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Risk factors for acute decompensated heart failure in type 2 diabetes patients

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Aim. To identify risk factors for acute decompensated heart failure (ADHF) in patients with type 2 diabetes (T2D).
Materials and methods. In the cardiology department, 129 patients with ADHF were registered within 8 months, 59 (45,7%) of them had T2D. The study included 117 ADHF patients who were divided into two groups depending on the presence of T2D: group 1 (n=49; 41,9%) — patients with T2D, group 2 (n=67; 55,9%) without T2D. The ADHF was verified by rapid progress of hypoperfusion and congestion, which required emergency hospitalization and inotropic and/or intravenous diuretic therapy. In the first 48 hours of hospitalization, echocardiography was performed, levels of N-terminal pro-brain natriuretic peptide (NT-proBNP) and creatinine were determined; the glomerular filtration rate was estimated.
Results. The incidence of T2D among patients with ADHF was 45,7%. There were following risk factors for ADHF in T2D patients: diabetic ketoacidosis (p=0,002), hypertensive crisis (p=0,017), history of acute coronary syndrome (p=0,048), atrial fibrillation (p=0,030), chronic kidney disease (p=0,003), pneumonia (p=0,035), progression of anemia (p=0,049), low prevalence of beta-blockers use (p=0,001), use of inappropriate antidiabetic drugs for HF patients (sulfonylureas, insulin). ADHF, assessed by NT-proBNP level, was significantly more severe in T2D patients (p=0,001) with pronounced congestion symptoms (p=0,001), which led to an increase in the need for diuretic therapy (p=0,002). Cardiac remodeling in T2D patients with ADHF is characterized mainly by the preserved left ventricular ejection fraction (LVEF), severe LV diastolic dysfunction (LVDD) and LV hypertrophy (LVH).
Conclusion. The development of ADHF in T2D patients is associated with various risk factors and is characterized by severe congestion symptoms, high need for diuretic therapy, mainly preserved LVEF in combination with severe LVDD and LVH.

Key words: acute decompensated heart failure, diabetes.

Relationships and Activities: not.

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The prevalence of type 2 diabetes (T2D) among patients hospitalized with heart failure (HF) is 44%, among patients with a preserved left ventricle ejection fraction (LVEF) — 46% [1]. In patients with acute decompensated HF (ADHF), the prevalence of T2D reaches 34%, prediabetes — 17% [2].

According to the meta-analysis of randomized clinical trials (RCT) and registers, T2D increases the risk of cardiovascular mortality in patients with acute HF by 32%, and re-hospitalizations — by 16% [3]. The mortality risk among patients with T2D and ADHF increases by 1,7 times during hospitalization, 1,4 times after 18 months, 1,3 times after 5 years [4].

Target glucose levels for patients with T2D and ADHF are not defined. According to a study by Shirakable A, et al., glucose levels >22,2 mmol/L increases the death risk in patients after ADHF during the year by 2,3 times, while levels <5,6 mmol/L — by 3,25 times [5].

There are conflicting data on risk factors, clinical features of ADHF, types of cardiac and target organ remodeling, the need and duration of parenteral diuretic therapy, the need for inotropic agents and modification of background HF therapy, the choice of antidiabetic therapy at the time of event and after it [1-3].

The solution to these issues will allow timely prevention of ADHF hospitalizations, which can significantly improve the prognosis and quality of life of T2D patients.

The aim of this study was to identify risk factors for ADHF in T2D patients.

Material and methods

This study was performed in accordance with the Helsinki declaration and Good Clinical Practice standards. The local medical ethics committee approved this study. All patients signed informed consent.

In the cardiology department of the multidisciplinary clinic, 129 patients with ADHF were registered in 8 months, of which 59 (45,7%) patients had T2D.

ADHF was verified following a rapid increase in symptoms and signs of hypoperfusion (low pulse pressure, cold extremities, fatigue, oliguria) and scongestion (orthopnea, jugular vein distention, lower extremity edema, ascites, hepatomegaly), which required emergency hospitalization, use of inotropic agents and/or intravenous diuretic therapy.

