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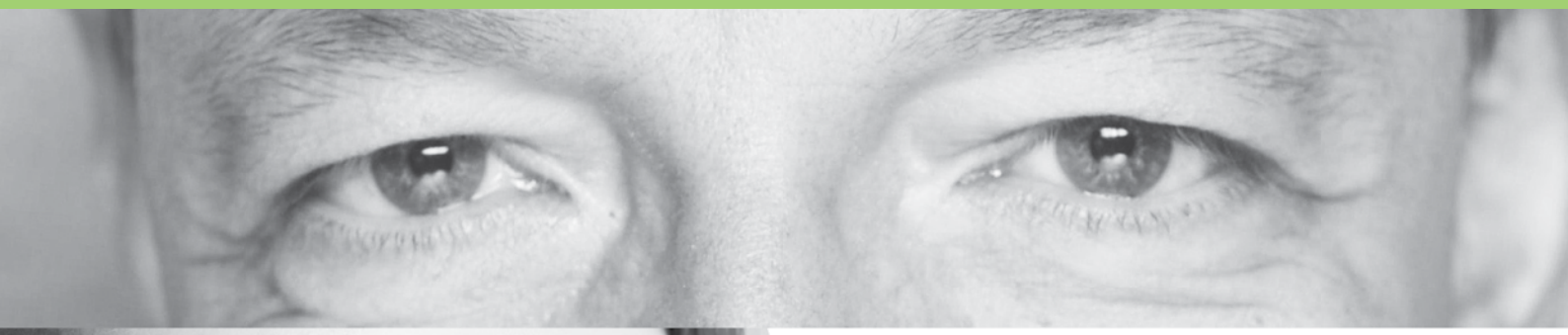

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Cardiometabolic determinants of elevated NT-proBNP concentrations in patients with type 2 diabetes mellitus and arterial hypertension (literature review and original data)

Dunaieva IP, Kravchun NO, Misiura KV, Tykha IA, Doroshenko OM, Yakovenko OL

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Abstract

The combination of arterial hypertension (AH) and type 2 diabetes mellitus (T2DM) is associated with a high risk of cardiac remodeling, left ventricular diastolic dysfunction, and the development of heart failure. The N-terminal fragment of the prohormone of brain natriuretic peptide (NT-proBNP) is considered a sensitive biomarker of myocardial stress, increased left ventricular filling pressure, and early structural and functional cardiac changes. However, the cardiometabolic factors determining elevated NT-proBNP concentrations in patients with combined AH and T2DM remain insufficiently studied.

Aim *To determine the cardiometabolic determinants of elevated NT-proBNP concentrations in patients with type 2 diabetes mellitus and arterial hypertension.*

Materials and Methods *The study included 136 patients with AH, divided into two groups: the first group comprised patients with isolated AH (n = 64), and the second group comprised patients with AH combined with T2DM (n = 72). The control group consisted of 20 apparently healthy individuals. All participants underwent a comprehensive clinical, laboratory, and instrumental examination, including assessment of lipid profile parameters, HbA1c, insulin, leptin, cystatin C (Cys C), NGAL, and NT-proBNP by enzyme-linked immunosorbent assay; evaluation of lymphocyte subpopulations; assessment of renal function parameters; and echocardiographic evaluation. To determine the factors associated with elevated NT-proBNP concentrations, univariate and multivariate logistic regression analyses were performed, with odds ratios and 95% confidence intervals calculated, and the prognostic performance of the models assessed using ROC analysis.*

Results *Patients with combined AH and T2DM had a more unfavourable cardiometabolic profile than patients with isolated AH. This was manifested by higher body mass index, total cholesterol, triglycerides, and atherogenic coefficient, increased leptin levels, activation of the cellular immune system, and more pronounced structural and functional cardiac remodelling. In the AH + T2DM group, increased left ventricular myocardial mass, left ventricular mass index, cardiac chamber dimensions, reduced left ventricular ejection fraction, and impaired diastolic function parameters were observed. NT-proBNP concentrations in patients with AH + T2DM tended to be higher than in the isolated AH group; however, the between-group difference did not reach statistical significance (p = 0.0791).*

According to multivariate logistic regression analysis, in patients with isolated AH, independent predictors of elevated NT-proBNP concentrations were aortic size and left ventricular myocardial mass; the model's prognostic performance was good (AUC = 0.868). In patients with combined AH and T2DM, the independent cardiometabolic determinants of elevated NT-proBNP concentrations were isovolumic relaxation time (IVRT), early diastolic filling velocity (VE), left ventricular mass index indexed to height, insulin concentration, and systolic blood pressure. The multivariate model in the AH + T2DM group also demonstrated good prognostic performance (AUC=0.803). Conclusion Elevated NT-proBNP concentrations in patients with AH and T2DM are formed under the influence of a complex of hemodynamic, metabolic, and structural-functional factors. In isolated AH, cardiac remodelling parameters are of primary importance, whereas in combined AH and T2DM, the spectrum of determinants expands to include indicators of diastolic dysfunction, left ventricular hypertrophy, insulin homeostasis, and systolic blood pressure. NT-proBNP may be considered an integral biomarker of early cardiac remodelling, subclinical diastolic dysfunction, and cardiometabolic risk stratification in patients with comorbid AH and T2DM.

