effect on hormone leptin levels at the error level $p<0.05$, namely such indicators as BFM ($\beta=0.23$, $p=0.01$), TG ($\beta=75.54$, $p=0.04$), HDL ($\beta=-1.25$, $p=0.00003$), VLDL ($\beta=-75.44$, $p=0.05$), atherogenic index ($\beta=-1.11$, $p=0.01$) and ionized calcium levels ($\beta=-0.48$, $p=0.001$), the level of triglycerides ($\beta=75.54$, $p=0.04$) and VLDL ($\beta=-75.44$, $p=0.05$) had the strongest effect on hormone leptin levels.

A regression coefficient R equal to 0.83 in multiple regression, obtained as a result of the study, is indicative of a high significance of the model we have got. The value of the R squared is close to 1, thus this model is adequate. Results of the calculations revealed that 70% of the effect, in the dependence model involving plasma levels of the hormone leptin and independent variables, accounts for the factors we have considered, and another 30% – for another factors (Table 4).

The F-value ($F=12.11$) in the Table V indicates that this model properly explains the overall variance of the dependent variable of the hormone leptin from the other morphometric, anthropometric, as well as clinical and lab parameters ($p=0.000002$) evaluated.

The result indicates that hormone leptin levels are highly likely to be predetermined by factorial causes, chosen in the study.

Conclusions.

1. Obesity is a significant social, economic and medical problem which is rapidly spreading.
2. The study indicated the levels of leptin, uric acid, triglycerides, total cholesterol, SBP, DBP and waist circumference to be statistically and significantly higher in patients with excess weight/obesity and hypertension than in healthy individuals ($p<0.05$).
3. Leptin levels predominantly correlated with total cholesterol ($r=0.40$, $p=0.00004$), HDL ($r=-0.43$, $p=0.0005$) and LDL ($r=0.4$ $p=0.0005$), uric acid ($r=0.32$ $p=0.00092$), ionized calcium levels ($r=-0.35$ $p=0.00027$), and least of all correlated with age ($r=0.22$ $p=0.02$).
4. In accordance with a multiple regression analysis, 6 indicators have the most significant effect on hormone leptin levels, namely BFM ($\beta=0.23$, $p=0.01$), TG ($\beta=75.54$, $p=0.04$), HDL ($\beta=-1.25$, $p=0.00003$), VLDL ($\beta=-75.44$, $p=0.05$), atherogenic index ($\beta=-1.11$, $p=0.01$), ionized calcium levels ($\beta=-0.48$, $p=0.001$), while the strongest effect on hormone leptin levels was provided by the level of triglycerides ($\beta=75.54$, $p=0.04$) and VLDL ($\beta=-75.44$, $p=0.05$).
5. Using a linear regression approach it was identified that the level of the hormone leptin, with 70% probability, is predetermined by factorial variables received in the study.
6. The study provided evidence of the strong correlation between hormone leptin levels and specific morphometric, anthropometric, as well as clinical and lab parameters, which may indicate its critical role in bringing cardiometabolic risk and stimulates further research and detection of brand new risk factors and parameters that may affect it.

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SUMMARY

CORRELATION BETWEEN THE LEVELS OF ADIPOSE-DERIVED HORMONE AND CARDIOMETABOLIC MARKERS IN PATIENTS WITH HYPERTENSION AND OBESITY

Goncharuk O., Matyukha L.

P. Shupyk National Healthcare University of Ukraine, Kyiv, Ukraine

The aim - to analyze the relationship between leptin levels and morphometric, anthropometric, biochemical parameters in patients with hypertension and obesity and in healthy individuals.

The study included 64 patients with obesity and hypertension and 21 healthy individuals. The groups were comparable in age and gender. Leptin was determined by enzyme immunoassay method. Data are presented as mean values and the error of the mean ($M \pm m$). Differences were considered statistically significant at $p < 0,05$.

It was found out a strong positive correlation in the group with hypertension and obesity between plasma leptin levels and total cholesterol ($r=0.40$, $p=0.00004$), strong negative correlation between leptin and HDL ($r=-0.43$, $p=0.0005$), uric acid ($r=0.32$ $p=0.00092$) and ionized calcium levels ($r=-0.35$ $p=0.00027$). Leptin levels in the group of healthy individuals correlated with a waist circumference ($r=0.78$, $p=0.005$), BFM, BMI and age ($r=0.92$, $r=0.94$, $r=0.81$, $p<0,05$), uric acid levels ($r=0.94$) and ionized calcium ($r=0.91$) at $p<0.05$.

The present study provides evidence that BFM, TG, HDL, VLDL, atherogenic index, ionized calcium levels and uric acid have a significant impact on serum leptin in patients with hypertension and obesity.

Keywords: leptin, obesity, hypertension, body fat mass.

