MINISTRY OF EDUCATION AND SCIENCE OF UKRAINE
SUMY STATE UNIVERSITY
ACADEMIC AND RESEARCH MEDICAL INSTITUTE

Eastern Ukrainian Medical Journal

116, Kharkivska st., Sumy 40007, Ukraine eumj.med.sumdu.edu.ua
e-mail: eumj@med.sumdu.edu.ua ISSN: 2663-5909 (print)/2664-4231 (online)

How to cite / Як цитувати статтю: Psarova VH, Kochuieva MM, Gogunska IV, Kyrychenko NM, Kochuiev GI, Cherkashyna AI, Ivanova DO. Influence of prediabetes on cardiovascular remodeling indicators in hypertensive patients with obesity. *East Ukr Med J*. 2024;12(4):877-885

DOI: https://doi.org/10.21272/eumj.2024;12(4):877-885

ABSTRACT

INFLUENCE OF PREDIABETES ON CARDIOVASCULAR REMODELING INDICATORS IN HYPERTENSIVE PATIENTS WITH OBESITY

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The purpose of the study was to evaluate the effect of prediabetes on cardiovascular remodeling in hypertensive patients with obesity.

Materials and methods: To examine 200 patients with stage II hypertension (grade 2) and obesity (grades I-II), clinical, anamnestic, anthropometric, biochemical, and instrumental methods were employed. Among these, 60 patients had carbohydrate metabolism disorders. The degree of these disorders was assessed using fasting glycemia, impaired glucose tolerance, glycosylated hemoglobin (HbA1c), and glucose tolerance tests. Impaired fasting glycemia was established at values of glucose concentration in venous plasma ≥ 6.1 and < 7.0 mmol/l, in whole capillary blood ≥ 5.6 and < 6.1 mmol/l, and glucose index < 7.8 mmol/l both in whole capillary blood and in venous plasma during the oral glucose tolerance test. Impaired glucose tolerance was diagnosed based on fasting glucose concentrations of < 6.1 mmol/l in whole capillary blood and < 7.0 mmol/l in venous plasma, and ≥ 7.8 < 11.1 mmol/l both in whole capillary blood and in venous plasma according to the oral glucose tolerance test. The HOMA index determined insulin resistance. The data were analyzed using the statistical software SPSS 17 (IBM) and Microsoft Office Excel 2003. Results are presented as means ± standard deviation, with significance as $p < 0.05$ in all cases.

Research results. In obese hypertensive patients with prediabetes, significantly higher values of the intima-media thickness of the common carotid artery and the carotid artery at the bifurcation level were recorded ($p = 0.027$ and $p = 0.012$ respectively), as well as a significantly higher pulse wave velocity in the carotid artery ($p = 0.022$). Cardiac remodeling assessment results demonstrated that patients with prediabetes had significantly larger left ventricular, left atrial, wall

thickness, and left ventricular myocardial mass index, with no differences in ejection fraction values, compared to patients without prediabetes. The presence of prediabetes was characterized by a decrease in the rate of early filling of the left ventricle (p = 0.000) and the ratio of the rates of early and late filling (p = 0.000) in the absence of significant differences in the levels of the integral index of diastolic function: the ratio of the maximum speed of early diastolic flow to the maximum speed of early filling of the left ventricle (E/e').

Conclusions. Violations of carbohydrate metabolism at the stage of prediabetes in hypertensive patients with obesity contribute to the progression of vascular remodeling. They are associated with the severity of hypertrophic changes in the left ventricle. Evaluation of indicators of cardiovascular remodeling depending on the variant of prediabetes (fasting hyperglycemia and impaired glucose tolerance) did not demonstrate reliable differences in any of the indicators.

Keywords: arterial hypertension, obesity, cardiovascular remodeling, prediabetes.

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ВПЛИВ ПРЕДІАБЕТУ НА ПОКАЗНИКИ СЕРЦЕВО-СУДИННОГО РЕМОДЕЛЮВАННЯ У ГІПЕРТЕНЗИВНИХ ПАЦІЄНТІВ ІЗ ОЖИРІННЯМ

Мета роботи – оцінити вплив прediабету на показники серцево-судинного ремоделювання у хворих на артеріальну гіпертензію з ожирінням.

