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DIAGNOSTIC AND PROGNOSTIC VALUE OF β2-MICROGLOBULIN IN THE DEVELOPMENT OF CARDIORENAL SYNDROME IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ARTERIAL HYPERTENSION*

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Arterial hypertension (AH) is the largest pandemic in human history, determining the structure of cardiovascular morbidity and mortality. Meanwhile, there is a clear correlation between AH and various pathological conditions and diseases that largely determine its progression and contribute to the development of cardiovascular complications. Such diseases include type 2 diabetes mellitus (T2DM), which is currently pandemic in nature.

These conditions are determined by many factors, including the development of coronary heart disease and its clinical specifics, the presence of risk factors, patient adherence to treatment, and many others, such as renal dysfunction, which is widespread among these categories of patients.

Concomitant heart and kidney disorders involve common pathogenesis, shared risk factors, and mutual burden. This interaction is called cardiorenal syndrome (CRS) [1–3]. This syndrome has been long studied by clinicians of various specialties and has gained relevance in the last decade [4–7].

Thus, a decrease in glomerular filtration rate below 30 ml/min/1.73 m^2 increases cardiovascular risk by 5.5 times [8, 9].

That is why diagnosing CRS at the earliest possible stage with the most modern diagnostic capabilities will enable us to initiate the necessary treatment promptly, prevent the development of complications, and reduce the mortality rate of such patients.

The only way to mitigate the likelihood of chronic kidney disease and acute renal failure, as well as the risks of cardiovascular complications associated with this disease, is to provide timely diagnosis and treatment.

Back in 1968, it was reported on the first results of studies on ß2-microglobulin (ß2-M), be-

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ing a low molecular weight ß-2-globulin with a mass of 11815 kDa, extracted from the urine of patients with tubular dysfunction [10]. Further studies have shown that ß2-M is a component of the light chain of the Major Histocompatibility Complex I (MHC I), which is found on the surface of all nucleated cells in the human body, except for red blood cells. What is significant is that the molecular weight of ß2-M depends on the glycosylation level. The gene for human leukocyte antigen ß2-M is located on chromosome 15. As shown by the studies, ß2-M is adjacent to the α-3 chain on the cell surface, but there is no transmembrane domain [11].

Adults maintain a constant rate of ß2-M secretion. It is excreted by the kidneys, where it undergoes filtration and then almost complete reabsorption and catabolism in the proximal tubules. It has been found that the ß2-M levels in blood and urine are independent of muscle condition, food intake, gender, and age [12]. A healthy person's ß2-M level depends only on the glomerular filtration rate and tubular reabsorption. According to several studies, ß2-M levels in the blood mainly reflect cell turnover and lymphocyte proliferation [13, 14]. ß2-M synthesis increases in various conditions associated with increased immune system activity, different types of inflammation, autoimmune diseases, viral infections, etc. There is evidence of the assessment of renal function and cardiovascular risk based on ß2-M [15, 16]. Recent studies have focused on the role of ß2-M in the development of cardiometabolic and renal disorders [17–19].

Therefore, it is of major scientific interest to determine the contribution of the ß2-M biomarker to the development of early nephrological complications in patients with T2DM and AH.

This study was aimed at evaluating the role of ß2-M in the development of renal complications in patients with type 2 diabetes mellitus and arterial hypertension.

MATERIALS AND METHODS

The set of studies was conducted according to the ethical and moral requirements of the Ukrainian Association for Bioethics and the Standards of GCP (1992), GLP (2002), the principles of the Declaration of Helsinki on Human Rights, the Council of Europe Convention on Human Rights and Biomedicine and approved by the Ethics and Bioethics Committee of Kharkiv National Medical University.

90 patients with AH (men/women — 48/42) and 20 control subjects were examined. All patients with AH aged 54.37±1.18 were treated at the clinic of the Government Institution «L. T. Malaya Therapy National Institute of the National Academy of Medical Sciences of Ukraine». During a thorough examination and follow-up of patients, they were classified into 3 groups: patients with AH — group 1 — 31 people; group 2 — AH in combination with T2DM — 31 people; group 3 — patients with AH, T2DM, and obesity — 28 people.

Body weight and height were measured in all patients, and BMI = body weight/height^2 (m^2) was calculated. Systolic and diastolic blood pressure were measured as well.

ß2-M levels in the patients' serum were determined by enzyme-linked immunosorbent assay using a Labline-90 analyzer (Austria) and a commercial test system manufactured by Orgentiec (ELISA, Germany) according to the manufacturer's instructions.

Cardiotrophin-1 (CTF-1), catestatin (CST), leptin, cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), N-terminal brain natriuretic peptide (NT-proBNP), 25-OH total vitamin D (Vitamin D3), and serum insulin levels were measured by enzyme-linked immunosorbent assay using a Labline-90 analyzer (Austria) with commercial test systems manufactured by Fine Test (ELISA, China), BT LAB (ELISA, China), DBC (ELISA, China), Elabscience (ELISA, Canada), Monobind Inc. (ELISA, USA), Orgentiec (ELISA, Germany) according to the manufacturer's instructions.

