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Effect of visceral obesity on main artery elasticity and vascular age in patients with hypertension, obesity, and type 2 diabetes

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Aim. To assess the effect of visceral obesity on main artery elasticity and vascular age in patients with hypertension (HTN), obesity, and type 2 diabetes (T2D).

Material and methods. A total of 320 patients with stage II-III HTN aged 45-70 years were divided into 4 groups: isolated HTN (group 1), HTN and obesity (group 2), HTN, obesity and T2D (group 3), HTN and T2D without obesity (group 4). We assessed the clinical status, parameters of visceral obesity, main artery elasticity, and vascular age. We used nonparametric statistics, Spearman correlation analysis.

Results. At least 50% of all patients had visceral obesity, despite no BMI-estimated obesity in groups 1 and 4: 57,5 vs 100,0 vs 100,0 vs 50,0% in groups 1, 2, 3 and 4, respectively ($p<0,0001$).

In the groups where hypertension was combined with obesity and T2D, the proportion of patients with leptin content above 32,7 ng/ml significantly increased to 80% (in total for groups 2 and 3) compared with 25,0% among HTN people without obesity (in total for groups 1 and 4). There was a significant increase in proportion of patients with a adiponectin decrease <14,6 ng/ml among patients with a combination of HTN and T2D ± obesity (45% in total for groups 3 and 4) in comparison with those with HTN and without T2D ± obesity (22,5% in total for groups 1 and 2). The visceral adiposity index (VAI) was significantly higher among patients with HTN, obesity and T2D compared with those with isolated HTN and HTN in combination with T2D only (2,96 [2,36; 3,98] vs 1,87 [1,40; 2,67] vs 2,22 [1,61; 3,26], respectively). A higher proportion of subjects with adipose tissue dysfunction was noted in groups 2 and 3 compared to groups 1 and 4 (75 vs 81,1 vs 41,5 vs 53,4%, respectively, $p_{1-2}<0,001$, $p_{1-3}<0,001$, $p_{2-4}=0,023$, $p_{3-4}=0,002$). The proportion of patients with a pulse wave velocity >10 m/s was consistently more common among patients of group 3 compared with patients in groups 1 and 2 (77,0 vs 57,9 and 55,3%, respectively, $p_{1-3}=0,004$, $p_{2-3}=0,006$).

Vascular age was significantly lower in group 1 compared with groups 3 and 4 (64,0 [57,8; 71,0] vs 69,0 [62,0; 73,0] and 69,5 [66,0; 74,3] years, respectively), as well as in group 2 compared with group 4 (64,0 [56,5; 70,5] vs 69,5 [66,0; 74,3] years). The 5-year risk of cardiovascular events was significantly higher among patients with hypertension, obesity and T2D and those with HTN and T2D without obesity, compared with patients with isolated HTN, and with those with HTN and obesity (5,9 [3,9; 7,9] and 6,5 [4,7; 8,7] vs 4,4 [2,7; 6,8] and 3,6 [2,4; 5,8], respectively).

Correlation analysis revealed the relationship between the visceral obesity parameters, main artery elasticity, vascular age and the 5-year risk of cardiovascular events, demonstrating the special aspects of HTN course in each of the studied groups.

Conclusion. The paper showed peculiarities of the effect of visceral obesity on main artery elasticity and vascular age in patients with HTN in combination with obesity and T2D.

Keywords: hypertension, visceral obesity, diabetes, vascular age.

Relationships and Activities. The study was performed at the expense of scientists' grant of Volgograd State Medical University (order 29-KO dated June 2, 2020).

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Currently, there is a tendency towards an increase in the prevalence of obesity [1] and a understandable increase in the number of persons with type 2 diabetes (T2D) [2]. Both diseases increase the risks of cardiovascular diseases and events. Thus, the presence of hypertension (HTN) in a patient with T2D additionally quadruples the risk [3].

