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A PREDICTION MODEL FOR CHRONIC HEART FAILURE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Abstract

Introduction. One of the common adverse complications that may occur in patients with type 2 diabetes mellitus (T2DM) after acute myocardial infarction (AMI) is the development and progression of chronic heart failure (CHF). Currently, it remains relevant to study biomarkers of energy homeostasis that are involved in the pathogenesis of CHF in T2DM patients.
Aim. To predict the development of CHF in patients with T2DM using a generalized linear mixed model (GLMM).
Materials and methods. A total of 74 patients with T2DM were examined after ST-segment elevation AMI (STEMI). Serum concentrations of adropin, fatty acid binding protein 4 (FABP4) and C1q/TNF-related protein 3 (CTRP3) were measured using enzyme-linked immunosorbent assays. Fasting blood glucose levels were determined by the glucose oxidase method. Serum high-density lipoprotein (HDL) levels were analyzed by the peroxidase enzymatic method. Low-density lipoprotein (LDL) levels were estimated by the Friedewald formula. The GLMM was applied to construct a predictive model of CHF in patients with T2DM.
Results. The study has revealed decreased serum adropin and CTRP3 levels and increased FABP4 levels in T2DM patients after STEMI. A predictive model of CHF development in patients with T2DM has been developed. The fixed effects of the model were represented by two univariate (adropin and CTRP3 on day 1) and two bivariate indicators (adropin and FABP4 on day 10, glucose on day 1 and 10), as well as the random effects were represented by five univariate indicators (acute heart failure, diastolic blood pressure on day 1, heart rate on day 10, LDL on day 1 and HDL on day 1). The accuracy of predicting the occurrence of NYHA functional class II within a year in patients with T2DM was 61.5%, while that of NYHA functional class III was 89.6%. The overall accuracy of the model was 79.7%.
Conclusions. Thus, the level of adropin on day 1 has been found to be a strong negative prognostic factor, while the combined effect of the adropin and FABP4 levels on day 10 – a positive prognostic factor. The combined effect of blood glucose levels on day 1 and 10, as well as a negative effect of CTRP3 concentration on day 1 has been shown. The option of using energy metabolism biomarkers in the model has indicated the importance of studying these parameters in T2DM patients.

Keywords: **energy metabolism, heart failure, myocardial infarction, type 2 diabetes mellitus**

INTRODUCTION

Diabetes mellitus (DM) is a risk factor for the occurrence and progression of chronic heart failure (CHF). Among the preconditions for the development of CHF are alterations in the extracellular matrix, resulting in myocardial fibrosis. DM also causes disturbances in lipid and carbohydrate metabolism, leading to an increase in the myocardial need for fatty acid oxidation and a decrease in glucose oxidation [1]. Changes in energy metabolism that affect the cardiomyocyte contractile function are extremely important in the pathogenesis of heart failure (HF). Once the contractile performance of the heart is impaired, a decrease in the mitochondrial oxidative function occurs, that is reversed by an accelerated production of adenosine triphosphate via the glycolysis pathway [2]. Therefore, understanding the metabolic fates characteristic of diabetic patients will help to slow the progression of HF. Particular attention is paid to patients with diabetes and reduced or preserved left ventricular ejection fraction, since mortality rates for this patient cohort are higher as compared to those among patients without diabetes [3]. The cause of the onset and progression of HF in patients with acute myocardial infarction (AMI) is the loss of cardiomyocytes due to cell death, replacement by non-contractile fibrotic scar tissue and alterations in neurohumoral processes [4]. It has been proven that adipokines, in particular C1q/TNF-related protein 3 (CTRP3), could be involved in the pathogenesis of HF [5]. The study has indicated that low serum adropin levels in combination with any level of N-terminal pro-B-type natriuretic peptide (NT-proBNP) and the use of sodium-glucose cotransporter 2 (SGLT2) inhibitors were independent predictors for the development of HF with preserved ejection fraction [6]. Therefore, the study on adipokine metabolism is of interest to scientists. Diabetic patients after AMI are at a higher risk of adverse complications. The use of prognostic models in clinical practice will help to identify type 2 diabetes mellitus (T2DM) patients who require careful monitoring during treatment for preventing subsequent potential complications [7]. To reduce the myocardial necrosis area, improve myocardial viability and accelerate the processes of cardiac healing are also critical issues in the treatment of patients after AMI to prevent the HF occurrence [8]. Currently, scientists are constantly searching for new biomarkers that could be involved in the pathogenesis of HF. Studying metabolic mechanisms underlying the development of HF in patients with T2DM after AMI continues to be one of the particularly interesting scientific fields of concern addressed. Examining the changes that occur in biomarkers of energy metabolism may provide a new therapeutic approach for timely correction of impaired cardiac energy metabolism and enhancement of the myocardial contractile performance.