РЕЗЮМЕ

ВЗАИМОДЕЙСТВИЕ МЕЖДУ УРОВНЕМ ЖИРНО-ПРОИЗВОДНОГО ГОРМОНА И КАРДИОМЕТАБОЛИЧЕСКИМИ МАРКЕРАМИ У ПАЦИЕНТОВ С ГИПЕРТЕНЗИЕЙ И ОЖИРЕНИЕМ

Гончарук О.Ю., Матюхина Л.Ф.

Национальный университет здравоохранения Украины им. П.Л. Шупика, Киев, Украина

Цель исследования - анализ взаимосвязей между уровнем сывороточного лептина и морфометрическими, антропометрическими, биохимическими показателями у пациентов с артериальной гипертензией и ожирением и у здоровых лиц.

В исследование включены 64 пациента с ожирением и артериальной гипертензией (основная группа) и 21 относительно здоровый пациент (контрольная группа). Группы были сопоставимы по возрасту и полу. Лептин определяли иммуноферментным методом. Данные представлены как средние значения и ошибка среднего ($M \pm m$). Статистически значимыми считали различия при $p < 0,05$.

У пациентов основной группы обнаружена сильная положительная корреляционная связь между уровнем лептина в плазме и общим холестерином ($r=0,40$, $p=0,00004$), сильная отрицательная корреляция между лептином и липопротеинами высокой плотности (ЛПВП) ($r=-0,43$, $p=0,0005$), мочевой кислотой ($r=0,32$ $p=0,00092$) и уровнем ионизированного кальция ($r=-0,35$ $p=0,00027$). Уровень лептина в группе здоровых лиц коррелировал с окружностью талии ($r=0,78$, $p=0,005$), жировой массой тела (ЖМТ), индексом массы тела и возрастом ($r=0,92$, $r=0,94$, $r=0,81$, $p < 0,05$), а также мочевой кислотой ($r=0,94$) и уровнем ионизированного кальция ($r=0,91$) при $p < 0,05$.

Результатами проведенного исследования доказано, что ЖМТ, тиреоглобулин, ЛПВП, липопротеины очень высокой плотности, индекс атерогенности, уровни ионизированного кальция и мочевой кислоты оказывают значительное влияние на уровень лептина в сыворотке крови у пациентов с артериальной гипертензией и ожирением.

რეზოუმე

ცემოვანი პორტალების დონის და კარდიომეტებოლური მარკერების ურთიერთქმედება პაციენტები პერიოდებით და სიმუშნით და ხარისხით და განვითარებით და მოწყობებრივად ანთროპომეტრიულ და ბიოქიმიურ მაჩვენებლებს შორის პაციენტებში არტერიული პიპერტენციით, ხიბეუქნით და ჯანმრთელ პირებში.

ო. გონჩარუკი, ლ. მათიუხინა

ჯანმრთელობის დაცვის ა. შემოქმედის სახ. ეროვნული უნივერსიტეტი, კიევი, უკრაინა

კვლევის მიზანს წარმოადგენდა ურთიერთკავშირის ანალიზი შრაგისმიერ ლეპტინისა და მოწყობებრივად, ანთროპომეტრიულ და ბიოქიმიურ მაჩვენებლებს შორის პაციენტებში არტერიული პიპერტენციით, ხიბეუქნით და ჯანმრთელ პირებში.

კვლევაში ჩართული იყო 64 პაციენტი სიმუშნით და არტერიული პიპერტენციით (ძირითადი ჯგუფი) და 21 უდარებელი ჯანმრთელი პირი (საკონტროლო ჯგუფი). ჯანმრთელი ასაკის და სექსის მიხედვით იყო

თაგსებადი. დატბითი განისაზღვრა იმუნოფერმენტული მეთოდით. მონაცემები წარმოდგენილია, როგორც საშუალო მაჩვენებლები და საშუალო ცდომილება ($M \pm m$). სტატისტიკურად მნიშვნელოვნად ითვლებოდა განსხვავება $p < 0,05$.

დადგენილია ძლიერი დადებითი კორელაციური კავშირი ძირითად ჯგუფში პლაზმაში ლეპტინის დონესა და საერთო ქოლესტერინის შორის ($r=0,40$, $p=0,00004$), ძლიერი უარყოფითი კორელაცია დატბითისა და მაღალი სიმკვრივის ლიპოპროტეინებს ($r=-0,43$, $p=0,0005$), შარდმეავას ($r=0,32$ $p=0,00092$) და იონიზებული კალციუმის ($r=-0,35$ $p=0,00027$) დონეებს შორის. ლეპტინის დონე ჯანმრთელ პირებში კორელირებდა

წელის გარშემოწერილობასთან ($r=0,78$, $p=0,005$), სხეულის ცხიმოვანი მასის ინდექსთან, სხეულის მასის ინდექსთან და ასაკთან ($r=0,92$, $r=0,94$, $r=0,81$, $p<0,05$), ასევე, შარდმეავასთან ($r=0,94$) და იონიზებული კალციუმის დონესთან ($r=0,94$) ($p<0,05$).

