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Research Article

Influence of Carbohydrate Metabolism Compensation on the Heart Failure Development in Patients with Arterial Hypertension and Obesity

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Abstract

Background: Arterial hypertension, especially in combination with diabetes mellitus, which significantly increases the risk of micro- and macrovascular complications, with hypercholesterinemia, coronary artery disease, congestive heart failure, is the main risk factor for the premature development of atherosclerosis, which generally leads to the development of severe long-term cardiovascular adverse effects. The aim of this study was to determine the state of carbohydrate metabolism compensation and its possible impact on the development of congestive heart failure, its progression in patients with hypertension and obesity.

Materials and Methods: A comprehensive examination of 34 patients with arterial hypertension and obesity and type II diabetes mellitus, 32 patients with AH and type II DM, 36 patients with AH and obesity, and 30 patients with AH has been carried out. The Control group consisted of 20 healthy individuals without arterial hypertension and carbohydrate metabolism disorders.

Results: The examined groups of patients did not differ significantly in age, gender, history of myocardial infarction. In the Control group, the subjects were younger and did not suffer from hypertension, type 2 diabetes mellitus, and obesity. At the same time, very pronounced changes in carbohydrate metabolism in patients with different variants of comorbid pathology have been identified. Comparative analysis of carbohydrate metabolism in the studied groups of patients showed that comorbidity of hypertension, type 2 diabetes mellitus, and obesity leads to significant shifts. Metabolic disorders in type 2 diabetes mellitus have been shown to lead to impaired energy metabolism in cardiomyocytes due to insulin resistance.

Conclusions: The study demonstrates that arterial hypertension, type 2 diabetes mellitus, obesity, hyperinsulinemia, and insulin resistance have a general pathogenetic effect on its progression in patients with cardiovascular pathology, lead to the summation and potentiation of cardiovascular risk.

Keywords: Arterial Hypertension; Type 2 Diabetes Mellitus; Obesity; Congestive Heart Failure

Introduction

It is now well known that arterial hypertension (AH), especially with comorbid diabetes mellitus (DM), which significantly increases the risk of micro- and macrovascular complications, with hypercholesterinemia, coronary artery disease (CAD), congestive heart failure (CHF), is the main risk factor for the premature development of atherosclerosis, which generally leads to the development of severe long-term cardiovascular adverse effects [1].

Myocardial damage is especially severe due to tissue ischemia, namely CAD, which develops in the presence of carbohydrate metabolism disorders, from prediabetes to DM, since many negative factors that are closely related to each other join these conditions [2–5].

This primarily applies to the insulin resistance syndrome (IRS), which includes chronic low-intensity inflammation, AH, dyslipidemia, and obesity. It should also be taken into account that it is the violation of carbohydrate metabolism that contributes to the coronary atherosclerosis development and realizes its negative impact on the CHF development due to the CAD progression [6-10].

Given the significant number of studies devoted to the study of the impact of carbohydrate metabolism disorders on the CHF development, as well as taking into account the comorbidity of diseases that occur in such patients, some unresolved issues remain in these issues.

Therefore, the purpose of this study was to determine the state of carbohydrate metabolism compensation and its possible impact on the development of congestive heart failure, its progression in patients with arterial hypertension and obesity.

Materials and Methods

A comprehensive examination of 34 patients with AH and obesity and type II DM, 32 patients with AH and type II DM, 36 patients with AH and obesity, and 30 patients with AH has been carried out. The Control group consisted of 20 practically healthy individuals without AH and carbohydrate metabolism disorders.

The study has been carried out in compliance with the main bioethical provisions of the Convention for the Protection of Human Rights and Dignity of the Human Being with regard to the Application of Biology and Medicine by the Council of Europe (dated April 4, 1997), the World Medical Association (WMA) Declaration of Helsinki Ethical Principles For Medical Research Involving Human Subjects (1964-2008), as well as the Order of the Ministry of Health of Ukraine No. 690 dated September 23, 2009.

All patients signed informed consent to participate in the study.

Exclusion criteria were as follows: acute myocardial infarction (MI) or unstable angina pectoris < 30 days prior to enrollment in the study, decreased left ventricular ejection fraction < 40%; hemodynamically significant heart defects; rheumatism and other systemic connective tissue diseases; acute and decompensated chronic diseases of internal organs; severe renal impairment (glomerular filtrate rate (GFR) < 30 ml/min/1.73m^2); hepatic decompensation; obstructive pulmonary disease; oncological diseases and other diseases with a poor prognosis; type I DM or achrestic diabetes; blood triglyceride (TG) level ≥ 4.5mmol/l, HbA1c level ≥ 11.0%.