AIM

To predict the development of CHF in patients with T2DM using a generalized linear mixed model (GLMM) to improve the quality of diagnostic modalities for these patients.

MATERIALS AND METHODS

Data from a prospective cohort study of 74 patients with T2DM were analyzed based on the results of examinations and treatment in the Cardiology Department of the State Institution «L. T. Malaya National Therapy Institute of the National Academy of Medical Sciences of Ukraine», and Kharkiv Railway Clinical Hospital No. 1 of the branch «Center of Healthcare» of Public Joint Stock Company «Ukrainian Railway». The cause of HF in diabetic patients was the first-time acute myocardial infarction with ST segment elevation (STEMI).

Age ≥ 45 years, first-time acute STEMI, cases of acute HF were specified as inclusion criteria for predicting HF in T2DM patients. All 74 patients met these criteria. Cases of identified severe comorbidities (type 1 diabetes mellitus (DM), non-ST-elevation myocardial infarction (NSTEMI), recurrent myocardial infarction, autoimmune diseases, New York Heart Association (NYHA) functional class I and IV CHF, chronic obstructive pulmonary disease, bronchial asthma, heart valve disease, symptomatic hypertension, severe liver and kidney dysfunction, severe anemia, bleeding), severe acute respiratory syndrome-related coronavirus 2, malignancy, inability to provide written informed consent were excluded from the present study.

Joint recommendations of the American Diabetes Association and the European Association for the Study of DM were used to diagnose and manage T2DM [9]. The study followed the recommendations of the European Society of Cardiology [10] for the diagnosis and treatment of STEMI patients. Patients with acute and chronic HF were diagnosed and managed according to the European [11] and American recommendations [12] and in compliance with the NYHA functional classification II-III due to post-infarction cardiosclerosis.

All the patients were informed about the voluntary and anonymous participation and signed an informed consent form prior to the study. Diagnostic tests were provided by the Biochemical Department of the Central Research Laboratory of Kharkiv National Medical University. Blood samples were collected on the 1st and 10th day of observation and stored at -20 °C until thawing for assay.

Serum high-density lipoprotein (HDL) cholesterol was measured by the enzymatic peroxidase method using an assay kit HDL Cholesterol liquicolor (Germany). Low-density lipoprotein (LDL) levels were estimated by the Friedewald formula. Fasting glucose concentrations were determined by the glucose oxidation method using the «Glucose liquicolor» reagent kit (Human, Germany). Serum levels of adropin, FABP4 and CTRP3 were measured by enzyme-linked immunosorbent assay on a «Labline-90» (Austria) analyzer using a commercial test system «Human Adropin» (Elabscience, USA), «Human Fatty acid binding protein 4» (Elabscience, USA) and «Human CTRP3» (Aviscera Bioscience Inc, Santa Clara, USA), respectively, according to manufacturers' protocols. The basic principle of the method used to quantify the levels of adropin, FABP4 and CTRP3 is the sandwich technique, consisting in an antibody pair of the capture antibody and biotin-labeled detection antibody binding to a captured target. The study has determined normal ranges for adropin – 23.58 ± 2.56 pg/mL, FABP4-5.02±1.92 ng/mL, CTRP3-325.97±42.22 ng/mL.