T2D was verified under the World Health Organization criteria (1999-2013).

The inclusion criterion was the presence of ADHF. There were following exclusion criteria: cardiogenic shock, pulmonary edema, acute thromboembolism, type 1 diabetes, prediabetes, acute coronary syndrome (ACS) and/or stroke less than a month before, aortic dissection, acute heart valve disorders (chordae tendinae rupture, etc.), major surgery less than a month before, heart injuries, infectious endocarditis, acute hepatitis and cirrhosis, end-stage renal disease, alcohol abuse, non-cardiac edema, cancer, dementia and mental illness.

A total of 117 patients with ADHF were examined, which were divided into 2 groups depending on the presence of T2D. The first group consisted of 49 (41,9%) patients with T2D, the second — 67 (55,9%) non-diabetic patients.

The congestion was assessed by congestion score of the European Society of Cardiology, endorsed by the European Society of Intensive Care Medicine (2010).

Depending on symptoms and signs of congestion and hypoperfusion, ADHF phenotypes were identified by hemodynamic profile based on the Forrester JS and Stevenson LW classification.

Echocardiography was performed in the first 48 hours of hospitalization using the VIVID 7 ultrasound system (GE Healthcare, USA) according to European Association of Cardiovascular Imaging and American Society of Echocardiography guidelines.

The concentration of serum N-terminal pro-brain natriuretic peptide (NT-proBNP) was determined by enzyme-linked immunosorbent assay using Vector-Best (Russia) reagent kit and Expert Plus Microplate Reader (Biochrom, UK). ADHF was diagnosed at NT-proBNP >300 pg/ml.

Renal function was assessed by serum creatinine concentration and glomerular filtration rate (GFR), calculated by CKD-EPI equation.

Statistical processing was performed using the STATISTICA 12.0 software package. The normality was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Quantitative traits were presented as mean and standard deviation (M±SD) or median and quartiles (Me [Q1; Q3]). For qualitative traits, the absolute frequency and the frequency in percent (%) were calculated. In non-normal distribution, statistical processing was carried out using Mann-Whitney test for quantitative traits and chi-squared test or Fisher's test ($n \leqslant 5$) for qualitative traits. The relationship between traits was studied using Spearman's rank correlation coefficient. The differences were considered significant at $p<0,05$.

Results

The prevalence of T2D among patients with ADHF amounted to 45,7%.

Table 1 presents anamnestic data before and during hospitalization for groups of subjects. Table 1 shows compared with the second group, in the first group ADHF were significantly more likely to be

Table 1

Comparison of anamnestic data of ADHF patients with/without T2D (n=117)

Parameter	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p
Age, years	66,0±6,8	70,0±9,2	0,008
Sex, m/f, abs./%	18/31 (37/63)	41/27 (60/40)	0,012/0,012
Smoking, abs./%	17/34,7	29/42,6	0,755
Duration of HF, years	5,8 [2,1;7,2]	5,2 [2,6;6,9]	0,237
Mean HF class before hospitalization	3,2 [2,5;3,8]	3,0 [2,4;3,6]	0,261
Duration of T2D, years	9,1 [3,8;17,5]	-	
DKA, abs./%	7/14,3	0/0	0,002
Hypertension, abs./%	47/95,9	62/91,2	0,317
Hypertensive crisis at admission, abs./%	4/8,2	0/0	0,017
CAD, abs./%	33/67,3	38/55,9	0,211
History of ACS, abs./%	24/49,0	21/30,9	0,048
History of CABG, abs./%	11/22,4	6/8,8	0,040
History of PCI, abs./%	12/24,5	13/19,1	0,485
History of AF, abs./%	10/20,4	9/13,2	0,300
AF with HR >110 bpm at admission, abs./%	8/16,3	3/4,4	0,030
History of PE, abs./%	2/4,1	0/0	0,093
Ventricular rhythm disturbances, abs./%	25/51,0	38/55,9	0,603
TIA, history of stroke, abs./%	12/24,5	7/10,3	0,182
Stage 3-4 CKD, abs./%	23/46,9	14/20,6	0,003
Pneumonia, abs./%	5/10,2	1/1,5	0,035
History of COPD, abs./%	7/14,3	9/13,2	0,871
Exacerbation of COPD, abs./%	4/4,1	1/1,5	0,078
Acute inflammatory diseases, abs./%	2/4,1	2/3,0	0,738
Progression of chronic anemia, abs./%	6/12,2	2/3,0	0,049