The pathogenesis of vascular involvement in patients with HTN combined with obesity, T2D are not only endothelial dysfunction, hyperuricemia, activation of the sympathoadrenal and renin-angiotensin-aldosterone systems, secretion of pro-inflammatory cytokines, microcirculation disorders, decreased elasticity of major vessels, but also additional ones inherent in obesity and T2D — visceral obesity, carbohydrate and lipid metabolism disorders, insulin resistance. In earlier papers, we have already considered the role of low-grade chronic inflammation, endothelial dysfunction, insulin resistance in target organ damage in hypertensive persons with obesity, T2D, as well as the importance of leptin and adiponectin in increasing the vascular stiffness in hypertensive people with obesity [4-9].

The aim was assess the effect of visceral obesity on main artery elasticity and vascular age in patients with hypertension (HTN), obesity, and T2D.

Material and methods

This open-label, comparative, prospective, parallel group study included 320 patients with stage II-III HTN aged 45-70 years with unreached target blood pressure (BP). Patients were randomized into 4 groups, matched by sex, age, smoking, history of HTN, office systolic BP (SBP) and heart rate (HR), depending on the presence/absence of obesity and/or T2D. The first group consisted of 102 patients with isolated HTN (without obesity and T2D), the second — 90 patients with HTN and obesity, the third — 96 patients with HTN, obesity and T2D, the fourth — 32 patients with HTN and T2D without obesity (Table 1). Patients with T2D (groups 3 and 4) were also comparable in disease duration and dosages of hypoglycemic medications. The 1st and 4th groups were control. There were following exclusion criteria: uncontrolled malignant hypertension, prior acute coronary syndrome and stroke within last 6 months, hemodynamically relevant heart defects and arrhythmias, type 1 diabetes, class III obesity, manifested liver failure, stage >C3b chronic disease kidney, alcohol abuse, any other diseases that could affect the study results. The nature of the study is an in parallel groups.

In all patients, we assessed their clinical status (complaints, medical and life history, risk factors for hypertension, overall health status, office BP, heart rate), anthropometric data (height, weight, body mass index (BMI), level of subcutaneous and visceral fat analyzed by bioelectrical impedance analysis using the Omron BF-508 system, waist circumference (WC), hip circumference (HC). Abdominal obesity was considered to be a waist-to-hip ratio (WHR) >0,9 for men and WHR >0,85 for women; WC ⩾102 cm for men and WC ⩾88 cm for women [1]; visceral obesity — visceral fat ⩾9% [1].

To determine obesity laboratory markers, the serum concentration of leptin (Diagnostics Biochem, Canada) and adiponectin (Mediagnost, GmbH, Germany) by sandwich enzyme-linked immunosorbent assay using a Uniplan analyzer, Russia. There were following reference values: for leptin — 3,7-11,1 ng/ml (for women ⩽27,6 ng/ml, for men ⩽13,8 ng/ml), for adiponectin — 8,2-19,1 ng/ml.

Visceral obesity index (VAI) was estimated. The severity of adipose tissue dysfunction (ATD) was assessed taking into account the patient age [10].

To analyze the main artery elasticity, the pulse wave velocity (PWV) was measured using a Poly-Spectr-8/E sphygmographic system (Russia). PWV in elastic (PWVe) and muscular (PWVm) arteries was assessed in the carotid-femoral and carotid-radial segments, respectively. The normal values of PWVm and PWVe were interpreted individually using the software, taking into account the sex and age of the patients.

Vascular age and 5-year cardiovascular risk were assessed using the ADVANT'AGE calculator for smartphones (version 2021).

Statistical analysis was performed using the Microsoft Excel 2010 and Statistica 10.0 software package. The normality of the distributions was assessed using the Shapiro-Wilk test. In non-normal distribution, nonparametric statistical methods were used. Quantitative data are presented as Me [Q25; Q75], where Me is the median, Q25 and Q75 are the 25th and 75th percentiles, respectively; qualitative variables are presented as prevalence (%). Multiple comparison of four independent samples was performed using the Kruskal-Wallis test. The differences were considered significant at $p<0,05$. When significant differences were identified according to the Kruskal-Wallis test, a subsequent Bonferroni-Dunn comparisons were carried out. In the case of dichotomous variables, the significance of the differences was analyzed using Fisher's exact test. To assess the relationship statistics, a Spearman correlation was used.