Statistical analysis of the findings was performed using the licensed program «IBM SPPS Statistics, version 27.0» (2020), license No. L-CZAA-BKKMKE. The examined parameters concerning the distribution normality were assessed according to the Kolmogorov-Smirnov criterion. The following main statistical parameters were calculated: mean (M) and the standard deviation (SD). Nominal variables were presented as number and percentage. The threshold significance level to test statistical hypotheses in the study was 0.05. The GLMM was applied to construct a prediction for the development of HF, since this statistical model provided a high consistency in predicting the HF development and testing new hypotheses based on identification of correlations equal to mean values of variables, their variances and covariances [13].

RESULTS

The study has revealed increased levels of adropin and CTRP3 by 35.68% and 14.32%, respectively, in diabetic patients on the 10th day of follow-up, as well as 28.49% decreased FABP4 levels as compared to the 1st day ($p<0.05$). There were no fatal outcomes among AHF patients during the follow-up period (Table 1).

A total of 138 parameters were measured in diabetic patients on the 1st and 10th day of follow-up. The presence of patients with NYHA functional class II-III HF in the sample has made it impossible to construct statistically significant correlations between *y* and the measured parameters. In that case, principal component analysis was used to select only the values with the highest degree of extraction at the first stage. The number of the GLMM model possible variables, the list of which is given in Table 2, has therefore considerably reduced.

The next stage was the sequential consideration of all options for including or excluding the selected variables from Table 2 into the number of fixed or random model effects. The criteria for selecting a qualitative statistically justified model were information criteria (Akaike and Bayesian) as well as the total statistical significance of the model and model variables.

Table 1

Main characteristics of the patients (n=74)

Characteristics		Values: M±SD, n (%)
Age, years		59.42±7.66
Sex, n (%)	Male	51 (68.9)
	Female	23 (31.1)
AHF, n (%)		14 (18.9)
Systolic BP on the 1st day, mm Hg		143.82±29.07
Systolic BP on the 10th day, mm Hg		130.62±12.14
Diastolic BP on the 1st day, mm Hg		83.76±12.35
Diastolic BP on the 10th day, mm Hg		80.68±6.48
Heart rate on the 1st day, beats per minute		86.14±20.96
Heart rate on the 10th day, beats per minute		72.28±6.49
LDL on the 1st day, mmol/L		5.1±1.39
HDL on the 1st day, mmol/L		1.13±0.32
Glucose on the 1st day, mmol/L		11.23±4.57
Glucose on the 10th day, mmol/L		7.91±1.95
Adropin on the 1st day, pg/mL		13.65±5.12
Adropin on the 10th day, pg/mL		18.52±3.82
FABP4 on the 1st day, ng/mL		10.53±2.01
FABP4 on the 10th day, ng/mL		7.53±1.83
CTRP3 on the 1st day, ng/mL		226.06±52.11
CTRP3 on the 10th day, ng/mL		258.43±35.73

Note: AHF – acute heart failure, BP – blood pressure, CTRP3 – C1q/TNF-related protein-3, FABP 4 – fatty acid-binding protein-4, HDL – high-density lipoprotein, LDL – low-density lipoprotein.

Table 2

Parameters selected for the GLMM

Parameter, units of measurement	Correlation with *y*
Adropin on the 1st day, pg/mL	0.930
Adropin on the 10th day, pg/mL	0.868
CTRP3 on the 1st day, ng/mL	0.842
CTRP3 on the 10th day, ng/mL	0.870
FABP4 on the 1st day, ng/mL	0.883
FABP4 on the 10th day, ng/mL	0.729
Systolic BP on the 1st day, mm Hg	0.865
Systolic BP on the 10th day, mm Hg	0.821
Diastolic BP on the 1st day, mm Hg	0.877
Diastolic BP on the 10th day, mm Hg	0.879
Heart rate on the 1st day, beats per minute	0.915
Heart rate on the 10th day, beats per minute	0.914
Glucose on the 1st day, mmol/L	0.844
Glucose on the 10th day, mmol/L	0.775
LDL on the 1st day, mmol/L	0.735
HDL on the 1st day, mmol/L	0.871
AHF	0.717

So, we tested all probable hypotheses and combinations of variables until the best predictive accuracy *y* of the statistically significant model and its independent variables were found, namely, fixed effects, presented in Table 3, and random effects, presented in Table 4.