Keywords: *arterial hypertension, type 2 diabetes mellitus, NT-proBNP, cardiometabolic risk, diastolic dysfunction, insulin*

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Introduction

Type 2 diabetes mellitus (T2DM) is one of the key factors in the development of cardiovascular complications, in particular diastolic dysfunction, left ventricular hypertrophy, and chronic heart failure. The combination of T2DM and arterial hypertension (AH) significantly increases the risk of cardiac remodeling, mediated by insulin resistance, activation of the sympathoadrenal system, chronic low-grade inflammation, endothelial dysfunction, and impairment of the adipokine profile. It has been established that patients with T2DM develop specific structural and functional myocardial changes, known as diabetic cardiomyopathy, characterized by left ventricular hypertrophy, increased myocardial stiffness, and impaired myocardial relaxation.

The heart is not considered a classical endocrine organ; however, it can perform an endocrine function by synthesizing and secreting natriuretic peptides, which play an important role in regulating water-electrolyte balance, vascular tone, and blood pressure [1].

In view of these pathophysiological changes and the described mechanisms of diabetic cardiomyopathy, natriuretic peptides, as biomarkers reflecting myocardial remodeling and hemodynamic load, are of particular interest [2].

At present, contemporary approaches to the management of patients with AH also involve the active use of biomarkers for early detection of target-organ damage and cardiovascular risk stratification, especially in patients with concomitant T2DM [3].

The family of natriuretic peptides includes atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), the N-terminal fragment of the precursor of brain natriuretic peptide (NT-proBNP), and C-type natriuretic peptide (CNP), which play an important role in the regulation of water-salt balance, vascular tone, and hemodynamics [4].

Natriuretic peptides, particularly NT-proBNP, play an important role in diagnosing subclinical heart failure (HF) and assessing myocardial stress. Elevated NT-proBNP concentrations reflect increased left ventricular (LV) filling pressure, impaired diastolic function, and myocardial remodelling. It has been shown that in patients with T2DM, increased NT-proBNP may be observed even in the absence of clinical manifestations of HF and may be associated with increased cardiovascular risk. The results of prospective studies demonstrate that elevated NT-proBNP concentrations in patients with T2DM are associated with a higher risk of HF, atrial fibrillation, and cardiovascular mortality [5–7].

According to population-based studies, NT-proBNP concentrations in patients with T2DM correlate with age, diabetes duration, body mass index (BMI), glycemic control, and blood pressure (BP). At the same time, some authors report an inverse association between NT-proBNP and obesity, which is explained by enhanced clearance of natriuretic peptides in adipose tissue and reduced secretion under conditions of insulin resistance.

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Analysis of the literature indicates that elevated NT-proBNP concentrations are associated with LV hypertrophy, increased left ventricular mass index (LVMI), impaired diastolic filling, obesity, insulin resistance, and adipokine dysfunction. Associations of NT-proBNP with insulin concentration, BMI, leptin, and indicators of cardiorenal interactions have also been demonstrated. A number of studies have established associations between NT-proBNP and glomerular filtration rate, cystatin C (Cys C) levels, and markers of tubular kidney injury [8–11].

It has been established that in patients with combined T2DM and AH, increased NT-proBNP is associated with more pronounced LV remodeling, increased left ventricular myocardial mass (LVM), impaired diastolic function, and increased myocardial stiffness. At the same time, indices of diastolic function, particularly early diastolic filling velocity (VE), the ratio of early to late diastolic filling velocities (VE/VA), and isovolumic relaxation time (IVRT), may act as independent predictors of increased NT-proBNP [12, 13].

Particular attention has recently been paid to the role of metabolic factors in the formation of elevated NT-proBNP concentrations. It has been shown that hyperinsulinemia and insulin resistance may contribute to myocardial remodelling, the development of diastolic dysfunction, and increased LV filling pressure. In addition, an association has been established between NT-proBNP and adipokines, particularly leptin, which regulates sympathetic activity, BP, and myocardial hypertrophy [14–18].

At the same time, research findings concerning the cardiometabolic determinants of elevated NT-proBNP concentrations in patients with combined T2DM and AH remain inconsistent. The role of diastolic function parameters, insulin, LVMI, and metabolic factors in the development of elevated NT-proBNP concentrations in this patient group has not been adequately studied. This substantiates the need for further studies that comprehensively assess clinical, metabolic, and structural-functional cardiac parameters.

Accordingly, the aim of the study was to determine the cardiometabolic determinants of elevated NT-proBNP concentrations in patients with T2DM and AH.

Materials and Methods

The study was conducted in accordance with the ethical principles and legal standards set forth in the Statute of the Ukrainian Association of Bioethics, as well as international standards of Good Clinical Practice (GCP, 1992) and Good Laboratory Practice (GLP, 2002). The provisions of the Declaration of Helsinki on ethical principles for medical research involving human subjects, as well as the Council of Europe Convention on Human Rights and Biomedicine, were observed. The study protocol was approved by the Commission on Ethics and Bioethics of Kharkiv National Medical University. All patients provided voluntary informed consent to participate in this study.

The study included 136 patients with AH. All examined participants were divided into two clinical groups. The first group comprised 64 patients with isolated AH (30 men and 34 women), with a mean age of 55.8 ± 8.31 years. The second group included 72 patients with combined AH and T2DM (34 men and 38 women), with a mean age of 51.98 ± 6.44 years. The control group consisted of 20 individuals who were practically healthy and without signs of cardiometabolic pathology.