Abbreviations: ADHF — acute decompensated heart failure, T2D — type 2 diabetes, HF — heart failure, DKA — diabetic ketoacidosis, CAD — coronary artery disease, ACS — acute coronary syndrome, CABG — coronary artery bypass grafting, PCI — percutaneous coronary intervention, AF — atrial fibrillation, HR — heart rate, PE — pulmonary embolism, TIA — transient ischemic attack, CKD — chronic kidney disease, COPD — chronic obstructive pulmonary disease.

caused by diabetic ketoacidosis (DKA), hypertensive crisis, ACS history, atrial fibrillation (AF) with heart rate (HR) >110 bpm, CKD, pneumonia, anemia aggravation.

Comparison of laboratory parameters between groups are presented in Table 2.

Significant differences in laboratory parameters between groups confirm differences in the prevalence of comorbidities. ADHF, assessed by the NT-proBNP level, was significantly more severe in T2D patients.

Correlation analysis of patients with ADHF and T2D revealed a direct moderate and strong significant relationship of plasma glucose (r=0,41; p=0,035), systolic blood pressure (r=0,38; p=0,011), HR >110 bpm (r=0,69; p=0,002), C-reactive protein (r=0,58; p=0,005) and inverse strong dependence of GFR (CKD -EPI) (r=-0,55; p=0,023) and hemoglobin (r=-0,56; p=0,018) with a NT-proBNP concentration.

Table 3 shows a comparison of symptoms in ADHF patients between groups.

Patients with ADHF and T2D had a higher frequency and severity of congestion symptoms, the wet+warm phenotype was more often recorded.

Correlation analysis of patients with ADHF and T2D revealed a direct moderate significant relationship between glycated hemoglobin (HbA_{1c}) and the average congestion score (r=0,44; p=0,026).

Characteristics of therapy for HF and T2D patients before and after hospitalization is presented in Table 4.

In both groups, before hospitalization, there was a low frequency of recommended drug use, which required its more intensive intake during hospitaliza-

Table 2

Comparison of laboratory data of ADHF patients with/without T2D (n=117)

Parameter	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p
Hemoglobin, g/L	121,5 [98,6;139,0]	130,7 [112,4;140,8]	0,011
Hematocrit, %	40,5 [34,5;44,1]	41,2 [36,8;45,0]	0,267
Fasting plasma glucose, mmol/L	9,1 [4,3;22,8]	5,5 [3,6;8,0]	<0,001
Persistent hyperglycemia >10 mmol/L, abs./%	12/24,5	0/0	0,001
HbA_{1c}, %	9,8 [6,7;12,5]	5,3 [4,5;5,9]	<0,001
Total cholesterol, mmol/L	5,2 [3,4;6,1]	5,7 [3,5;6,6]	0,672
Total protein	62,8 [61,3;69,5]	66,5 [62,2;71,6]	0,348
Albumin	36,8 [33,4;39,8]	38,1 [33,5;39,0]	0,389
Plasma sodium, mmol/L	144,3±6,1	142,8±6,4	0,078
Plasma potassium, mmol/L	4,8 [4,2;5,1]	4,2 [3,6;4,6]	<0,001
Total bilirubin, μmol/L	18,3 [12,5;20,0]	19,4 [13,2;22,4]	0,189
ALT, U/L	22,8 [18,9;38,6]	25,0 [19,2;36,7]	0,672
AST, U/L	34,6 [22,3;45,1]	32,9 [21,7;43,0]	0,785
Serum creatinine, μmol/L	111,6 [86,2;143,1]	99,8 [78,4;132,9]	<0,001
Urea	8,0 [5,7;10,8]	7,8 [5,7;9,2]	0,763
GFR (CKD-EPI), ml/min/1,73 m^2	61,3 [44,8;72,7]	68,7 [49,1;77,5]	0,005
ESR, mm/h	23,8 [15,3;47,3]	18,7 [12,3;27,9]	0,008
C-reactive protein, mg/L	14,5 [4,1;26,2]	8,2 [3,3;12,4]	<0,001
NT-proBNP, pg/ml	987,9 [563,0;1676,7]	846,6 [453,3;1156,8)	<0,001