This study was performed in accordance with the Helsinki declaration, Good Clinical Practice, World Medical Association (2008), and the Constitution of the Russian Federation. The study was approved by the Regional Ethics Committee. All patients signed written informed consent.

Table 1

Clinical and demographic parameters in studied patients (Me [25%; 75%])

Parameter	Group 1 HTN without obesity and T2D	Group 2 HTN + obesity without T2D	Group 3 HTN + obesity + T2D	Group 4 HTN + T2D without obesity
Number of patients, n	102	90	96	32
Men/women, %	34,4/65,6	37,8/62,2	32,3/67,7	34,4/65,6
Age, years	62,0 [55,0; 66,0]	62,0 [55,3; 65,8]	62,0 [58,0; 65,0]	63,0 [60,0; 66,0]
BMI, kg/m^2	26,7*,† [25,4; 28,7]	32,9†† [31,1; 36,0]	34,7§§ [32,5; 37,5]	27,2 [25,9; 28,5]
WC, cm	94,0*,† [83,0; 100,0]	105,0†† [99,3; 111,8]	107,0§§ [102,0; 116,0]	93,5 [88,3; 99,3]
HC, cm	102,0*,† [99,0; 105,0]	115,0†† [110,0; 125,0]	116,0§§ [108,0; 122,0]	103,5 [98,0; 105,3]
WHR	0,91 [0,82; 0,96]	0,91 [0,85; 0,99]	0,94 [0,88; 1,00]	0,91 [0,87; 0,96]
Patients with abdominal obesity assessed by WHR,%	51,2*,†,§	73,7**	86,3	71,9
Patients with abdominal obesity assessed by WC,%	61,0*,†,§	100,0††	100,0§§	90,6
Subcutaneous fat, %	30,7*,† [26,0; 39,2]	45,1†† [39,3; 49,4]	44,7§§ [38,1; 50,0]	35,2 [27,0; 40,1]
Visceral fat, %	10,5*,† [8,0; 13,0]	14,0†† [11,0; 16,0]	14,0§§ [13,0; 17,0]	9,5 [8,0; 11,0]
Prevalence of visceral obesity, %	57,5*,†	100,0††	100,0§§	50,0
Smokers, %	21,6	21,1	20,8	21,9
Duration of HTN, years	12,0 [8,0; 19,0]	12,0 [7,0; 20,0]	15,0 [9,5; 20,0]	12,0 [7,0; 20,0]
Prevalence of statin therapy, %	8,8†,§	7,8**,††	50,0	59,4
Duration of T2D, years	0†,§	0**,††	7,0 [3,0; 10,0]	7,0 [4,5; 10,0]
Office SBP, mm Hg	160 [150; 170]	160 [150; 170]	159 [150; 170]	160 [150; 164]
Office DBP, mm Hg	100†,§ [91; 103]	100**,†† [94; 108]	93 [90; 100]	90 [83; 100]
Office PP, mm Hg	60†,§ [50; 70]	60 [55; 70]	62 [60; 77]	70 [60; 75]
Heart rate, bpm	70 [65; 75]	73 [64; 78]	70 [64; 76]	70 [65; 80]

Note: * — significance of differences between groups 1 and 2, † — significance of differences between groups 1 and 3, § — significance of differences between groups 1 and 4, ** — significance of differences between groups 2 and 3, †† — significance of differences between groups 2 and 4 groups, §§ — significance of differences between 3 and 4 groups.

Abbreviations: HTN — hypertension, DBP — diastolic blood pressure, BMI — body mass index, HC — hip circumference, WC — waist circumference, WHR — waist–to–hip ratio, PP — pulse pressure, SBP — systolic blood pressure, T2D — type 2 diabetes.