The fixed (main) effects of the model are given by two one-factor and two two-factor indicators (Table 3), and the random effects are given by five one-factor indicators (Table 4). The overall accuracy of the model was 79.7%.

Table 3

Indicators expressing fixed GLMM effects

Parameters	Statistical significance, p	Coefficient in the GLMM, X
One-factor indicators		
Adropin on the 1st day	p = 0.030	-0.159
CTRP3 on the 1st day	p = 0.008	-0.020
Two-factor indicators (combined impact of two-factor indicators)		
Adropin on the 10th day and FABP4 on the 10th day	p = 0.034	0.017
Glucose on the 1st and 10th day	p = 0.018	-0.019

Table 4

Parameters expressing random GLMM effects

Parameters	Covariations
One-factor indicators	
AHF	2.657
Diastolic BP on the 1st day	0.312
Heart rate on the 10th day	0.002
LDL on the 1st day	0.332
HDL on the 1st day	2.849

DISCUSSION

The results obtained from the study have added to international research results that have been undertaken in recent years on the development of predictive models for determining the risk of HF in diabetic patients. The Action to Control Cardiovascular Risk in Diabetes trail analyzed risk factors for HF in patients with T2DM over a median follow-up of 4.9 years, identifying the main predictors of increased risk for cardiac events (body mass index (BMI), age, systolic and diastolic BP, creatinine, fasting plasma glucose, HDL, QRS complex duration, history of AMI, and coronary artery bypass grafting. After determining the predictors, a model was constructed to predict the risk of HF in T2DM patients in outpatient settings [14]. Another study has found higher levels of **NT-proBNP**, troponin T, and BMI as independent predictors of HF risk in T2DM patients prescribed SGLT2 inhibitors therapy. The model was designed to identify patients at high risk for HF [15]. The Strong Heart Study has identified BMI, smoking, glycated hemoglobin, albuminuria, and prior AMI as predictors of HF in populations at high risk of diabetes [16]. A similar cohort study was conducted to examine T2DM patients without HF. The model included 9 variables: age, blood urea nitrogen, coronary heart disease, atrial fibrillation, glycated hemoglobin, systolic BP, blood albumin, chronic kidney disease, and smoking history. High risk patients had a >5% probability of hospitalization for HF within 5 years [17].

Biomarkers of energy homeostasis have been examined demonstrating reduced serum adropin, CTRP3 and increased FABP4 levels in diabetic patients. Low adropin levels have been found in patients with prediabetes after STEMI, that could be a reason for including serum adropin levels in long-term prognosis for patients after STEMI [18]. The study has shown reduced serum CTRP3 levels in patients with reduced left ventricular ejection fraction, while patients with higher NYHA class have been revealed with lower CTRP3 levels [19]. Circulating FABP 4 levels have been found to be independently associated with hospitalization for HF in T2DM and potentially useful for risk stratification to prevent hospitalization for HF. The study has determined an association between FABP4 levels and the occurrence of HF in patients with T2DM, that has been considered significant in the stratification of HF risk [20].

The constructed statistical model with a high accuracy of 89.6% predicted the probability of developing NYHA functional class III HF within a year in diabetic patients after AMI based on the measurement data on the 1st and 10th day. Qualitative analysis of the coefficients for fixed GLMM factors has shown that the level of adropin on the 1st day was a strong negative prognostic factor, while the combined effect of adropin and FABP4 levels on the 10th day was a positive prognostic factor. The combined factor of the blood glucose level on the 1st and 10th day, as well as CTRP3 on the 1st day, has shown a negative effect. The overall accuracy of the model was 79.7%.

CONCLUSIONS

1. Energy imbalance has been revealed in diabetic patients based on the reduced levels of adropin and CTRP3 and increased levels of FABP4.