The exclusion criteria were acute infectious and autoimmune diseases, oncological pathology, chronic kidney disease stage ≥3b (glomerular filtration rate <35 mL/min/1.73 m^2), symptomatic AH, severe T2DM, type 1 diabetes mellitus, pregnancy, the presence of any type of dependence, and acute inflammatory conditions.

All study participants underwent comprehensive clinical, laboratory, and instrumental examination. Biochemical testing included determination of blood lipid profile parameters: total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and triglycerides (TG). Renal functional status was assessed by creatinine and urea concentrations and by estimated glomerular filtration rate (eGFR), determined using standard methods.

To assess carbohydrate metabolism, insulin and glycated haemoglobin (HbA1c) concentrations were measured by enzyme-linked immunosorbent assay (ELISA). Concentrations of NT-proBNP,

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leptin, Cys C, and neutrophil gelatinase-associated lipocalin (NGAL) were also determined by ELISA using certified commercial kits, according to the manufacturers' instructions.

To assess the functional status of the immune system, the composition of peripheral blood lymphocyte subpopulations was investigated. The analysis was performed by immunoperoxidase staining with monoclonal antibodies to lymphocyte surface markers, with determination of their relative abundance. The percentage of total T lymphocytes ($CD3^+$), T helper cells ($CD4^+$), T cytotoxic/suppressor cells ($CD8^+$), and T active lymphocytes ($CD25^+$) was determined. In addition, the immunoregulatory index ($CD4^+/CD8^+$, Th/Ts) was calculated.

Echocardiographic examination was performed using an Ultima PA ultrasound system (Radmir, Ukraine) with a sector phased-array transducer at 2–3 MHz, in accordance with the recommendations of the American Society of Echocardiography. The dimensions of the cardiac chambers (aorta, left and right atria, right and left ventricles), left ventricular end-diastolic dimension (LVEDD) and end-systolic dimension (LVESD), interventricular septal thickness (IVST), left ventricular posterior wall thickness (LVPWT), LVM, LVMI, relative wall thickness (RWT), and left ventricular ejection fraction (LVEF) were determined. Diastolic function was assessed by the parameters of early diastolic filling velocity (VE), late diastolic filling velocity (VA), their ratio (VE/VA), and IVRT.

Statistical analysis of the obtained results was performed using Statistica 14.0 software (StatSoft Inc., USA) and Microsoft Office Excel 2013. The normality of distribution of quantitative variables was assessed using the Shapiro–Wilk test. For the normal distribution, data were presented as the mean ± standard deviation (M ± SD). To evaluate factors associated with elevated NT-proBNP concentrations, univariate logistic regression was used to determine odds ratios (ORs) and 95 % confidence intervals (CIs). Variables that demonstrated a statistically significant association in univariate analysis were included in the multivariate logistic regression model to determine independent cardiometabolic determinants of elevated NT-proBNP concentrations. The prognostic value of the models was assessed using ROC analysis with calculation of the area under the curve (AUC). Differences were considered statistically significant at $p < 0.05$.

Results

Comparative analysis of clinical and metabolic parameters in patients with AH and in those with AH combined with T2DM showed the presence of more pronounced metabolic disorders in patients with comorbid pathology (Table 1).

In patients with AH with T2DM, BMI was higher than in the isolated AH group (32.55 ± 6.00 vs 27.86 ± 2.30 kg/m^2; $p < 0.001$). At the same time, no significant differences were found between the groups in SBP and DBP levels ($p > 0.05$). HbA1c values also did not differ significantly between the groups.

Analysis of the blood lipid spectrum showed more pronounced atherogenic changes in patients with combined AH and T2DM. In this group, increases in TC (6.29 ± 1.65 vs. 5.49 ± 1.30 mmol/L; $p = 0.0047$), TG (3.13 ± 2.17 vs. 1.86 ± 0.80 mmol/L; $p = 0.0002$), and the atherogenic coefficient (4.94 ± 2.74 vs. 3.64 ± 1.44; $p = 0.0054$) were observed. No significant differences were detected between the groups in HDL-C, LDL-C, or VLDL-C.

Investigation of immune system parameters demonstrated considerable differences between the groups. In patients with AH combined with T2DM, increased levels of $CD3^+$, $CD4^+$, $CD8^+$, and $CD25^+$ lymphocytes, as well as a marked increase in the $CD4^+/CD8^+$ immunoregulatory index ($p < 0.001$), were observed, suggesting activation of the cellular immune system in the comorbid course of the disease.

Assessment of renal functional parameters showed that creatinine and urea concentrations did not differ significantly between the groups. At the same time, patients with AH and T2DM had a decrease in Cys C concentration ($p = 0.0293$). Patients with comorbid pathology also showed an increase in leptin ($p = 0.0486$), whereas NGAL and insulin levels did not differ significantly between the groups. NT-proBNP concentrations tended to be higher in patients with combined AH and T2DM; however, the difference did not reach statistical significance ($p = 0.0791$).