Results

There were significant differences in BMI between 1 and 2, 1 and 3, 2 and 4, 3 and 4 groups: BMI was higher in groups 2 and 3 (p<0,0001).

WC and HC were also significantly higher in the groups of patients with HTN and obesity, as well as with HTN, obesity and T2D in comparison with those with HTN and HTN + T2D without obesity (p<0,0001). There was a tendency towards higher values of WHR among persons with HTN, obesity and T2D. However, the differences were not significant.

Noteworthy is the high percentage of patients with abdominal obesity determined by WC, WHR, and visceral fat in all studied groups. As for proportion of obese (assessed by WC) patients, significant differences were noted between 1 and 2, 1 and 3 groups. In group 1, the proportion of patients with abdominal obesity assessed by WHR was significantly lower in comparison with groups 2, 3 and 4 (Table 1).

Subcutaneous and visceral fat levels were lower in groups 1 and 4 compared to groups 2 and 3 (p<0,0001 for both). At the same time, at least 50% of patients in all groups had visceral obesity, despite the absence of obesity assessed by BMI in groups 1 and 4: 57,5 vs 100,0 vs 100,0 vs 50,0% in 1, 2, 3 and 4 groups, respectively (p<0,0001).

The proportion of statin therapy was significantly higher among patients with HTN, obesity and T2D, as well as among those with HTN and T2D without obesity, which is due to compliance with national guidelines and standards for the management of T2D patients. Statin dosages was comparable in all 4 groups.

Differences were noted between groups 1 and 2 in comparison with groups 3 and 4 in terms of office diastolic BP (DBP) — p<0,0001: lower DBP values

Table 2

Laboratory markers of obesity in studied patients (Me [25%; 75%])

Parameter	Group 1 HTN without obesity and T2D	Group 2 HTN + obesity without T2D	Group 3 HTN + obesity + T2D	Group 4 HTN + T2D without obesity
Leptin, ng/ml	15,2* [6,6; 32,7]	53,8 [38,4; 75,8]	42,8 [25,1; 54,0]	13,2 [9,9; 18,2]
Adiponectin, ng/ml	21,7 [14,6; 32,9]	18,6 [15,3; 22,4]	16,5 [11,2; 20,5]	17,5 [7,1; 27,6]

Note: * — significance of differences between groups 1 and 2.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes.

Table 3

Elasticity parameters of the main arteries in studied patients (Me [25%; 75%])

Parameter	Group 1 HTN without obesity and T2D	Group 2 HTN + obesity without T2D	Group 3 HTN + obesity + T2D	Group 4 HTN + T2D without obesity
PWVm, m/s	8,2 [7,4; 10,0]	8,4 [7,7; 9,2]	9,0 [8,1; 10,3]	8,9 [7,0; 10,8]
PWVm >10 m/s, % of patients	44,7	34,2**	55,2	44,8
PWVe, m/s	8,9† [8,2; 10,2]	8,8** [7,7; 10,6]	10,4 [9,1; 12,4]	9,2 [8,3; 11,4]
PWVe >10 m/s, % of patients	57,9†	55,3**	77,0	62,1
PWVm/PWVe	0,93 [0,77; 1,03]	0,92 [0,83; 1,03]	0,87 [0,80; 0,97]	0,91 [0,80; 1,03]

Note: † — significance of differences between groups 1 and 3, ** — significance of differences between groups 2 and 3.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes, PWVm — pulse wave velocity in muscular arteries, PWVe — pulse wave velocity in elastic arteries.