კვლევის შედეგებით დადგენილია, რომ სხეულის ცხიმოვანი მასის ინდექსი, თირუოგლობულინი, მაღალი სიმკვრივის ლიპოპროტეინები, ძალიან მაღალი სიმკვრივის ლიპოპროტეინები, ათეროგენობის ინდექსი, იონიზებული კალციუმის და შარდმეავას დონე მნიშვნელოვან გავლენას ახდენს ლეპტინის დონეზე არტერიული ჰიპერტენზიის და სიმცუქნის მქონე პაციენტების სისხლის შრატში.

NEUROLOGICAL MANIFESTATIONS OF PROLACTINOMA (CASE REPORT)

Naumova L., Milevska-Vovchuk L., Burak A., Krytskyy T., Pankiv I.

I. Horbachevsky Ternopil National Medical University, Department of Internal Medicine №1, Ukraine

A transient or stable increase of prolactin levels in blood in non-pregnant and non-lactating women is assessed as hyperprolactinemia. In most cases, hyperprolactinemia is caused by the pathology of the pituitary gland of organic or functional genesis and is considered as a marker of dysfunction of hypothalamic-pituitary regulation, which leads to a multifaceted complex of signs of neuroendocrine disorders[1].

Microprolactinomas are considered to be among the most common functioning pituitary adenomas. And although in the structure of general cancer they take a relatively small percentage - 1-2%, but the moral, economic and social damage they cause to society is enormous. And if the connection of prolactinoma today is clearly associated with the development of hyperprolactinemic hypogonadism, the problems of mental disorders on the background of hyperprolactinemia remain unexplored [2,3].

Psychoautonomic disorders play an important role in the clinical picture of pituitary microprolactinoma in women of reproductive age. They develop in more than 40% of cases and lead to a decreased quality of life, the development of social maladaptation and even disability of patients [4].

Various affective phenomena such as anxiety, depression, phobias are accompanied by the syndrome of autonomic dysfunction. Often, the first symptoms of depressive and anxiety disorders develop in 8-12 months after the onset of menstrual disorders. There is a noticeable correlation between the degree of expression of depressive and anxiety disorders and the depth of changes in the hormonal status of women. Neurological manifestations of hormonally active pituitary prolactin in women of reproductive age are often represented by headache, autonomic dysfunction and psychoaffective changes. This complex of signs is often obligatory for patients of reproductive age with pituitary microprolactinomas and is often evaluated by patients in isolation, without indication of concomitant gynecological symptoms, which requires active interviewing regarding the ovarian-menstrual cycle violation as a clinical criterion of hormonal status defect, which directly correlates with the clinical picture

of neurological disorders. This approach will provide an early diagnostics and timely commencement of a pathogenetic antihyperprolactinemic therapy, against which this group of symptoms is largely eliminated [5].

Bromocriptine shrinks the tumor and returns prolactin levels to normal in the majority of patients. To avoid side effects such as nausea and dizziness, it is important to start the bromocriptine treatment slowly. Usual maintenance doses are 2.5 (one tablet) to 7.5 mg (3 tablets) daily.

Bromocriptine treatment should not be interrupted without consulting an endocrinologist. Prolactin levels often rise again in most people when the drug is discontinued.

Another dopamine agonist is cabergoline, which may be more effective and better tolerated than bromocriptine. Another advantage of cabergoline is that it may be prescribed as a weekly dosage of 0.5 (one tablet) to 2.0 (4 tablets) mg weekly [6].

One of the most principal and frequent reasons that cause the failure of the menstrual cycle are diseases of ovaries, for example, the infections of pelvic organs and genitals caused by Chlamydia, Ureaplasma, Mycoplasma. Another reason is the dysfunction of hypothalamic-pituitary system and thyroid gland. In case of hormone changes the menstrual cycle becomes irregular. Moreover, external factors, such as climate changes, excessive exercises, smoking, alcohol, drugs, diet, stress can also influence the regularity of the menstrual cycle. Besides, the irregular menstrual cycle is found in patients who take some hormone drugs, antidepressants, anticoagulants. In some episodes the failure of the menstrual cycle is a hereditary disease.

Prolactinomas belong to the group of benign adenomas, most often found among pituitary tumors (up to 30%), extremely rarely become malignant and observed in women of the child-bearing age group, 6-10 times more often than in men. The size of prolactinoma usually does not exceed 2-3 mm, but in men, as a rule, there are large adenomas more than 1 cm in diameter [7].

Prolactinomas are hormone-active pituitary adenomas secreting prolactin - a "milk hormone" that stimulates postpartum lactation in women. Normally, in smaller quantities, prolactin