The level of HbA1c (%) in the blood was determined by the photometric ion-exchange method using test systems from Human GmbH (Germany). In order to determine the content of insulin in the blood serum, an enzyme-linked immunosorbent assay was applied, using the Insulin ELISA kit (DRG Instruments GmbH, Germany). Fasting blood glucose values were determined by the glucose oxidase method using a Humalyzer 2000 biochemical analyzer (Germany).

Body mass index (BMI) was calculated using the Quetelet formula

BMI = weight (kg)/height^2 (m^2).

Statistical data processing was carried out, using Microsoft Excel spreadsheets and Statistica 6.0 software (Stat Soft Inc, USA, free version). With a normal distribution, quantitative data were presented as the arithmetic mean and standard deviation (M ± σ).

In order to assess the statistical significance of differences between groups, the Student's t-test (with a normal distribution) and the Mann-Whitney test with continuity correction were used; in order to determine the nature of the strength of the relationship between the parameters, the Spearman rank correlation coefficient was used.

Results and Discussion

The examined groups of patients did not differ significantly in age, gender, and the presence of MI in the past medical history (Table 1). In the Control group, compared with the patients of the study groups, the subjects were younger and did not suffer from AH, type II DM, and obesity.

Indicator	Control, n = 20 (1)	AH + OB + type II DM, n = 34 (2)	AH + type II DM, n = 32 (3)	AH + OB, n = 36 (4)	AH, n = 30 (5)
Age, year, (M ± σ)	56.0 ± 1.29	64.38 ± 1.08* p_{1-2}<0.001	64.87 ± 1.19* p_{1-3}<0.001	63.47 ± 1.28* p_{1-4}<0,001	62.98 ± 1.91* p_{1-5}<0.01
Gender, w/m, n (%)	12/8 (60%/40%)	20/14 (59%/41%)	19/13 (59%/41%)	21/15 (58%/42%)	18/12 (60%/40%)
MI in past medical history, n (%)	0 (0%)	20 (58.82%)	19 (59.30%)	21 (58.33%)	18 (60%)
AH, n (%)	0 (0%)	34 (100%)	32 (100%)	36 (100%)	30 (100%)
BMI, kg/m^2, (M ± σ)	23.21 ± 3.14	33.64 ± 2.52* p_{1-2}<0.05	30.24 ± 2.36	32.0 ± 2.44* p_{1-4}<0.05	28.81 ± 3.5

Table 1: Clinical characteristics of the examined persons.

Note: *Statistically significant differences between the indicators are shown.

At the same time, very distinct changes in the indicators of carbohydrate metabolism have been determined in patients with different variants of comorbid pathology (Table 2).

Thus, blood glucose levels were probably higher in patients with AH, type II DM. and obesity by 39.72% and 44.5%, respectively, compared with patients with AH and controls ($p < 0.05$; $p < 0.001$); insulin level - by 72.74% and 74.54%, respectively ($p < 0.05$; $p < 0.001$); HOMA-IR index - by 87.16% and 89%, respectively ($p < 0.05$; $p < 0.001$).

A comparative analysis of carbohydrate metabolism in all groups of patients (see Table 2) has proved that the comorbidity of AH, type II DM, and obesity leads to significant shifts in all studied parameters of carbohydrate metabolism.

Thus, the glucose level in patients of Group 2 was 2.81% higher compared to patients of Group 3; 18.6% higher compared with Group 4, and 44.5% higher than those in the Control group ($p < 0.05$; $p < 0.001$).

The HbA1c level in comorbid patients of Group 2 was almost 11% higher compared with persons of Group 3 and by 40.2% compared with Group 5.

Also, other indicators of carbohydrate metabolism differed significantly between the study groups and were higher in patients with AH, type II DM, and obesity.

The severity of changes in carbohydrate metabolism depending on the severity of CHF has been analyzed and more significant shifts in the levels of HbA1c, insulin, and the HOMA-IR index against the background of an increase in the severity of CHF have been proved (Table 3).

Thus, the obtained results indicate that metabolic disorders in type II DM lead to disorders of energy metabolism in cardiomyocytes caused by insulin resistance.

The study demonstrates that AH, type II DM, obesity, hyperinsulinemia, and insulin resistance have a general pathogenetic effect on its progression in patients with cardiovascular pathology, lead