Table 4

Assessment of vascular age and 5-year cardiovascular risk in studied patients (Me [25%; 75%])

Parameter	Group 1 HTN without obesity and T2D	Group 2 HTN + obesity without T2D	Group 3 HTN + obesity + T2D	Group 4 HTN + T2D without obesity
Vascular age, years	64,0†,§ [57,8; 71,0]	64,0†† [56,5; 70,5]	69,0 [62,0; 73,0]	69,5 [66,0; 74,3]
5-year risk.	4,4†,§ [2,7; 6,8]	3,6**,†† [2,4; 5,8]	5,9 [3,9; 7,9]	6,5 [4,7; 8,7]
5-year risk grade				
Low, % of patients	16,9†,§	17,5**,††	3,1	0,0
Moderate, % of patients	46,5§	44,4	33,3	25,0
High, % of patients	36,6†,§	33,3**,††	61,5	75,0
Very high, % of patients	0,0*	4,8	2,1	0,0

Note: * — significance of differences between groups 1 and 2, † — significance of differences between groups 1 and 3, § — significance of differences between groups 1 and 4, ** — significance of differences between groups 2 and 3, †† — significance of differences between groups 2 and 4 groups, §§ — significance of differences between 3 and 4 groups.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes.

are characteristic of patients with T2D. An increase in office pulse pressure (PP) was naturally revealed in persons of groups 3 and 4 compared with groups 1 and 2 (p=0,0009 for both).

In all studied groups, a relationship was found between the level of visceral fat and WC (r=0,74 vs r=0,61 vs r=0,55 vs r=0,59 in groups 1, 2, 3 and 4, respectively, p<0,05).

There were significant correlations between BMI and WC (r=0,79), HC (r=0,79), subcutaneous (r=0,64) and visceral fat (r=0,67) extent, and leptin level (r=0,55).

Of all the revealed relationships with the WHR, the most clinically and pathogenetically important are correlations with the levels of visceral fat (r=0,52, p<0,05) and adiponectin (r=-0,34, p<0,05).

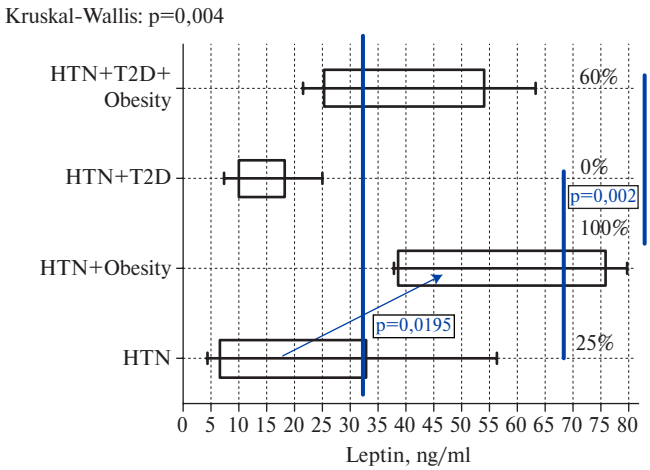

Figure 1. Distribution of patients by leptin level and proportion of subjects with analyte >32,7 ng/ml.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes.

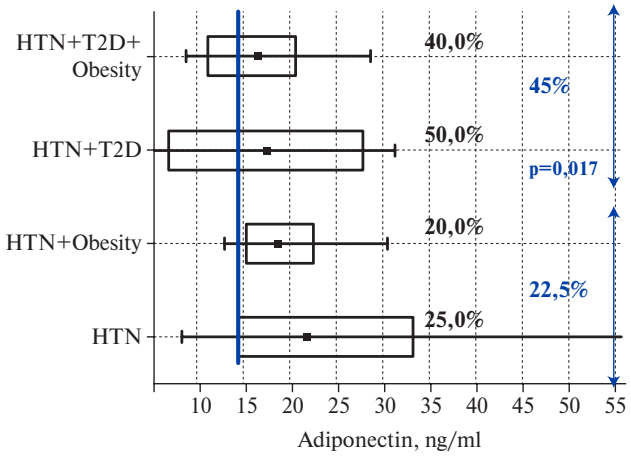

Figure 2. Distribution of patients by adiponectin level and proportion of patients with analyte <14,6 ng/ml.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes.