Матеріали і методи: Клінічні, анамнестичні, антропометричні, біохімічні та інструментальні методи використовували для обстеження 200 хворих на артеріальну гіпертензію II стадії 2 ступеня з ожирінням I-II ступеня, із яких 60 пацієнтів мали порушення вуглеводного обміну. Для визначення ступеня порушень вуглеводного обміну оцінювали глікемію натще, порушення толерантності до глюкози, глікозильований гемоглобін, проводили глюкозотолерантний тест. Порушену глікемію натще встановлювали при значеннях концентрації глюкози в венозній плазмі ≥ 6,1 і < 7,0 ммоль/л, в цільній капілярній крові ≥ 5,6 и < 6,1 ммоль/л та показнику глюкози < 7,8 ммоль/л як в цільній капілярній крові так і в венозній плазмі при проведенні перорального глюкозотолерантного тесту. Порушення толерантності до глюкози діагностували за показниками концентрації глюкози натще < 6,1 ммоль/л в цільній капілярній крові й < 7,0 ммоль/л в венозній плазмі та ≥ 7,8 < 11,1 ммоль/л як в цільній капілярній крові так і в венозній плазмі за даними перорального глюкозотолерантного тесту. Інсулінорезистентність визначали за індексом HOMA. Статистична обробка отриманих даних проводилась 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. За наявності прediабету у гіпертензивних пацієнтів з ожирінням зафіксовано достовірно більші значення товщини інтима-медіа загальної сонної артерії та сонної

артерії на рівні біфуркації (p = 0,027 і p = 0,012 відповідно), а також достовірно більшу швидкість пульсової хвилі в сонній артерії (p = 0,022). Результати оцінювання кардіального ремоделювання продемонстрували, що пацієнти з прediабетом мали достовірно більші розміри лівого шлуночка, лівого передсердя, товщину стінок та індекс маси міокарда лівого шлуночка за відсутності відмінностей щодо значення фракції викиду, порівняно з пацієнтами без прediабету. Наявність прediабету характеризувалася зниженням швидкості раннього наповнення лівого шлуночка (p = 0,000) і співвідношення швидкостей раннього та пізнього наповнення (p = 0,000) за відсутності достовірних відмінностей щодо рівнів інтегрального показника діастолічної функції: співвідношення максимальної швидкості раннього діастолічного потоку до максимальної швидкості раннього наповнення лівого шлуночка (E/e´).

Висновки. Порушення вуглеводного обміну на етапі прediабету у гіпертензивних пацієнтів з ожирінням сприяють прогресуванню судинного ремоделювання та асоціюються з вираженістю гіпертрофічних змін лівого шлуночка. Оцінювання показників серцево-судинного ремоделювання залежно від варіанта прediабету (гіперглікемія натще і порушення толерантності до глюкози) не продемонструвало достовірних відмінностей за жодним із показників.

Ключові слова: артеріальна гіпертензія, ожиріння, серцево-судинне ремоделювання, прediабет.

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LIST OF ABBREVIATIONS

A – maximum late (atrial) filling speed;
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;
LVEF – left ventricular 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;
EDV – end-diastolic volume;
ESD – end-systolic volume;
LAD – left atrial diameter;
AD – aortic diameter;
RAV – right atrial volume;
LAV – left atrial volume;
DM – diabetes mellitus;
IFG – impaired fasting glucose;
HbA1c – glycosylated hemoglobin HbA1c;
AH – arterial hypertension.