2. High prognostic values of the constructed model have been detected, since the predictive accuracy of developing NYHA functional class II HF within a year in diabetic patients after AMI was 61.5%, and NYHA functional class III HF – 89.6%. The overall accuracy of the model was 79.7%. The highest level of the model sensitivity has been identified specifically for the prediction of NYHA functional class III HF, indicating the prediction of worse HF course.

Perspectives for further research include the study on the association between energy metabolism biomarkers and the incidence of new adverse complications in diabetic patients after AMI in the long term.

COMPLIANCE WITH ETHICAL REQUIREMENTS

The study was approved by the Ethics and Bioethics Committee of Kharkiv National Medical University (protocol No. 3 of 02/11/22) and was conducted with the written voluntary consent of the study participants in accordance with the principles of bioethics defined in the Helsinki Declaration «Ethical Principles for Medical Research Involving Human Participants».

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AUTHOR CONTRIBUTIONS

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REFERENCES

1. Ho, K. L., Karwi, Q. G., Connolly, D., Pherwani, S., Ketema, E. B., Ussher, J. R., & Lopaschuk, G. D. (2022). Metabolic, structural and biochemical changes in diabetes and the development of heart failure. Diabetologia, 65(3), 411-423. https://doi.org/10.1007/s00125-021-05637-7
2. Nagoshi, T., Yoshimura, M., Rosano, G. M., Lopaschuk, G. D., & Mochizuki, S. (2011). Optimization of cardiac metabolism in heart failure. Current pharmaceutical design, 17(35), 3846-3853. https://doi.org/10.2174/138161211798357773
3. Rosano, G. M., Vitale, C., & Seferovic, P. (2017). Heart failure in patients with diabetes mellitus. Cardiac failure review, 3(1), 52-55. https://doi.org/10.15420/cfr.2016:20:2
4. Jenča, D., Melenovský, V., Stehlik, J., Staněk, V., Kettner, J., Kautzner, J., Adámková, V., & Wohlfahrt, P. (2021). Heart failure after myocardial infarction: incidence and predictors. ESC heart failure, 8(1), 222-237. https://doi.org/10.1002/ehf2.13144
5. Bibi, F., Aslam, A., Naseem, H., Khurshid, H., Haider, A., Ashraf, H., Rafaqat, S. (2025). Adipokines pathogenesis in heart failure. Cardiovascular innovations and applications, 10(1). https://doi.org/10.15212/CVIA.2024.0065
6. Berezina, T. A., Berezin, O. O., Hoppe, U. C., Lichtenauer, M., & Berezin, A. E. (2024). Adropin predicts asymptomatic heart failure in patients with type 2 diabetes mellitus independent of the levels of natriuretic peptides. Diagnostics (Basel, Switzerland), 14(16), 1728. https://doi.org/10.3390/diagnostics14161728
7. Arnold, S. V., Spertus, J. A., Jones, P. G., McGuire,D. K., Lipska, K. J., Xu, Y., Stolker, J. M., Goyal, A., & Kosiborod, M. (2016). Predicting adverse outcomes after myocardial infarction among patients with diabetes mellitus. Circulation. Cardiovascular quality and outcomes, 9(4), 372-379. https://doi.org/10.1161/CIRCOUTCOMES.115.002365
8. Halade, G. V., & Lee, D. H. (2022). Inflammation and resolution signaling in cardiac repair and heart failure. EBioMedicine, 79, 103992. https://doi.org/10.1016/j.ebiom.2022.103992
9. Davies, M. J., Aroda, V. R., Collins, B. S., Gabbay, R. A., Green, J., Maruthur, N. M., Rosas, S. E., Del Prato, S., Mathieu, C., Mingrone, G., Rossing, P., Tankova, T., Tsapas, A., & Buse, J. B. (2022). Management of hyperglycemia in type 2 diabetes, 2022. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes care, 45(11), 2753-2786. https://doi.org/10.2337/dci22-0034
10. Byrne, R. A., Rossello, X., Coughlan, J. J., Barbato, E., Berry, C., Chieffo, A., Claeys, M.J., Dan, G.-A., Dweck, M.R., Galbraith, M., Gilard, M., Hinterbuchner, L., Jankowska, E.A., Jüni, P., Kimura, T., Kunadian, V., Leosdottir, M., Lorusso, R., Pedretti, R.F.E., Rigopoulos, A.G., Rubini Gimenez, M., Thiele, H., Vranckx, P., Wassmann, S., Wenger, N.K., Ibanez, B., ESC Scientific Document Group. (2023). 2023 ESC Guidelines for the management of acute coronary syndromes: Developed by the task force on the management of acute coronary