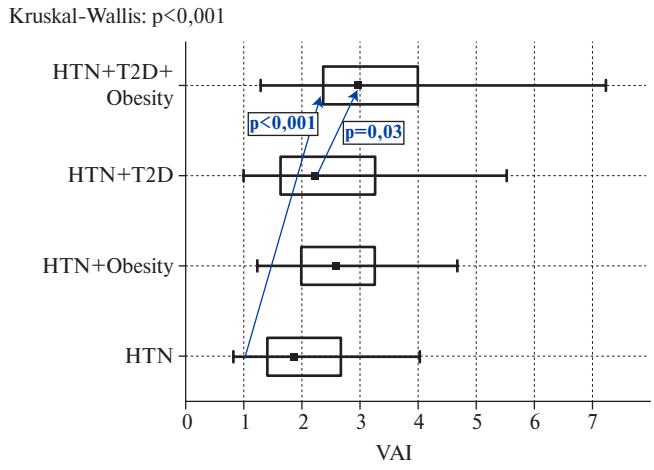

Figure 3. Distribution of patients by VAI.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes, VAI — visceral adiposity index.

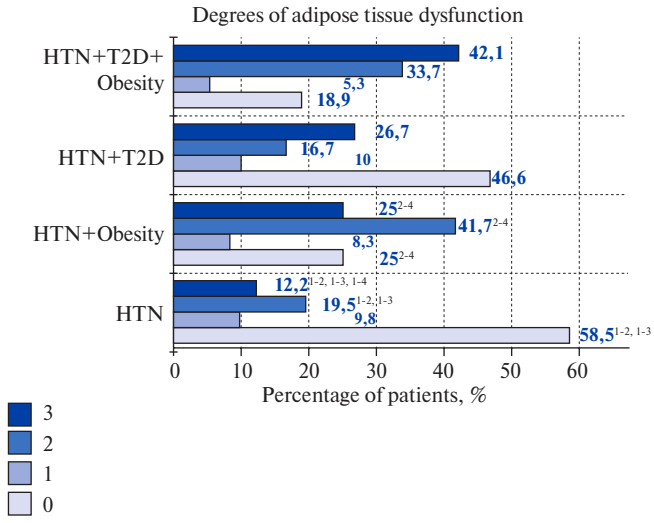

Figure 4. Distribution of patients by the severity of adipose tissue dysfunction.
Note: 0 — no dysfunction, 1 — mild dysfunction, 2 — moderate dysfunction, 3 — severe dysfunction, 1-2, 1-3, 1-4, 2-4 — significant differences between 1 and 2, 1 and 3, 1 and 4, 2 and 4 groups.
Abbreviations: HTN — hypertension, T2D — type 2 diabetes.

The analysis of obesity markers revealed an increase in the serum concentration of leptin in patients with HTN, obesity and those with hypertension, obesity, and T2D (Table 2). However, the differences reached the significance level only when comparing groups 1 and 2 (15,2 [6,6; 32,7] vs 53,8 [38,4; 75,8] ng/ml, p=0,02), which is probably associated with a large variation. At the same time, in the groups where HTN was combined with obesity ± T2D, the proportion of patients with leptin >32,7 ng/ml significantly increased to 80% (total for 2 and 3 groups) compared with 25,0% among hypertensive patients without obesity (total for groups 1 and 4) — Figure 1.

Correlation analysis showed the presence of significant relationships between the concentration of leptin and PWVe (r=0,58) in the group of isolated HTN, as well as between leptin levels and PP (r=0,99), ATD (r=0,59) in those with HTN and obesity.

Noteworthy is the decrease in serum adiponectin levels when obesity, T2D, and especially the combination of obesity and T2D are combines with HTN, but no significant differences were found between the groups (Table 2). According to interquartile intervals, adiponectin level of 14,6-22,5 ng/ml was in the majority of studied patients. However, there was a significant increase in the prevalence of adiponectin <14,6 ng/ml among

patients with a combination of HTN and T2D ± obesity (45% for groups 3 and 4) in comparison with hypertensive patients without T2D ± obesity (22,5% for groups 1 and 2) (Figure 2).