INTRODUCTION

Data from population studies show that diabetes correlates with various types of subclinical damage to target organs and leads to an increased risk of cardiovascular complications [1]. Early detection of diabetic heart disease should be a clinical priority because timely prevention plans or medical interventions can delay or avoid heart failure [2]. Prediabetes, a state of high risk for diabetes, is usually defined as a blood glucose concentration above normal but below the threshold for diabetes [3]. There is increasing evidence that prediabetes is also relevant for subclinical target organ damage compared to the standard glucose group [4]. However, the connection between early changes in the heart's and blood vessels' structure and function and prediabetes remains inconclusive [5, 6]. Cardiac remodeling is characterized by cardiac chamber enlargement and dysfunction closely associated with an increased risk of fatal cardiovascular events. In particular, structural and functional changes in the left heart are associated with diabetes and abnormal glucose homeostasis [7]. Left ventricular hypertrophy (LVH), a marker of cardiac damage, is a powerful independent predictor of cardiovascular morbidity and mortality. LVH was considered a complication of various metabolic disorders, even in the absence of hypertension [8]. Diabetes can be a stimulus for LVH by directly affecting the myocardium, while LVH can negatively affect the development of myocardial ischemia, arrhythmias, congestive heart failure, and sudden death [8, 9]. The left atrium (LA) is often structurally and functionally related to LV filling pressure. Increased LA suggests an essential association with the burden of cardiovascular disease risk and clinical outcomes in various populations [10]. Several experimental and clinical studies have shown that LA can be a sensitive indicator directly involved in the association with atrial fibrillation, stroke, and cardiovascular mortality from all causes [11]. Although LA is classically accepted as an expected outcome of left ventricular dysfunction in patients with diabetes, results regarding the utility of LV size in the diabetic population are conflicting [12]. In addition, there are limited data on the effect of long-term diabetes and prediabetes on LA.

Functional and structural microvascular damage occurs in patients with diabetes mellitus (DM) and often precedes the development of complications such as progressive diabetic retinopathy, renal failure, and overt cardiovascular diseases [13]. Early subclinical changes in micro- and macrocirculation can also be present in prediabetes. Identifying such changes may be crucial, as they may facilitate the early detection of vascular damage and prevent the complications associated with DM [14].

Arterial stiffness is a subclinical indicator of cardiovascular diseases (CVD) and an independent predictor of vascular dysfunction, which leads to changes in central hemodynamics [15]. Although evidence suggests that arterial stiffness increases in patients with type 2 diabetes and is strongly associated with its complications [16], knowledge about arterial stiffness in developing type 2 diabetes is limited [17, 18]. Recent evidence suggests that increased arterial stiffness may be evident before the onset of type 2 diabetes and among individuals with prediabetes. However, the findings remain inconclusive. It is noteworthy that abnormal glucose metabolism is a crucial factor contributing to the gradual increase in arterial stiffness from normal to prediabetes and to type 2 diabetes [19].

The purpose of the study was to evaluate the effect of prediabetes on cardiovascular remodeling in hypertensive patients with obesity.

MATERIALS AND METHODS

Clinical and anamnestic, anthropometric, biochemical, 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). Inclusion criteria: prediabetes (fasting hyperglycemia or impaired glucose tolerance); glomerular filtration rate (GFR) > 60 ml/min/1.73m2; normocreatininemia; absence of proteinuria (only microalbuminuria is allowed), left ventricular ejection fraction (LVEF) > 50 %; age 45–55 years. Patients with secondary AH, stage III AH grade 3, obesity grade III, LVEF < 50 %, 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.

The "IMAGIC Agile" ultrasound scanner, manufactured by "Kontron Medical" in France, was used to evaluate the morphofunctional properties of the heart and blood vessels.

The assessment included the volumes of the left and right atria (LAV and RAV), end-systolic and end-diastolic diameters of the left ventricle (LVESD and LVEDD), and diameters of the left atrium and aorta (LAD and AD).

Additionally, measurements were taken for the thickness of the posterior wall of the left ventricle in systole (TPWs) and diastole (TPWd) and the thickness of the interventricular septum in systole (TIVSs) and diastole (TIVSd).

The left ventricle's relative wall thickness (RWT) was calculated using the formula:

RWT = (TPWd + TIVSd) / LVEDD

The ejection fraction (EF) was calculated using 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 using the Dereveux method:

LVM = 1,04 x [(TIVSd + TPWd + LVEDD)3 – (LVEDD)3] – 13,6.