Table 1. *Clinical-metabolic and cardiac parameters in patients with AH and in patients with AH combined with T2DM*

Parameters	AH group	AH+T2DM group	p-value
Age, years	55,8 ± 8,31	51,98 ± 6,44	0,0030
BMI, kg/m^2	27,86 ± 2,30	32,55± 6,00	0,0000
SBP, mmHg	143,09 ± 11,24	140,02 ± 22,09	0,3092
DBP, mmHg	87,42 ± 8,94	84,94 ± 13,78	0,2113
HbA1c, %	6,97 ± 1,58	7,33 ± 1,66	0,1773
TC, mmol/L	5,49 ± 1,30	6,29 ± 1,65	0,0047
TG, mmol/L	1,86 ± 0,80	3,13 ± 2,17	0,0002
HDL-C, mmol/L	1,18 ± 0,31	1,13 ± 0,26	0,3868
LDL-C, mmol/L	3,44 ± 1,29	3,69 ± 1,07	0,3223
VLDL-C, mmol/L	0,96 ± 0,68	1,07 ± 0,43	0,3463
AC	3,64 ± 1,44	4,94 ± 2,74	0,0054
CIC, units	96,47 ± 9,74	31,08 ± 8,55	0,0001
CD3$^+$, %	65,29 ± 3,76	149,83 ± 21,66	0,0001
CD4$^+$, %	38,76 ± 3,07	53,15 ± 4,08	0,0001
CD8$^+$, %	21,77 ± 1,91	29,84 ± 3,57	0,0001
CD25$^+$, %	7,18 ± 1,36	24,08 ± 3,89	0,0001
Immunoregulatory index (CD4$^+$/CD8$^+$)	1,84 ± 0,28	11,40 ± 2,72	0,0001
Blood urea, mmol/L	5,60 ± 1,40	6,08 ± 1,65	0,0904
Creatinine, mmol/L	96,39 ± 16,26	99,10 ± 23,30	0,4745
Cys C, mg/L	150,21 ± 33,51	131,10 ± 26,70	0,0293
Leptin, ng/mL	21,70 ± 11,16	25,03 ± 9,18	0,0486
NGAL, pg/mL	17,67 ± 4,52	17,03 ± 4,37	0,3827
NT-proBNP, pg/mL	413,39 ± 130,84	453,49 ± 141,46	0,0791
Insulin, μIU/mL	21,11±14,98	22,48 ± 12,62	0,5475
Aorta, cm	3,28 ± 0,25	3,79 ± 0,09	0,0001
LA, cm	3,61 ± 0,27	4,47 ± 0,12	0,0001
RA, cm	3,84 ± 0,24	4,23±0,11	0,0001
LVEDD, cm	4,53 ± 0,43	5,90 ±0 ,09	0,0001
LVESD, cm	3,33±0,46	4,19±0,12	0,0001
RV, cm	1,73±0,08	1,35±0,06	0,0001
IVST, cm	1,25±0,07	1,32±0,07	0,0001
LVPWT, cm	1,26±0,04	1,20±0,05	0,0001
LVM, g	205,52±30,91	261,72±17,83	0,0001
LVMI, g/m^2	121,87±16,74	136,08±9,00	0,0001
LVRWT	0,53±0,05	0,49±0,03	0,0001
LVEF, %	57,47±3,67	48,53±3,14	0,0001
VE, cm/s	62,49±8,33	64,01±7,92	0,2734
VA, cm/s	72,98±8,33	83,22±6,59	0,0001
VE/VA	0,86±0,13	0,79±0,12	0,0012
IVRT, ms	105,40±10,97	117,04±11,30	0,0001

Abbreviations: AH — arterial hypertension, T2DM — type 2 diabetes mellitus, BMI — body mass index, SBP — systolic blood pressure, DBP — diastolic blood pressure, HbA1c — glycated haemoglobin, TC — total cholesterol, TG — triglycerides, HDL-C — high-density lipoprotein cholesterol, LDL-C — low-density lipoprotein cholesterol, VLDL-C — very-low-density lipoprotein cholesterol, AC — atherogenic coefficient, CIC — circulating immune complexes, CD3$^+$ — total T lymphocytes, CD4$^+$ — T helper cells, CD8$^+$ — T cytotoxic/suppressor cells, CD25$^+$ — T active lymphocytes, CD4$^+$/CD8$^+$— immunoregulatory index, Cys C — cystatin C, NGAL — neutrophil gelatinase-associated lipocalin, NT-proBNP — the N-terminal fragment of the precursor of brain natriuretic peptide, LA — left atrium, RA — right atrium, LVEDD — left ventricular end-diastolic dimension, LVESD — left ventricular end-systolic dimension, RV — right ventricle, IVST— interventricular septal thickness, LVPWT — left ventricular posterior wall thickness, LVM— ventricular myocardial mass, LVMI — left ventricular mass index, LVRWT — left ventricular posterior wall thickness, LVEF — left ventricular ejection fraction, VE —diastolic filling velocity, VA — late diastolic filling velocity, IVRT— isovolumic relaxation time.

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Analysis of echocardiographic parameters showed more pronounced cardiac remodelling in patients with combined AH and T2DM. In this group, enlargement of the aorta, left atrium, right atrium, and left ventricle was detected (p <0.001). Increases in LVEDD and LVESD were also observed.

In patients with AH and T2DM, increases in LVM (261.72 ± 17.83 vs. 205.52 ± 30.91 g; p < 0.001) and LVMI (136.08 ± 9.00 vs. 121.87 ± 16.74 g/m^2; p < 0.001) were observed. At the same time, decreases in RWT and LVEF were noted (p < 0.001), indicating more pronounced structural and functional myocardial remodeling.