In group 4, the adiponectin level correlated with office (r=-0,76), office DBP (r=-0,85), PWVe (r=-0,97) — p<0,05 for all.

VAI was significantly higher among patients with HTN, obesity and T2D compared with those with isolated HTN and HTN in combination with T2D without obesity (2,96 [2,36; 3,98] vs 1,87 [1,40; 2,67] vs 2,22 [1,61; 3,26], respectively) (Figure 3).

A higher proportion of patients with adipose tissue dysfunction was noted in groups 2 and 3 compared to groups 1 and 4 (75 vs 81,1 vs 41,5 vs 53,4%, respectively, p_{1-2}<0,001, p_{1-3}<0,001, p_{2-4}=0,023, p_{3-4}=0,002). The distribution of patients depending on ATD is shown in Figure 4.

When HTN combined with obesity and T2D, there is an increase in PWVm but the differences between the groups did not reach the significance level (Table 3).

PWVe was significantly higher among patients with HTN, obesity and T2D in comparison with both subjects with isolated HTN and those with HTN and obesity (10,4 [9,1; 12,4] vs 8,9 [8,2; 10,2] and 8,8 [7,7; 10,6] m/s, respectively). The proportion of patients with PWVe >10 m/s was consistently more common among group 3 patients compared with groups 1 and 2 (77,0 vs 57,9 and 55,3%, respectively, p_{1-3}=0,004, p_{2-3}=0,006). The correlation was established between PWVe and leptin concentration (r=0,58), ATD (r=0,53) in group 1, as well as between PWVe and adiponectin concentration (r=-0,96) in group 4 (p<0,05 for all).

Vascular age was significantly lower in group 1 in comparison with groups 3 and 4 (64,0 [57,8; 71,0] vs 69,0 [62,0; 73,0] and 69,5 [66,0; 74,3] years, respectively), as well as in group 2 in comparison with 4 (64,0 [56,5; 70,5] vs 69,5 [66,0; 74,3] years) (Table 4).

The five-year risk of CVE was significantly higher among patients with HTN, obesity and T2D and those with HTN and T2D without obesity compared with subjects with isolated HTN and HTN + obesity (5,9 [3, 9; 7,9] and 6,5 [4,7; 8,7] vs 4,4 [2,7; 6,8] and 3,6 [2,4; 5,8], respectively). Risk stratification showed that the total percentage of persons with high and very high risk was significantly higher among groups 3 and 4 compared to groups 1 and 2.

Intragroup correlation analysis revealed significant correlations between visceral fat level and BMI (r=0,79), heart rate (r=0,55), 5-year cardiovascular risk (r=0,64) among patients with isolated HTN, as well as significant relationships between the visceral fat level and adiponectin (r=-0,80), vascular age (r=0,58), 5-year cardiovascular risk (r=0,72) in patients with HTN and obesity without T2D.

Correlation analysis in all groups revealed a significant relationship of age with vascular age (r=0,78) and 5-year CVD risk (r=0,54).

Discussion

Significant differences in BMI between groups 1 and 2, 1 and 3, 2 and 4, 3 and 4 are due to the study design. With an increase in BMI, the percentage of subcutaneous and visceral fat, as well as WHR naturally increased.

Identified high prevalence of abdominal obesity in the patients with isolated HTN and HTN in combination with T2D without obesity (with normal or overweight), as well as of visceral obesity in people with normal and overweight (according to BMI) has an important practical significance. So, for the diagnosis of obesity, it is advisable to assess not only BMI, but also WC, WHR, as well as the visceral fat proportion.

Significantly higher numbers of office PP in patients with a combination of HTN and T2D are associated with a decrease in office DBP and indicate an increase in arterial stiffness and subclinical target organ damage [11].