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

LVMI = LVM / S

The body surface area (S) was calculated using the Du Bois formula:

S = 0,007184 × H0.725 × W0.425 ,

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. [20, 21]. 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.

To determine the degree of carbohydrate metabolism disorders, fasting glycemia, impaired glucose tolerance, glycosylated haemoglobin, and a glucose tolerance test were evaluated. Impaired fasting glycemia was established at values of glucose concentration in venous plasma ≥ 6.1 and < 7.0 mmol/l, in whole capillary blood ≥ 5.6 and < 6.1 mmol/l, and glucose index < 7.8 mmol/l both in whole capillary blood and in venous plasma during the oral glucose tolerance test. Impaired glucose tolerance was diagnosed based on fasting glucose concentrations of < 6.1 mmol/l in whole capillary blood and < 7.0 mmol/l in venous plasma, and ≥ 7.8 < 11.1 mmol/l both in whole capillary blood and in venous plasma according to the oral glucose tolerance test. Insulin resistance was determined according to the HOMA model:

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

The data were analyzed using the statistical software SPSS 17 (IBM) and Microsoft Office Excel 2003. Results are presented as means ± standard deviation, with significance as $p < 0.05$ 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

Evaluation of the characteristics of cardiovascular remodeling indicators in hypertensive patients with obesity depending on the presence and absence of prediabetes showed that in the presence of prediabetes, significantly higher values of CIMT and CIMT bifurcation ($p = 0.027$ and $p = 0.012$, respectively), as well as substantially larger cPWV ($p = 0.022$), were noted. This is explained by the fact that violations of carbohydrate metabolism already at the stage of prediabetes contribute to the progression of vascular remodeling (Table 1).

Assessment of systolic function in obese hypertensive patients showed that patients with prediabetes had significantly larger left ventricular, left atrial, wall thickness, and LVMI, with no differences in LVEF, compared with nonprediabetic patients. Thus, the presence of prediabetes was associated with greater severity of LV hypertrophic changes (Table 1).

Assessment of the diastolic function of hypertensive patients showed that in the presence of prediabetes, there was a decrease in the speed of early LV filling ($p = 0.000$) and the ratio of the speeds of early and late filling of the LV ($p = 0.000$) in the absence of significant differences in the levels of the integral indicator of diastolic function (E/e') (Table 1).

A comparative assessment of the indicators of cardiovascular remodeling in hypertensive patients with obesity and prediabetes depending on the variant of prediabetes (fasting hyperglycemia and impaired glucose tolerance) did not show significant differences in any of the indicators (Table 2).

Table 1 – Indicators of cardiovascular remodeling in hypertensive obese patients depending on the presence and absence of prediabetes