Assessment of diastolic function showed impaired relaxation processes in patients with comorbid pathology. In the AH+T2DM group, an increase in VA, a decrease in VE/VA, and prolongation of IVRT (p <0.01) were established, indicating the formation of LV diastolic dysfunction.

The results demonstrate that the combination of T2DM and AH is associated with more pronounced metabolic disorders, immune activation, structural cardiac remodeling, and deterioration of myocardial diastolic function. This may contribute to increased NT-proBNP concentrations and the development of cardiometabolic risk in this patient group.

To determine factors associated with elevated NT-proBNP concentrations in patients with AH, univariate and multivariate logistic regression analyses were performed (Table 2).

According to the results of univariate logistic regression analysis, an association between elevated NT-proBNP concentrations and several

Table 2. *Univariate and multivariate cardiometabolic predictors of elevated NT-proBNP concentrations in patients with AH*

Dependent variable: NT-proBNP (AH)				
Univariate logistic regression analysis (χ^2 = 33.49; P = 0,0040) AUC = 0,961 (0,875–0,994)				
Parameters	**β-coefficient**	**OR**	**95% CI**	**p-value**
RA, cm	8,16570	3518,1713	0,0476 — 259960,7324	0,1534
HbA1c, %	-1,02012	0,3606	0,0808 — 1,6098	0,1814
VE, cm/s	-0,36349	0,6952	0,3829 — 1,2624	0,2323
Leptin, ng/mL	-0,20970	0,8108	0,6612 — 0,9944	0,0440
Aorta, cm	-7,35058	0,0006	0,0000 — 2031,8955	0,3358
LVMM, g	-0,13102	0,8772	0,7613 — 1,0108	0,0700
RV, cm	-24,96691	0,0000	0,0000 — 250,7437	0,1085
BMI, kg/m^2	-0,83501	0,4339	0,1734 — 1,0857	0,0744
SBP, mmHg	-0,076157	0,9267	0,7654 — 1,1220	0,4351
LVMI2, g/m	0,57892	1,7841	0,7792 — 4,0849	0,1708
Insulin, μIU/mL	0,023359	1,0236	0,9330 — 1,1230	0,6213
Blood urea, mmol/L	0,68856	1,9909	0,6211 — 6,3811	0,2466
Blood creatinine, μmol/L	0,082305	1,0858	0,9661 — 1,2203	0,1671
TC, mmol/L	0,20005	1,2215	0,4514 — 3,3053	0,6937
LA, cm	5,99668	402,0897	0,0291 — 5549,5550	0,2176
Multivariate logistic regression analysis (χ^2 =18,41; P = 0.0010) AUC = 0,868 (0,754–0,943)				
VE, см/с	-0,15548	0,8560	0,7133 — 1,0273	0,0947
Leptin, ng/mL	-0,069652	0,9327	0,8686 — 1,0016	0,0552
Aorta, cm	-4,95705	0,0070	0,0001 — 0,5128	0,0235
LVMM, g	-0,039621	0,9612	0,9310 — 0,9923	0,0149

Abbreviations: NT-proBNP — the N-terminal fragment of the precursor of brain natriuretic peptide, AH — arterial hypertension, AUC — the area under the curve, RA— right atrium, CI — confidence interval, OR— odds ratio, HbA1c — glycated haemoglobin, LVMM — left ventricular myocardial mass, RV — right ventricle, BMI— body mass index, SBP — systolic blood pressure, LVMI2 — left ventricular myocardial index 2, TC — total cholesterol, LA— left atrium, VE— diastolic filling velocity.

clinical-metabolic and echocardiographic parameters was established. A statistically significant association was identified for leptin (OR = 0.8108; 95 % CI 0.6612–0.9944; p = 0.0440). At the same time, a tendency toward association was observed for LVM (p = 0.0700), BMI (p = 0.0744), and VE velocity (p = 0.2323).

Other parameters, including HbA1c, insulin, SBP, LVMI, renal function parameters, and lipid profile parameters, did not demonstrate a statistically significant association with elevated NT-proBNP concentrations in univariate analysis (p >0.05). The area under the ROC curve (AUC) was 0.961 (95 % CI 0.875–0.994), indicating high prognostic ability of the model.

Subsequent multivariate logistic regression analysis identified independent determinants of elevated NT-proBNP concentrations in patients with AH. The model included diastolic function parameters, metabolic parameters, and echocardiographic characteristics.

The independent predictors of elevated NT-proBNP concentrations were aortic dimension (OR = 0.0070; 95 % CI 0.0001–0.5128; p = 0.0235) and LVM (OR = 0.9612; 95 % CI 0.9310–0.9923; p = 0.0149). A tendency toward association with VE (p = 0.0947) and leptin (p = 0.0552) was also observed.

The area under the ROC curve for the multivariate model was AUC = 0.868 (95 % CI 0.754–0.943), indicating good prognostic value of the obtained model.

The obtained results demonstrate that elevated NT-proBNP concentrations in patients with AH are predominantly associated with structural and functional cardiac changes and parameters of cardiac remodeling, whereas metabolic parameters have a less pronounced influence.

To determine cardiometabolic determinants of elevated NT-proBNP concentrations in patients with AH and T2DM, univariate and multivariate logistic regression analyses were performed (Table 3).