Abbreviations: ADHF — acute decompensated heart failure, T2D — type 2 diabetes, HbA_{1c} — glycated hemoglobin, ALT — alanine aminotransferase, AST — aspartate aminotransferase, GFR — glomerular filtration rate, ESR — erythrocyte sedimentation rate, NTproBNP — N-terminal pro-brain natriuretic peptide.

Table 3

Comparison of clinical data of ADHF patients with/without T2D (n=117)

Parameter	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p
Congestion score	4,5 [2,5;6,5]	3,0 [2,0;4,0]	<0,001
Frequency of congestion, abs./%	41/83,7	45/66,2	0,035
Resting HR per minute	91,3±22,6	88,8±20,9	0,444
Resting RR per minute	22,6±3,8	21,9±4,0	0,143
SBP, mm Hg	138,7±25,5	134,9±23,2	0,373
DBP, mm Hg	88,7±12,7	86,1±10,8	0,329
PP, mm Hg	52,5±17,4	49,7±15,6	0,315
Wet-warm phenotype, abs./%	42/85,7	47/69,1	0,038
Wet-cold phenotype, abs./%	2/4,1	7/10,3	0,214
Dry-warm phenotype, abs./%	4/8,2	10/14,7	0,283
Dry-cold phenotype, abs./%	1/2,0	5/7,4	0,199
SpO_2, %	94,5±3,4	95,1±3,8	0,078

Abbreviations: ADHF — acute decompensated heart failure, T2D — type 2 diabetes, HR — heart rate, RR — respiratory rate, SBP — systolic blood pressure, DBP — diastolic blood pressure, PP — pulse pressure, SpO_2 — arterial blood oxygenation.

tion. Patients with ADHF and T2D took beta-blockers significantly less prior to hospitalization. The frequency of use of glucose-lowering drugs not recommended for HF was high.

Table 5 shows characteristics of in-hospital treatment of patients with ADHF by groups. The need for diuretic therapy in the first group was significantly higher than in the second.

Table 4

Comparison of therapy data of ADHF patients with/without T2D (n=117)

Drugs before/after admission to the hospital, abs./% (p)	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p_{1-2}
ACE inhibitors	22/44,4 38/77,6 p*<0,001	33/48,5 55/80,9 p*<0,001	0,698 0,660
ARB	10/20,4 11/22,4 p*=0,806	12/17,6 13/19,1 p=0,825	0,707 0,660
Beta blockers	12/24,5 44/89,8 p*<0,001	36/52,3 66/97,1 p*<0,001	0,003 0,103
MCRA	5/10,2 40/81,6 p*<0,001	7/10,3 56/82,4 p*<0,001	0,988 0,921
Oral loop diuretics	18/36,7 42/85,7 p*<0,001	23/33,8 61/89,7 p*<0,001	0,745 0,512
Digoxin	9/18,4 12/24,5 p*=0,461	8/11,8 12/17,6 p*=0,333	0,318 0,366
Anticoagulants	5/10,2 15/30,6 p*=0,013	4/5,9 21/30,9 p*<0,001	0,387 0,976
Sulfonylureas	23/46,9 18/36,7 p*=0,306	-	-
Metformin	29/59,2 12/24,5 p*<0,001	-	-
Insulin therapy	10/20,4 27/55,1 p*<0,001	-	-
SGLT2 inhibitors	2/4,1 2/4,1 p*=0,999	-	-
Combination of glucose-lowering drugs	35/71,4 16/32,7 p*<0,001	-	-

Note: p* — before and after admission to the hospital.