Indicators	AH + obesity, normoglycemia	AH + obesity, prediabetes	p
	n=140	n=60	
CIMT [mm]	0.90 ± 0.08	0.93 ± 0.10	0.027
CIMT bifurcation [mm]	1.34 ± 0.15	1.40 ± 0.14	0.012
cPWV [m/s]	8.45 ± 1.13	8.83 ± 0.92	0.022
aPWV [m/s]	8.39 ± 1.01	8.70 ± 1.12	0.052
EDVD (%)	6.95 ± 1.12	6.82 ± 1.24	0.467
TIVSd [cm]	1.16 ± 0.12	1.19 ± 0.12	0.100
TIVSs [cm]	1.45 ± 0.15	1.50 ± 0.13	0.010
TPWd [cm]	1.17 ± 0.13	1.21 ± 0.15	0.075
TPWs [cm]	1.59 ± 0.32	1.66 ± 0.40	0.145
LVEDD[cm]	4.86 ± 0.30	4.97 ± 0.42	0.035
LVESD[cm]	3.20 ± 0.23	3.27 ± 0.34	0.070
EDV [mL]	111.18 ± 16.46	117.67 ± 23.64	0.027
ESV [mL]	41.17 ± 7.55	43.91 ± 11.44	0.047
LVEF (%)	62.99 ± 3.45	62.99 ± 3.02	0.994
LVM [g]	256.90 ± 57.30	281.29 ± 84.18	0.018
LVMI [g/m^2]	121.38 ± 27.20	134.04 ± 38.08	0.008
RWT	0.48 ± 0.05	0.48 ± 0.04	0.590
LAD [mm]	38.18 ± 3.03	38.51 ± 3.64	0.499
AD [mm]	33.19 ± 1.67	32.70 ± 0.85	0.031
Mean pulmonary AP [mm Hg] by Kitabatake	16.2 ± 3.27	16.28 ± 3.11	0.989
RAV [mL]	39.79 ± 5.01	38.55 ± 4.13	0.094
LAV [mL]	51.23 ± 4.50	53.85 ± 5.48	0.001
e´[cm/s]	11.77 ± 2.17	10.83 ± 2.23	0.006
E [cm/s]	68.9 ± 9.97	62.39 ± 9.34	0.000
A [cm/s]	78.60 ± 11.53	78.83 ± 9.09	0.892
E/A	0.89 ± 0.15	0.80 ± 0.14	0.000
DT [s]	0.15 ± 0.10	0.15 ± 0.09	0.906
IVRT [s]	0.12 ± 0.03	0.12 ± 0.03	0.767
E/e´	5.99 ± 1.07	5.93 ± 1.25	0.725

Notes: *the difference between groups is significant at $p < 0.05$*

Thus, it was established that the presence of prediabetes in hypertensive patients with obesity is associated with greater severity of vascular remodeling and, to a lesser extent, with the cardiac remodeling in the absence of significant differences in various variants of prediabetes (fasting hyperglycemia and impaired glucose tolerance).

DISCUSSION

Prediabetes is an essential metabolic status because there is a high potential for future progression to diabetes. People with prediabetes have an increased risk of cardiovascular disease (CVD) and mortality. Endothelial and microvascular dysfunction is considered a critical step in the development and progression of CVD. Structural and functional changes in the microvascular bed have been consistently documented in patients with diabetes. However, such changes remain poorly understood in prediabetes but are currently attracting attention as markers of subclinical and future cardiovascular disease [4, 13]. A population-based study (Markus MRP et al.) demonstrated that higher glucose levels in the prediabetic range and insulin resistance could lead to increased arterial stiffness and concentric cardiac remodeling. At that time, the Multi-Ethnic Study of Atherosclerosis (MESA) showed that individuals with impaired fasting

glucose (IFG) had no significant difference in LVM compared with individuals with average fasting glucose. A recent analysis of the same cohort [22] demonstrated that subjects with IFG had a higher LVM score. In contrast, an analysis of the Framingham Heart Study [23] showed that the HOMA-IR index was inversely proportional to LVM. The study presented by our author team demonstrated that impaired carbohydrate metabolism in hypertensive and obese patients at the stage of prediabetes contributes to the progression of vascular remodeling and is associated with the severity of hypertrophic changes in the left ventricle. It should be noted that the presence of prediabetes was associated to a greater extent with vascular remodeling than with cardiac remodeling in the absence of significant differences for different variants of prediabetes.

Table 2 – Indicators of cardiovascular remodeling in hypertensive obese patients with various variants of prediabetes