According to the results of univariate logistic regression analysis, a statistically significant association of elevated NT-proBNP concentrations with parameters of diastolic function and metabolic variables was established. In particular, significant associations were identified for IVRT (OR = 0.9115; 95 % CI 0.8465–0.9815; p = 0.0141), BMI (OR = 1.1653; 95 % CI 1.0075–1.3480; p = 0.0394), LVMI2 calculated using the height-indexed parameter (OR = 1.2903; 95 % CI 1.0867–1.5321; p = 0.0036), and SBP (OR = 1.0444; 95 % CI 1.0040–1.0864; p = 0.0310).

At the same time, a tendency toward association of elevated NT-proBNP concentrations with VE (p = 0.0908), insulin (p = 0.0578), and LV RWT (p = 0.0613) was observed. Other clinical, metabolic, and structural-functional parameters did not demonstrate a statistically significant association with elevated NT-proBNP concentrations (p > 0.05). The area under the ROC curve for the univariate model was AUC = 0.961 (95 % CI 0.875–0.994).

Subsequent multivariate logistic regression analysis identified independent cardiometabolic predictors of elevated NT-proBNP concentrations in patients with AH and T2DM. The independent factors included diastolic function parameters and metabolic variables. In particular, statistically significant predictors were IVRT (OR = 0.9434; 95 % CI 0.8956–0.9937; p = 0.0279), VE (OR = 0.9065; 95 % CI 0.8419–0.9761; p = 0.0093), LVMI2 (OR = 1.1750; 95 % CI 1.0396–1.3281; p = 0.0098), insulin (OR = 1.0560; 95 % CI 1.0053–1.1092; p = 0.0301), and SBP (OR = 1.0319; 95 % CI 1.0028–1.0618; p = 0.0312).

The area under the ROC curve of the multivariate model was 0.803 (95 % CI 0.701–0.882), indicating good prognostic ability.

Thus, in patients with AH and T2DM, elevated NT-proBNP concentrations are associated with a combination of metabolic disorders, myocardial hypertrophy, and left ventricular diastolic dysfunction, reflecting the formation of cardiometabolic myocardial remodelling.

Discussion

In the present study, it was established that in patients with T2DM and AH, elevated NT-proBNP concentrations are associated with parameters of diastolic dysfunction, LVMI, insulin, and SBP. The obtained results support the concept of cardiometabolic myocardial remodeling in patients with a comorbid course of T2DM and AH.

NT-proBNP is a sensitive marker of increased ventricular filling pressure and early manifestations

Table 3. *Univariate and multivariate cardiometabolic predictors of elevated NT-proBNP concentrations in patients with AH and T2DM*

Dependent variable: NT-proBNP (AH+T2DM)				
Univariate logistic regression analysis (χ^2 = 33.49; P = 0,0040) AUC = 0,961 (0,875–0,994)				
Parameters	**β-coefficient**	**OR**	**95% CI**	**p-value**
VE/VA	9,37775	11822357	0,0001–2,61E+012	0,3387
TC, mmol/L	0,13814	1,1481	0,7326–1,7993	0,5467
IVRT, mc	-0,092671	0,9115	0,8465–0,9815	0,0141
Leptin, ng/mL	-0,039841	0,9609	0,8899–1,0377	0,3094
VA, cm/s	0,11127	1,1177	0,9259–1,3493	0,2468
VE, cm/s	-0,20372	0,8157	0,6442–1,0329	0,0908
DBP, mmHg	-0,025334	0,9750	0,9225–1,0305	0,3699
Age, years	0,016202	1,0163	0,9189–1,1240	0,7526
LV RWT, cm	-18,94307	0,0000	0,0000–2,4492	0,0613
BMI, kg/m^2	0,15302	1,1653	1,0075–1,3480	0,0394
LVMI1, g/m^2	-0,037118	0,9636	0,8908–1,0423	0,3543
LVMI2, g/m^{2,7}	0,25490	1,2903	1,0867–1,5321	0,0036
Insulin, μIU/mL	0,066192	1,0684	0,9978–1,1440	0,0578
LVEDD, cm	-7,32530	0,0007	0,0000–4,1643	0,1009
SBP, mmHg	0,043410	1,0444	1,0040–1,0864	0,0310
LVPWT, cm	4,55601	95,2024	0,0000–26959,7271	0,5478
IVST, cm	-4,13007	0,0161	0,0000–489,7188	0,4330
RV, cm	-5,97586	0,0025	0,0000–244,7604	0,3074
LVEF, %	0,044202	1,0452	0,8434–1,2953	0,6864
Multivariate logistic regression analysis (χ^2 = 25,66; P = 0.0003) AUC = 0,803 (0,701–0,882)				
IVRT, mc	-0,058317	0,9434	0,8956–0,9937	0,0279
VE, cm/s	-0,098169	0,9065	0,8419–0,9761	0,0093
BMI, kg/m^2	0,086432	1,0903	0,9897–1,2011	0,0802
LVMI2, g/m	0,16129	1,1750	1,0396–1,3281	0,0098
Insulin, μIU/mL	0,054450	1,0560	1,0053–1,1092	0,0301
SBP, mmHg	0,031418	1,0319	1,0028–1,0618	0,0312