syndromes of the European Society of Cardiology (ESC). European heart journal, 44(38), 3720-3826. https://doi.org/10.1093/eurheartj/ehad191

11. McDonagh, T. A., Metra, M., Adamo, M., Gardner, R. S., Baumbach, A., Böhm, M., Burri, H., Butler, J., Čelutkienė, J., Chioncel, O., Cleland, J. G. F., Crespo-Leiro, M. G., Farmakis, D., Gilard, M., Heymans, S., Hoes, A. W., Jaarsma, T., Jankowska, E. A., Lainscak, M., Lam, C. S. P., … ESC Scientific Document Group (2023). 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. European heart journal, 44(37), 3627-3639. https://doi.org/10.1093/eurheartj/ehad195
12. Heidenreich, P.A., Bozkurt, B., Aguilar, D., Allen, L.A., Byun, J.J., Colvin, M.M., Deswal, A., Drazner, M.H., Dunlay, S.M., Evers, L.R., Fang, J.C., Fedson, S.E., Fonarow, G.C., Hayek, S.S., Hernandez, A.F., Khazanie, P., Kittleson, M.M., Lee, C.S., Link, M.S., Milano, C.A., Nnacheta, L.C., Sandhu, A.T., Stevenson, L.W., Vardeny, O., Vest, A.R., Yancy, C.W. (2022). 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation, 145, 895-1032. https://doi.org/10.1161/CIR.0000000000001063
13. Stroup, W.W. (2012). Generalized Linear Mixed Models: Modern Concepts, Methods and Applications: CRC Press., 555.
14. Segar, M. W., Vaduganathan, M., Patel, K. V., McGuire, D. K., Butler, J., Fonarow, G. C., Basit, M., Kannan, V., Grodin, J. L., Everett, B., Willett, D., Berry, J., & Pandey, A. (2019). Machine learning to predict the risk of incident heart failure hospitalization among patients with diabetes: the WATCH-DM Risk Score. Diabetes care, 42(12), 2298-2306. https://doi.org/10.2337/dc19-0587
15. Said, F., Arnott, C., Voors, A. A., Heerspink, H. J. L., & Ter Maaten, J. M. (2024). Prediction of new-onset heart failure in patients with type 2 diabetes derived from ALTITUDE and CANVAS. Diabetes, obesity & metabolism, 26(7), 2741-2751. https://doi.org/10.1111/dom.15592
16. Martinez-Morata, I., Domingo-Relloso, A., Zhang, Y., Fretts, A. M., Pichler, G., Garcia Pinilla, J. M., Umans, J. G., Cole, S. A., Sun, Y., Shimbo, D., Navas-Acien, A., & Devereux, R. B. (2024). Heart failure risk prediction in a population with a high burden of diabetes: evidence from the strong heart study. Journal of the American Heart Association, 13(17), e033772. https://doi.org/10.1161/JAHA.123.033772
17. Williams, B. A., Geba, D., Cordova, J. M., & Shetty, S. S. (2020). A risk prediction model for heart failure hospitalization in type 2 diabetes mellitus. Clinical cardiology, 43(3), 275-283. https://doi.org/10.1002/clc.23298
18. Berezina, T. A., Berezin, O. O., Hoppe, U. C., Lichtenauer, M., & Berezin, A. E. (2024). Low levels of adropin predict adverse clinical outcomes in outpatients with newly diagnosed prediabetes after acute myocardial infarction. Biomedicines, 12(8), 1857. https://doi.org/10.3390/biomedicines12081857
19. Gao, C., Zhao, S., Lian, K., Mi, B., Si, R., Tan, Z., Fu, F., Wang, S., Wang, R., Ma, X., & Tao, L. (2019). C1q/TNF-related protein 3 (CTRP3) and 9 (CTRP9) concentrations are decreased in patients with heart failure and are associated with increased morbidity and mortality. BMC cardiovascular disorders, 19(1), 139. https://doi.org/10.1186/s12872-019-1117-0
20. Lee, C. H., Kan, A. K. C., Lui, D. T. W., Fong, C. H. Y., Chan, D. S. H., Yuen, M. M. A., Chow, W. S., Woo, Y. C., Xu, A., & Lam, K. S. L. (2021). Prospective association of serum adipocyte fatty acid-binding protein with heart failure hospitalization in diabetes. ESC heart failure, 8(5), 3964-3974. https://doi.org/10.1002/ehf2.13472