Indicators	**AH + obesity, fasting hyperglycemia**	**AH + obesity, impaired glucose tolerance**	**p**
	n=30	n=30	
CIMT [mm]	0.93 ± 0.09	0.93 ± 0.12	0.862
CIMT bifurcation [mm]	1.40 ± 0.15	1.40 ± 0.13	0.856
cPWV [m/s]	8.75 ± 0.91	8.92 ± 0.94	0.480
aPWV [m/s]	8.81 ± 1.03	8.60 ± 1.21	0.467
EDVD (%)	6.99 ± 1.33	6.65 ± 1.14	0.285
TIVSd [cm]	1.19 ± 0.09	1.19 ± 0.15	0.992
TIVSs [cm]	1.50 ± 0.14	1.51 ± 0.12	0.922
TPWd [cm]	1.19 ± 0.11	1.22 ± 0.19	0.447
TPWs [cm]	1.63 ± 0.32	1.70 ± 0.46	0.495
LVEDD[cm]	4.95 ± 0.37	4.99 ± 0.46	0.725
LVESD[cm]	3.24 ± 0.29	3.31 ± 0.38	0.433
EDV [mL]	116.39 ± 21.02	118.96 ± 26.31	0.678
ESV [mL]	42.64 ± 9.82	45.18 ± 12.89	0.394
LVEF (%)	63.54 ± 3.10	62.44 ± 2.87	0.156
LVM [g]	274.09 ± 59.98	288.49 ± 103.50	0.512
LVMI [g/m^2]	131.92 ± 29.70	136.16 ± 45.37	0.670
RWT	0.48 ± 0.04	0.48 ± 0.04	0.903
LAD [mm]	38.64 ± 3.87	38.39 ± 3.46	0.795
AD [mm]	32.63 ± 0.87	32.76 ± 0.85	0.551
Mean pulmonary AP [mm Hg] by Kitabatake	16.14 ± 3.12	16.43 ± 3.14	0.718
RAV [mL]	38.77 ± 4.33	38.33 ± 3.97	0.683
LAV [mL]	54.84 ± 6.01	52.86 ± 4.78	0.162
e´[cm/s]	10.69 ± 2.29	10.98 ± 2.21	0.627
E [cm/s]	61.45 ± 10.33	63.33 ± 8.29	0.441
A [cm/s]	78.83 ± 8.68	78.83 ± 9.62	0.999
E/A	0.79 ± 0.14	0.81 ± 0.14	0.416
DT [s]	0.16 ± 0.12	0.14 ± 0.04	0.540
IVRT [s]	0.12 ± 0.02	0.12 ± 0.03	0.492
E/e´	5.90 ± 1.23	5.95 ± 1.29	0.877

Notes: the difference between groups is significant at $p < 0.05$

Despite the currently known research data, the connection between early structural and functional changes of the heart and blood vessels with prediabetes remains inconclusive [5, 6] and requires further research.

CONCLUSIONS

Violations of carbohydrate metabolism at the stage of prediabetes in hypertensive patients with obesity contribute to the progression of vascular remodeling. They are associated with the severity of hypertrophic changes in the left ventricle.

Evaluation of cardiovascular remodeling indicators in hypertensive patients with obesity and prediabetes depending on the variant of prediabetes (fasting hyperglycemia and impaired glucose tolerance) did not show significant differences in any of the indicators.

AUTHOR CONTRIBUTIONS

Valentyna H. Psarova, Doctor of Medical Sciences, MD, PhD, Professor of the Sumy State University: work concept and design, data collection and analysis, responsibility for statistical analysis, writing (not revising) sections of the manuscript, final approval of the article.

Maryna M. Kochuieva, Doctor of Medical Sciences, MD, PhD, Professor of the Shupyk National Healthcare University of Ukraine: work concept and design, writing (not revising) sections of the manuscript, data collection and analysis, critical review, final approval of the article.

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Nataliia M. Kyrychenko, MD, PhD, Associate Professor of the Sumy State University: data collection and analysis, writing (not revising) sections of the manuscript

Gennadii I. Kochuiev, MD, PhD, Associate Professor of the Kharkiv National Medical University: collection of data, writing (not revising) sections of the manuscript

Anastasiia L. Cherkashyna, Intern Doctor of the Sumy Regional Clinical Hospital: collection of data, writing (not revising) sections of the manuscript

Daria O. Ivanova, Student of the Sumy State University: collection of data, writing (not revising) sections of the manuscript.

FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Ceyhan K, Kadi H, Koç F, Çelik A, Öztürk A, Önalan O. Longitudinal left ventricular function in normotensive prediabetics: a tissue Doppler and strain/strain rate echocardiography study. *J Am Soc Echocardiogr.* 2012 Mar;25(3):349-356. https://doi.org/10.1016/j.echo.2011.11.018. Cited in: PubMed; PMID: 22177116.
2. Jorgensen PG, Jensen MT, Mogelvang R, Fritz-Hansen T, Galatius S, Biering-Sørensen T et al. Impact of type 2 diabetes and duration of type 2 diabetes on cardiac structure and function. *Int J Cardiol.* 2016 Oct;221:114-121. https://doi.org/10.1016/j.ijcard.2016.07.083. Cited in: PubMed; PMID: 27423078.
3. Echouffo-Tcheugui JB, Perreault L, Ji L, Dagogo-Jack S. Diagnosis and Management of Prediabetes: A Review. *JAMA.* 2023 Apr;329(14):1206-1216. https://doi:10.1001/jama.2023.4063. Cited in: PubMed; PMID: 37039787
4. Fang ZY, Schull-Meade R, Downey M, Prins J, Marwick TH. Determinants of subclinical diabetic heart disease. *Diabetologia.* 2005 Feb;48(2):394-402. https://doi:10.1007/s00125-004-1632-z.
5. Li T, Li G, Guo X, Li Z, Yang J, Sun Y. The influence of diabetes and prediabetes on left heart remodeling: A population-based study. *Journal of Diabetes and its Complications.* 2021 Feb;35(2):107771. https://doi:10.1016/j.jdiacomp.2020.107771.
6. Markus MRP, Rospleszcz S, Ittermann T, Baumeister SE, Schipf S, Siewert-Markus U et al. Glucose and insulin levels are associated with arterial stiffness and concentric remodeling of the heart. *Cardiovasc Diabetol.* 2019 Nov;18:145. https://doi:10.1186/s12933-019-0948-4. Cited in: PubMed; PMID: 31684945.
7. Zoppini G, Bonapace S, Bergamini C, Rossi A, Trombetta M, Lanzoni L et al. Evidence of left atrial remodeling and left ventricular diastolic dysfunction in type 2 diabetes mellitus with preserved systolic function. *Nutr Metab Cardiovasc Dis.* 2016 May;26(11): 1026-1032.