Abbreviations: ADHF — acute decompensated heart failure, T2D — type 2 diabetes, ACE inhibitors — angiotensin converting enzyme inhibitors, ARB — angiotensin II receptor blockers, MCRA — mineralocorticoid receptor antagonists, SGLT2 — sodium/glucose cotransporter 2.

Parameters of cardiac structure and function by groups according to echocardiography are presented in Table 6.

LVEF and the prevalence of HF with preserved EF (HFpEF), the severity of left ventricular diastolic dysfunction (LVDD), the left ventricular myocardial mass index (LVMI) were significantly higher in patients with ADHF and T2D.

Correlation analysis of patients with ADHF and T2D revealed a direct moderate and strong significant relationship between LVEF (r=0,32; p=0,018), E/e' (r=0,49; p=0,002), LVMI (r=0,65; p<0,001) and HbA_{1c}.

There are following study limitations: a small sample of patients with T2D and ADHF; to determine the predictor value of newly revealed risk factors for ADHF in T2D patients, contingency tables should be created and the odds ratio and relative risk should be calculated, indicating sensitivity and specificity for each factor.

Discussion

In our study, the prevalence of T2D among patients with ADHF was 45,7%. In single studies, such data are presented. Similar value was obtained in a study by Khoo K, et al. with 1191 patients, where it amounted to 49% [2].

Table 5

Comparison of ADHF therapy in patients with/without T2D (n=117)

Parameter	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p
Intravenous furosemide			
Starting dose on the first day, mg	60 [40;100]	60 [60;80]	0,435
Total dose during hospitalization, mg	640 [480;960]	480 [320;800]	0,002
Duration of treatment, days	7,2 [6,1;9,8]	5,2 [3,0;6,3]	<0,001
Oral loop diuretics			
Daily dose of furosemide, mg	40,0 [40,0;80,0]	40,0 [40,0;80,0]	0,873
Daily dose of torasemide, mg	20,5 [5,9;18,5]	15,1 [5,0;12,6]	0,014
Intravenous nitroglycerin, abs./%	25/51,0	30/44,1	0,461
Inotropic support/Vasopressors, abs./%	4/8,2; 4/8,2	2/2,9; 2/2,9	0,207 0,207

Note: ADHF — acute decompensated heart failure, T2D — type 2 diabetes.

Table 6

Comparison of ADHF therapy in patients with/without T2D (n=117)

Parameter	Group 1 (ADHF+T2D, n=49)	Group 2 (ADHF, n=68)	p
LVEF, %	52,2±12,7	45,3±13,1	0,004
LVEF >50%, abs./%	34/69,4	25/38,8	<0,001
LVEF of 40-49%, abs./%	9/18,4	29/42,6	0,006
LVEF <40%, abs./%	6/12,2	14/20,6	0,237
E/e´ mean	11,4 [4,7;17,0]	14,5 [9,7;18,3]	<0,001
E/e´ mean >14, abs./%	28/57,1	29/42,6	0,122
LVMI, g/m^2	112,2 [89,6;131,9]	101,4 [76,5;123,]	<0,001
LAV/BSA, ml/m^2	38, 2 [33,1;44,2]	35,1 [25,41;44,03]	0,143
LAV/BSA >34 ml/m^2	40/81,6	49/72,1	0,232
RAD/BSA, cm/m^2	2,9 [1,8;3,3]	2,6 [1,6;3,1]	0,076
RAD/BSA >2,5 cm/m^2, abs./%	37/75,5	40/58,8	0,061
PASP, mm Hg	41,7 [30,2;56,4]	37,4 [32,0;49,7]	0,035
Pulmonary hypertension, abs./%	38/77,6	47/69,1	0,313
IVC diameter, mm	0,24 [0,20;0,25]	0,20 [0,20;0,23]	0,018
IVC extension, abs./%	23/46,9	22/32,4	0,110
No IVC collapse, abs./%	22/44,9	19/27,9	0,058