Abbreviations: AH — arterial hypertension, T2DM — type 2 diabetes mellitus, BMI — body mass index, SBP — systolic blood pressure, DBP — diastolic blood pressure, AUC — the area under the curve, CI — confidence interval, OR— odds ratio, TC — total cholesterol, IVRT — isovolumic relaxation time, LV — left ventricle, RWT — relative wall thickness, LVMI1, g/m^2 — left ventricular mass index normalized to body surface area, LVMI2, g/m^{2,7} — left ventricular mass index normalized to height raised to the power of 2.7, LVRWT — left ventricular posterior wall thickness, IVST— interventricular septal thickness, RV — right ventricle, LVEF — left ventricular ejection fraction, RV — right ventricle, BMI— body mass index, NT-proBNP — the N-terminal fragment of the precursor of brain natriuretic peptide, VE — diastolic filling velocity, VA — late diastolic filling velocity.

of HF. According to previous studies, in patients with T2DM, NT-proBNP concentrations may be elevated even in the absence of clinical signs of HF, which is associated with the development of diabetic cardiomyopathy and subclinical diastolic dysfunction. It has been shown that hyperglycemia, insulin resistance, and oxidative stress contribute to myocardial fibrosis, increased LV stiffness, and elevated filling pressure, which stimulate natriuretic peptide secretion [19–23].

In our study, one of the independent predictors of elevated NT-proBNP concentrations was diastolic function parameters, particularly IVRT and VE. Similar results have been reported in several studies, showing that impaired myocardial relaxation is one of the early mechanisms underlying increased NT-proBNP in patients with metabolic disorders [24, 25]. Prolongation of IVRT reflects slowing of active LV relaxation, which is accompanied by increased end-diastolic pressure and stimulation of natriuretic peptide synthesis.

An important finding of the study is the established association between NT-proBNP

and LVMI. Left ventricular hypertrophy is known to be one of the key mechanisms of increased myocardial stress. In patients with combined T2DM and AH, left ventricular hypertrophy develops more rapidly and is more pronounced, reflecting the simultaneous effects of hemodynamic overload and metabolic factors. The literature shows that an increase in LVMI is associated with increased NT-proBNP concentrations independently of LVEF, confirming the role of structural remodelling in the formation of myocardial stress [26–28].

In the present study, an independent association between NT-proBNP concentrations and insulin was also established. This is consistent with the literature, which indicates that insulin resistance plays a role in the development of cardiometabolic remodeling [29]. Hyperinsulinemia contributes to activation of the sympathetic nervous system, sodium retention, increased BP, and stimulation of cardiomyocyte hypertrophy. In addition, insulin resistance is associated with the development of interstitial myocardial fibrosis and deterioration of diastolic function, which may lead to increased secretion of natriuretic peptides.

Another important finding is the established association between NT-proBNP concentrations and SBP. Increased afterload increases LV wall stress and stimulates natriuretic peptide synthesis. In patients with combined T2DM and AH, this mechanism is intensified due to reduced vascular elasticity, endothelial dysfunction, and activation of neurohumoral systems.

The role of obesity and BMI also deserves separate attention. Univariate analysis showed an association between BMI and elevated NT-proBNP concentrations, although in the multivariate model, this association was only marginally significant. According to the literature, the relationship between NT-proBNP and obesity is complex and inconsistent. Some studies demonstrate reduced natriuretic peptide concentrations in obesity due to increased clearance in adipose tissue, whereas others show increased NT-proBNP in the presence of cardiac remodelling [30, 31].

Similar results were also obtained in our previous study, which showed that elevated NT-proBNP concentrations are associated with parameters of diastolic dysfunction, insulin resistance, and myocardial remodelling [32].

The obtained data also confirm the important role of the combined effects of metabolic and hemodynamic factors in the development of elevated NT-proBNP concentrations. In patients with AH and T2DM, several pathogenetic mechanisms are implemented simultaneously, including myocardial hypertrophy, diastolic dysfunction, insulin resistance, activation of the sympathoadrenal system, and endothelial dysfunction. This leads to increased LV filling pressure and stimulation of NT-proBNP secretion.

Thus, elevated NT-proBNP concentrations in patients with AH and T2DM reflect the complex influence of metabolic and structural-functional cardiac changes. Determination of cardiometabolic determinants of elevated NT-proBNP concentrations enables this biomarker to be considered an integral indicator of cardiometabolic risk and early myocardial remodelling in this patient group.

Conclusion

1. In patients with arterial hypertension and type 2 diabetes mellitus, more pronounced cardiometabolic disorders were established compared with patients with isolated arterial hypertension, manifested by increased body mass index, atherogenic changes in the lipid profile, increased leptin and insulin levels, and structural-functional cardiac remodelling. The combination of metabolic and hemodynamic factors is associated with increased left ventricular myocardial mass, cardiac chamber enlargement, and diastolic dysfunction.
2. Elevated NT-proBNP concentrations in patients with arterial hypertension are predominantly associated with structural and functional cardiac changes. According to multivariate logistic regression analysis, left ventricular myocardial mass and aortic dimensions were identified as independent predictors of elevated NT-proBNP concentrations in patients with arterial hypertension, indicating the leading role of cardiac remodelling in the formation of myocardial stress.
3. In patients with combined arterial hypertension and type 2 diabetes mellitus, independent cardiometabolic determinants of elevated NT-proBNP concentrations are parameters

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of diastolic dysfunction, left ventricular mass index, insulin concentration, and systolic blood pressure. This confirms the interrelationship between insulin resistance, myocardial hypertrophy, impaired left ventricular relaxation, and increased myocardial stress in patients with a comorbid course of the disease.