Резюме

МОДЕЛЬ ПРОГНОЗУВАННЯ ХРОНІЧНОЇ СЕРЦЕВОЇ НЕДОСТАТНОСТІ У ПАЦІЄНТІВ ІЗ ЦУКРОВИМ ДІАБЕТОМ 2 ТИПУ

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Вступ. Однією із частих несприятливих ускладнень, що може бути у хворих із цукровим діабетом (ЦД) 2 типу на тлі перенесеного гострого інфаркту міокарда (ГІМ) є розвиток та прогресування хронічної серцевої недостатності (ХСН). Нині залишається актуальним вивчення біомаркерів енергетичного гомеостазу, які беруть участь у патогенезі розвитку ХСН у хворих із ЦД 2 типу.
Мета. Спрогнозувати розвиток ХСН у хворих із ЦД 2 типу за допомогою узагальненої лінійної змішаної моделі (GLMM).
Матеріали та методи. Обстежено 74 хворих із ЦД 2 типу після перенесеного ГІМ із елевацією сегменту ST. Вміст адропіну, білку, що зв'язує жирні кислоти 4 (FABP 4) і C1q/TNF-зв'язуючий білок 3 (CTRP3) виявлено за допомогою імуноферментного методу. Рівень глюкози в крові визначався глюкооксидантним методом натщесерце. Рівень ліпопротеїдів високої щільності (ЛПВЩ) сироватки крові аналізували пероксидазним ферментативним методом. Вміст ліпопротеїдів низької щільності (ЛПНЩ) оцінювали за формулою Фрідевальда. Для побудови прогностичної моделі ХСН у хворих із ЦД 2 типу застосували GLMM.
Результати. У дослідженні визначено зниження рівнів адропіну і CTRP 3 та збільшення вмісту FABP 4 у хворих із ЦД 2 типу після STEMI. Розроблена прогностична модель розвитку ХСН у хворих із ЦД 2 типу. Фіксовані ефекти моделі наведені двома однофакторними (адропін та CTRP 3 на 1 добу) та двома двофакторними показниками (адропін і FABP 4 на 10 добу, глюкоза на 1 і 10 добу), а випадкові ефекти – п'ятьма однофакторними показниками (гостра серцева недостатність, діастолічний артеріальний тиск на 1 добу, пульс на 10 добу, ЛПНЩ на 1 добу та ЛПВЩ на 1 добу). Точність передбачення виникнення протягом року II функціонального класу за NYHA у хворих із ЦД 2 типу склала 61,5%, тоді як передбачення III функціонального класу за NYHA – 89,6%. Загальна точність моделі становила 79,7%.
Висновки. Отже, рівень адропіну на 1 добу виступає сильним негативним прогностичним фактором, а спільний вплив рівнів адропіну і FABP 4 на 10 добу – позитивним прогностичним фактором. Спільний вплив рівнів глюкози в крові на 1 і 10 добу, а також вміст CTRP 3 на 1 добу – дають негативний ефект. Застосування у моделі біомаркерів енергетичного обміну вказує на важливість вивчення їх у хворих із ЦД 2 типу.

Ключові слова: **енергетичний обмін, серцева недостатність, інфаркт міокарда, цукровий діабет 2 типу**

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