Indicator	Control, n = 20 (1)	AH + OB + type II DM, n = 34 (2)	AH + type II DM, n = 32 (3)	AH + OB, n = 36 (4)	AH, n = 30 (5)
HbA1c, %	4.68 ± 0.49* p_{1-2} < 0.001 p_{1-3} < 0.001	8.38 ± 0.29* p_{2-3} < 0.05 p_{2-4} < 0.001 p_{2-5} < 0.001	7.46 ± 0.34* p_{3-4} < 0.001 p_{3-5} < 0.001	5.27 ± 0.51	5.01 ± 0.13
Blood glucose, mmol/l	4.14 ± 0.14* p_{1-2} < 0.001 p_{1-3} < 0.001 p_{1-4} < 0.001 p_{1-5} < 0.05	7.46 ± 0.46* p_{2-4} < 0.05 p_{2-5} < 0.001	7.25 ± 0.35* p_{3-4} < 0.05 p_{3-5} < 0.01	6.07 ± 0.51* p_{4-5} < 0.01	4.49 ± 0.08
Insulin, mcU/ml	7.97 ± 0.34* p_{1-2} < 0.001 p_{1-3} < 0.001 p_{1-4} < 0.001 p_{1-5} < 0.05	31.81 ± 0.86* p_{2-3} < 0.05 p_{2-4} < 0.001 p_{2-5} < 0.001	29.34 ± 0.51* p_{3-4} < 0.001 p_{3-5} < 0.001	13.91 ± 0.43* p_{4-5} < 0.001	8.82 ± 0.25
HOMA-IR	1.47 ± 0.42* p_{1-2} < 0.001 p_{1-3} < 0.001 p_{1-4} < 0.01 p_{1-5} < 0.05	13.55 ± 0.67* p_{2-3} < 0.001 p_{2-4} < 0.001 p_{2-5} < 0.001	9.45 ± 0.67* p_{3-4} < 0.001 p_{3-5} < 0.001	3.78 ± 0.58	2.71 ± 0.31

Table 2: Indicators of carbohydrate metabolism in patients with comorbid pathology.

Note: *Statistically significant differences between the indicators are shown.

Indicator	CHF I FC	CHF II FC	CHF III FC
HOMA-IR	7.13 ± 0.36* $p_{1-2} < 0.001$ $p_{1-3} < 0.0001$	9.44 ± 0.43* $p_{2-3} < 0.001$	11.22 ± 0.5
HbA1c, %	9.37 ± 0.53* $p_{1-2} < 0.001$ $p_{1-3} < 0.001$	11.33 ± 0.25* $p_{2-3} < 0.001$	13.14 ± 0.48
Blood glucose, mmol/l	46.89 ± 0.31* $p_{1-2} < 0.001$ $p_{1-3} < 0.001$	7.78 ± 0.27* $p_{2-3} < 0.001$	8.88 ± 0.43
Insulin, mcU/ml	22.57 ± 1.34* $p_{1-2} < 0.001$ $p_{1-3} < 0.001$	26.57 ± 1.19* $p_{2-3} < 0.001$	30.24 ± 1.26

Table 3: Indicators of carbohydrate metabolism in the examined patients depending on the CHF severity.

Note: *Statistically significant differences between the indicators are shown.

to the summation and potentiation of cardiovascular risk, which was also reflected in the works carried out by other researchers [11-15].

Conclusions

- In patients with arterial hypertension in combination with type 2 diabetes mellitus and obesity, a significant increase in the levels of glycated hemoglobin, insulin, and the HOMA-IR insulin resistance index has been found not only in comparison with persons in the Control group, but also with patients with arterial hypertension and diabetes mellitus without obesity.
- In patients with myocardial infarction in the past, no significant changes in carbohydrate metabolism have been defined in comparison with persons without myocardial infarction in past medical history.
- The deterioration of carbohydrate metabolism in patients with comorbid pathology occurred simultaneously with an increase in the functional class of congestive heart failure.

Bibliography

1. Chichareon P., *et al*. *Atherosclerosis* 295 (2020): 45-53.
2. Bancks MP., *et al*. *Diabetes Care* 42.3 (2019): 457-465.
3. Mak K-H., *et al*. *European Journal of Preventive Cardiology* (2021): zwab011.
4. Gholap NN., *et al*. *Diabetes, Obesity and Metabolism* 19.3 (2017): 364-374.
5. Cepик CA., *et al*. *Problems of Endocrine Pathology* 78.4 (2021): 57-64.
6. Gorb YuG., *et al*. *UZHMS* 4.1 (2019): 8-20.
7. Dal Canto E., *et al*. *European Journal of Preventive Cardiology* 26.2 (2019): 25-32.
8. Thomas MC. *Current Cardiology Reviews* 12.3 (2016): 249-255.
9. Arnett DK., *et al*. *Journal of the American College of Cardiology* 74.10 (2019): 1376-1414.
10. Li S, Nemeth I, Donnelly L, et al. *Diabetes Care* 43.2 (2020): 426-432.
11. Ormazabal V., *et al*. *Cardiovascular Diabetology* 17.1 (2018): 122.
12. Zhou D., *et al*. *Scientific Reports* 8.1 (2018): 9418.
13. Pashentseva AV., *et al*. *Obesity and Metabolism* 14.2 (2017): 9-17.
14. Sokolova LA., *et al*. *Arterial. Hypertense* 2017; 23.4 (2017): 294-302.
15. Hill MA., *et al*. *Metabolism* 119 (2021): 154766.