4. Determination of cardiometabolic determinants of elevated NT-proBNP concentrations in patients with arterial hypertension and type 2 diabetes mellitus allows this biomarker to be considered an integral indicator of early cardiac remodelling and subclinical diastolic dysfunction. The use of NT-proBNP in the comprehensive assessment of patients with cardiometabolic pathology may contribute to early stratification of the risk of cardiovascular complications and optimisation of therapeutic tactics.

The authors declare no conflict of interest.

The study was conducted at the authors' own expense.

Authors' contributions: 1 – study concept and design, 2 – collection of factual material, 3 – processing of factual material, 4 – manuscript drafting, 5 – manuscript editing and critical review, 6 – project administration.

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Кардіометаболічні детермінанти підвищених концентрацій NT-proBNP у пацієнтів із цукровим діабетом 2-го типу та артеріальною гіпертензією (огляд літератури та власні дані)

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Резюме
Поєднання артеріальної гіпертензії (АГ) та цукрового діабету (ЦД) 2-го типу асоціюється з високим ризиком кардіального ремоделювання, діастолічної дисфункції лівого шлуночка та розвитку серцевої недостатності. N-кінцевий фрагмент попередника мозкового натрійуретичного пептиду (NT-proBNP) розглядається як чутливий біомаркер міокардіального стресу, підвищення тиску наповнення лівого шлуночка та ранніх структурно-функціональних змін серця. Однак кардіометаболічні чинники, що визначають підвищені концентрації NT-proBNP у пацієнтів із поєднанням АГ та ЦД 2-го типу, залишаються недостатньо вивченими.
Мета Визначити кардіометаболічні детермінанти підвищених концентрацій NT-proBNP у пацієнтів із ЦД 2-го типу та АГ.
Матеріали і методи У дослідження включено 136 пацієнтів з АГ, які були розподілені на дві групи: до першої групи увійшли пацієнти з ізольованою АГ (n=64), до другої групи — пацієнти з АГ у поєднанні з ЦД 2-го типу (n=72). Контрольну групу становили 20 практично здорових осіб. Усім обстеженим проводили комплексне клініко-лабораторне та інструментальне дослідження, включаючи визначення показників ліпідного профілю, HbA1c, інсуліну, лептину, Cys C, NGAL та NT-proBNP методом імуноферментного аналізу, оцінку субпопуляцій лімфоцитів, показників функції нирок та ехокардіографічних параметрів. Для визначення факторів, асоційованих із підвищеними концентраціями NT-proBNP, застосовували

універіативний і мультиваріативний логістичний регресійний аналіз із розрахунком відношення шансів, 95 % довірчого інтервалу та оцінкою прогностичної здатності моделей за допомогою ROC-аналізу.
Результати У пацієнтів із поєднанням АГ та ЦД 2-го типу виявлено більш несприятливий кардіометаболічний профіль порівняно з хворими з ізольованою АГ. Це проявлялося підвищенням індексу маси тіла, загального холестерину, тригліцеридів і коефіцієнта атерогенності, збільшенням рівня лептину, активацією клітинної ланки імунітету, а також більш вираженим структурно-функціональним ремоделюванням серця. У групі АГ+ЦД 2-го типу встановлено збільшення маси міокарда лівого шлуночка, індексу маси міокарда лівого шлуночка, розмірів камер серця, зниження фракції викиду лівого шлуночка та погіршення показників діастолічної функції. Концентрації NT-proBNP у пацієнтів із АГ+ЦД 2-го типу мали тенденцію до підвищення порівняно з групою ізольованої АГ, однак міжгрупова різниця не досягала статистичної значущості (p=0,0791). За результатами мультиваріативного логістичного регресійного аналізу у пацієнтів з ізольованою АГ незалежними предикторами підвищених концентрацій NT-proBNP були розмір аорти та маса міокарда лівого шлуночка; прогностична здатність моделі була доброю (AUC=0,868). У пацієнтів із поєднанням АГ та ЦД 2-го типу незалежними кардіометаболічними детермінантами підвищених концентрацій NT-proBNP визначено IVRT, швидкість раннього діастолічного наповнення (VE), індекс маси міокарда лівого шлуночка, індексований до зросту, концентрацію інсуліну та систолічний артеріальний тиск. Мультиваріативна модель у групі АГ+ЦД 2--го типу також продемонструвала добру прогностичну здатність (AUC=0,803).
Висновки Підвищені концентрації NT-proBNP у пацієнтів з артеріальною гіпертензією та цукровим діабетом 2-го типу формуються під впливом комплексу гемодинамічних, метаболічних і структурно-функціональних чинників. При ізольованій АГ провідне значення мають показники кардіального ремоделювання, тоді як при поєднанні АГ із ЦД 2-го типу спектр детермінант розширюється та включає показники діастолічної дисфункції, гіпертрофії лівого шлуночка, інсулінового гомеостазу та систолічного артеріального тиску. NT-proBNP може розглядатися як інтегральний біомаркер раннього кардіального ремоделювання, субклінічної діастолічної дисфункції та стратифікації кардіометаболічного ризику у пацієнтів із коморбідним перебігом АГ та ЦД 2-го типу.

Ключові слова: артеріальна гіпертензія, цукровий діабет 2 типу, NT-proBNP, кардіометаболічний ризик, діастолічна дисфункція, інсулін.

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