Revealing significant correlations between BMI, WC, HC, WHR, levels of subcutaneous and visceral fat and laboratory markers of obesity confirms the pathogenetic role of obesity in the progression of target organ damage. Different nature of interrelations in each of the studied groups indicates a different degree of significance of pathogenetic links as obesity and T2D join the HTN.

A significant increase in PWVe among patients with HTN, obesity and T2D in comparison with both individuals with isolated HTN and those with HN and obesity (10,4 [9,1; 12,4] vs 8,9 [8,2; 10,2] and 8,8 [7,7; 10,6] m/s, respectively) is associated with early main artery remodeling in patients with HTN and comorbidities. Apparently, both obesity and T2D potentiate the negative effect on the vascular wall.

The highest percentage of patients with PWVe >10 m/s, which is a sign of asymptomatic vascular involvement and an independent prognostic marker for fatal and non-fatal cardiovascular events, was naturally more common among patients in group 3 compared with groups 1 and 2 (77,0 vs 57,9 and 55,3%, respectively, p_{1-3}=0,004, p_{2-3}=0,006). This indicates an increase in stiffness and allow assessing the true arterial wall damage [12].

The concept of main artery stiffness is associated with the concept of vascular age. Currently, a new risk stratification algorithm in hypertensive patients receiving antihypertensive therapy is being

used — the ADVANT'AGE vascular age calculator for smartphones (version 2021). Demographic parameters, smoking status, SBP, previous antihypertensive therapy and diabetes, total cholesterol, high-density lipoprotein cholesterol, glucose and creatinine are taken into account.

A significant increase in vascular age in groups 3 and 4 compared with group 1, as well as in group 2 compared with group 4, was associated with an increase in the 5-year cardiovascular risk among patients with HTN, obesity and T2D and those with HTN and T2D without obesity in comparison with patients with isolated HTN, as well as with HNT and obesity. Risk stratification revealed that the total proportion of patients with high and very high risk was significantly higher among patients of groups 3 and 4 compared with those in groups 1 and 2. This justifies a high cardiovascular mortality among patients with a combination of HTN and T2D and/or obesity.

It is acknowledged that adipokines can not only affect vascular function, but also contribute to the strengthening of the relationship between obesity and HTN [13].

In parallel with a decrease in the main artery elasticity, there was an increase in the concentration of leptin and a decrease of adiponectin level with an increase in BMI in hypertensive patients. One of the pathogenetic mechanisms of increasing the large vessel stiffness is associated with the production of hormones and cytokines by metabolically active adipose tissue, including angiotensinogen and angiotensin II. The protective role of adiponectin and the negative role of leptin in main artery damage are discussed. The obtained data are comparable with foreign studies, which indicate that the serum concentration of adipokines can be a predictor of arterial stiffness in patients with HTN [14]. There are publications on leptin activation by the sympathetic nervous system in obesity [15]. In addition to chronic hyperleptinemia due to tissue resistance to leptin, local synthesis of angiotensinogen by adipocytes and hyperinsulinemia contribute to the development and progression of HTN in obese patients.

Adiponectin is an anti-inflammatory adipokine and insulin sensitizer [16]. Vascular adiponectin protection may be associated with an improvement in endothelial dysfunction, a decrease in oxidative stress, and an increase in endothelial nitric oxide synthase expression due to the activation of adenosine 5'-monophosphate-activated protein kinase by AdipoR1 and the action of a peroxisome proliferator-activated receptor (PPAR)-α2 signaling [16].

Established correlations of PWVe with leptin concentration and ATD in group 1, as well as of PWVe and adiponectin concentration in group 4 indicate a pathogenetic relationship between the main artery elasticity and adipokine status in the studied groups of hypertensive patients.

Conclusion

Thus, the data obtained showed peculiarities of the effect of visceral obesity on main artery elasticity and vascular age in patients with HTN in combination with obesity and T2D.

Relationships and Activities: none.

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