https://doi.org/10.1016/j.numecd.2016.05.004. Cited in: PubMed; PMID: 27352987.
8. Modin D, Møgelvang R, Jørgensen PG, Jensen MT, Seferovic JP, Biering-Sørensen T. Left ventricular concentric geometry predicts incident diabetes mellitus independent of established risk factors in the general population: the Copenhagen City Heart Study. *Cardiovasc Diabetol.* 2019 Mar;18(1):37. https://doi:10.1186/s12933-019-0842-0.
9. De Jong KA, Lopaschuk GD. Complex Energy Metabolic Changes in Heart Failure With Preserved Ejection Fraction and Heart Failure With Reduced Ejection Fraction. *Can J Cardiol.* 2017 Mar;33(7):860-871. https://doi:10.1016/j.cjca.2017.03.009.
10. Patel DA, Lavie CJ, Milani RV, Ventura HO. Left atrial volume index predictive of mortality independent of left ventricular geometry in a large clinical cohort with preserved ejection fraction. *Mayo Clin Proc.* 2011 Aug ;86(8):730-7. https://doi:10.4065/mcp.2010.0682. Cited in: PubMed; PMID: 21803955.
11. Khan MA, Yang EY, Zhan Ya, Judd RM, Chan W, Nabi F et al. Association of left atrial volume index and all-cause mortality in patients referred for routine cardiovascular magnetic resonance: a multicenter study. *J Cardiovasc Magn Reson.* 2019 Jan;21(1):4. https://doi.org/10.1186/s12968-018-0517-0. Cited in: PubMed; PMID: 30612579.
12. Salvatore T, Pafundi PC, Galiero R, Albanese G, Di Martino A, Caturano A et al. The Diabetic Cardiomyopathy: The Contributing Pathophysiological Mechanisms. *Front. Med.* 2021 June;8. https://doi.org/10.3389/fmed.2021.695792. Cited in: PubMed; PMID: 34277669.
13. Lamprou S, Koletsos N, Mintziori G, Anyfanti P, Trakatelli C, Kotsis V et al. Microvascular and Endothelial Dysfunction in Prediabetes. *Life.* 2023 Feb;13(3):644. https://doi.org/10.3390/life13030644.
14. Buysschaert M, Medina JL, Bergman M, Shah, A, Lonier J. Prediabetes and associated disorders. *Endocrine*. 2015 Mar;48(2):371-393. https://doi.org/10.1007/s12020-014-0436-2. Cited in: PubMed; PMID: 25294012.
15. Lyle AN, Raaz U. Killing Me Unsoftly: Causes and Mechanisms of Arterial Stiffness. *Arterioscler Thromb Vasc Biol*, 2017 Feb; 37(2):e1-e11. https://doi:10.1161/ATVBAHA.116.308563. Cited in: PubMed; PMID: 28122777.
16. Prenner SB, Chirinos JA. Arterial stiffness in diabetes mellitus. *Atherosclerosis*, 2015 Feb;238:370-379. https://doi:10.1016/j.atherosclerosis.2014.12.023.
17. Wang M, Huang J, Wu T, Qi L. Arterial Stiffness, Genetic Risk, and Type 2 Diabetes: A Prospective Cohort Study. *Diabetes Care*. 2022 Apr; 45(4):957-964. Cited in: PubMed; PMID: 35076696.
18. Patoulias D, Papadopoulos C, Stavropoulos K, Zografou I, Doumas M, Karagiannis A. Prognostic value of arterial stiffness measurements in cardiovascular disease, diabetes, and its complications: The potential role of sodium-glucose co-transporter-2 inhibitors. *J Clin Hypertens (Greenwich).* 2020 Apr;22:562-571. https://doi:10.1111/jch.13831. Cited in: PubMed; PMID: 32058679.
19. Cozma A, Sitar-Taut A, Orăşan O, Leucuta D, Alexescu T, Stan A et al. Determining Factors of Arterial Stiffness in Subjects with Metabolic Syndrome. *Metab Syndr Relat Disord*. 2018 Oct;16:490-496. https://doi:10.1089/met.2018.0057.
20. Ivanova OV, Rogoza AN, Balahonova TV, Soboleva GN, Atkov OYu, Karpov YuA. Opredelenie chuvstvitelnosti plechevoy arterii k napryazheniyu sdviga na endoteliy, kak metod otsenki sostoyaniya endoteliy zavisimoy vazodilatatsii s pomoschyu ultrazvuka vyisokogo razresheniya u bolnyih s arterialnoy gipertoniey [Sensitivity of endothelium of the brachial artery to shear stress-method to evaluate endothelial function in patients with hypertension]. Cardiology. 1998;(3):37-42. Russian.
21. Celermajer DS, Sorensen KE, Cooh VM, Spiegelhalte DJ, Miller OI, Sullivane ID et al. Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis. Lancet. 1992 Nov 7;340(8828):1111–5. https://doi.org/10.1016/0140-6736(92)93147-f. Cited in: PubMed; PMID: 1359209.
22. Shah RV, Abbasi SA, Heydari B, Rickers C, Jacobs DR Jr, Wang L, Kwong RY, Bluemke DA, Lima JA, Jerosch-Herold M. Insulin resistance, subclinical left ventricular remodeling, and the obesity paradox: MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol*. 2013 Apr;61(16):1698-706. https://doi:10.1016/j.jacc.2013.01.053. Cited in: PubMed; PMID: 23500236.
23. Velagaleti RS, Gona P, Chuang ML, Salton CJ, Fox CS, Blease SJ, Yeon SB, Manning WJ, O'Donnell CJ. Relations of insulin resistance and glycemic abnormalities to cardiovascular magnetic resonance measures of cardiac structure and function: the Framingham Heart Study. *Circ Cardiovasc Imaging*. 2010 Mar;3(3):257-63. https://doi:10.1161/CIRCIMAGING.109.911438. Cited in: PubMed; PMID: 20208015.

Received 31.07.2024
Accepted 22.10.2024

Одержано 31.07.2024
Затверджено до друку 22.10.2024