Abbreviations: ADHF — acute decompensated heart failure, T2D — type 2 diabetes, LVEF — left ventricular ejection fraction, E — early ventricular filling velocity, e' mean — mean early diastolic mitral annular velocity, LVMI — left venricular mass index, LAV — left atrial volume, BSA — body surface area, RAD — right atrial diameter, PASP — pulmonary arterial systolic pressure, IVC — inferior vena cava.

Clinicians are well aware that DKA can cause ADHF, especially in elderly patients. In our study, DKA was recorded in 14,3% of patients with ODS. However, the literature data do not describe pathogenesis of this relationship. DKA is considered mainly from the perspective of respiratory failure; there are no guidelines on rehydration and diuretic therapy in ODS due to DKA [6].

A number of authors agree that in patients with T2D, the risk of ADHF is associated, as shown in our study, with atherosclerosis-related diseases, a history of arrhythmias, CKD, and a low beta-blocker use [7]. Many researchers have also demonstrated that pneumonia and inflammatory diseases can cause rehospitalizations with HF, already within 30 days in T2D patients [8].

Rubin DJ, et al. proposed a special calculator for risk of rehospitalizations in T2D patients — DERRI (Diabetes Early Readmission Risk Indicator), which included the same laboratory parame-

ters that are presented in our study as risk factors for ADHF [9].

In our study, 46,9% of patients with T2D and HF took sulfonylureas prior to hospitalization. Recently, more and more data has been collecting that these glucose-lowering drugs not only increases the risk of HF, but also related hospitalizations. According to the study by Heaton PC, et al., sulfonylureas were found to increase the risk of hospitalization with HF by 29% [10].

Endocrinologists regulate that insulin therapy should be used only with persistent hyperglycemia >10 mmol/L, regardless of the time it was determined [11]. In our study, only 24,5% of T2D patients had persistent hyperglycemia >10 mmol/L on the first day of hospitalization, however, the frequency of insulin therapy during hospitalization was significantly increased from 20,4% to 55,1% (p<0,001). According to the post-hoc analysis of EVEREST study (Efficacy of Vasopressin Antagonism in Heart Failure Outcome Study with Tolvaptan), insulin therapy in patients with ADHF increases the risk of cardiovascular mortality and hospitalization with HF by 25% [12].

In a meta-analysis of 11 observational studies, it was determined that the use of metformin reduces the risk of all-cause mortality by 22% in HF patients [13]. In our study, the frequency of metformin use prior to hospitalization was 59,2%; during hospitalization it was significantly reduced to 24,5%. The unreasonable withdrawal of metformin in ADHF is probably associated with a risk of lactic acidosis. According to Chang CH, et al., the frequency of lactic acidosis in the metformin group is not higher than when using other antidiabetic therapy [14]. Moreover, data have been obtained that the metformin use for T2D patients in acute heart failure reduces the death risk by 67% (p<0,001) [15].

Khoo K, et al., confirmed our results in relation to cardiac structural and functional features in T2D patients, characterized by the predominance of preserved LVEF, severe LVDD and LV concentric hypertrophy [2].

Conclusion

The incidence of T2D among patients with ADHF was 45,7%. There were following risk factors for ADHF in T2D patients: DKA, hypertensive crisis, history of ACS, AF with HR >110 bpm, CKD, pneumonia, progression of anemia, low beta-blockers use, use of inappropriate antidiabetic drugs for HF patients (sulfonylureas, insulin). ADHF, assessed by NT-proBNP level, was significantly more severe in T2D patients with severe congestion symptoms, which led to an increase in the need for diuretic therapy with a group without T2D. Cardiac remodeling in T2D patients with ADHF is characterized mainly by the preserved LVEF, severe LVDD and LV hypertrophy.

Relationships and Activities: not.

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