Biochemical studies (serum creatinine, urea, lipid spectrum, glycosylated hemoglobin) were performed using a Labline-90 analyzer (Austria). Serum urea levels were measured by the kinetic, enzymatic method with urease-glutamic dehydrogenase using Liquick Cor-UREA 30 kits (Cormay, Poland) according to the manufacturer's instructions. Serum creatinine levels were measured by the modification of the Jaffe method without deproteinization

using Liquick Cor-CREATININ 30 reagent kits (Poland) according to the manufacturer's instructions. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were determined by the enzymatic method using reagent kits Cholesterol liquicolor, HDL-C, and Triglycerides liquicolor (Human, Germany) according to the manufacturer's instructions. The level of very low-density lipoprotein cholesterol (VLDL-C) was calculated using the TG/2.22 formula; the level of low-density lipoprotein cholesterol (LDL-C) was calculated using the W. T. Friedewald formula, 2004:

$$LDL\text{-}C = TC - (HDL\text{-}C + TG / 2.22), \text{ mmol/L}.$$

The following were the exclusion criteria for the study: Type 1 diabetes mellitus, congenital heart and urinary tract defects, artificial pacemakers, artificial heart valves, stage II B and III heart failure, acute heart attack, infectious and severe inflammatory processes, and hematological diseases.

Each patient underwent stable antihypertensive and glucose-lowering therapy, which was not changed during the course of this study.

Statistical data analysis was performed using Statistica, 12 (Stat Soft Inc, USA) and Microsoft Office Excel 2013. The data are presented as mean (M) and standard deviation (δ). Differences between groups of mean values were evaluated using the Student's t-test. Mathematical models were built using Spearman's rank correlation coefficient to determine the closeness (strength) and direction of the correlation between two features using the IBM SPSS Statistics analysis package.

RESULTS AND THEIR DISCUSSION

Our patient groups were representative in terms of age, systolic, and diastolic blood pressure. When comparing the study groups of patients, no significant differences were found in urea, creatinine, and lipid metabolism components. Patients with T2DM had a significant difference ($p < 0.05$) in carbohydrate metabolism (HbA1c, insulin) from those in other groups. Obese patients differed significantly from other groups in terms of BMI and body weight (Table 1).

The ß2-M level in the observed groups of patients differed significantly from that of healthy subjects (Fig. 1).

Table 1

Clinical profile of the examined patients

Parameter, unit	Group 1 n = 31	Group 2 n = 31	Group 3 n = 28	Control group n = 20
Age, year	54.37 ± 8.25*	55.89 ± 7.65*	56.20 ± 7.18*	42.45 ± 7.19
Body weight, kg	79.65 ± 7.72*	84.44 ± 9.00*	102.27 ± 6.93*	70.05 ± 7.55
BMI, kg/m^2	26.81 ± 1.44*	28.25 ± 1.61*	35.45 ± 2.73*	24.09 ± 0.59
SBP, mm Hg	143.29 ± 11.53*	146.39 ± 13.15*	147.69 ± 11.01*	119.25 ± 2.94
DBP, mm Hg	87.63 ± 8.38*	90.14 ± 7.67*	89.16 ± 10.27*	75.50 ± 4.26
Creatinine, μmol/L	92.25 ± 14.60*	96.64 ± 16.55*	93.10 ± 15.50*	86.20 ± 11.19
Urea, mmol/L	5.82 ± 1.80	5.68 ± 1.80	6.18 ± 2.32	5.68 ± 1.05
TC, mmol/L	5.52 ± 1.25*	5.21 ± 1.37*	5.68 ± 1.28*	4.26 ± 1.06
HDL-C, mmol/L	1.20 ± 0.40	1.25 ± 0.41	1.28 ± 0.31	1.43 ± 0.35
TG, mmol/L	1.90 ± 0.95*	1.83 ± 0.99*	2.08 ± 1.06*	1.37 ± 0.62
VLDL-C, mmol/L	0.91 ± 0.95*	0.95 ± 0.57*	0.92 ± 0.47*	0.61 ± 0.28
LDL-C, mmol/L	3.52 ± 1.23*	3.26 ± 1.36*	3.47 ± 1.25*	2.21 ± 0.74
Insulin, mIU/L	15.75 ± 7.50*	21.44 ± 16.55*	20.06 ± 11.60*	14.38 ± 2.69
HbA1c, %	5.85 ± 1.64	7.46 ± 1.2*	7.12 ± 0.83*	4.80 ± 0.32

Note:

* $p < 0.05$ relative to the control group.

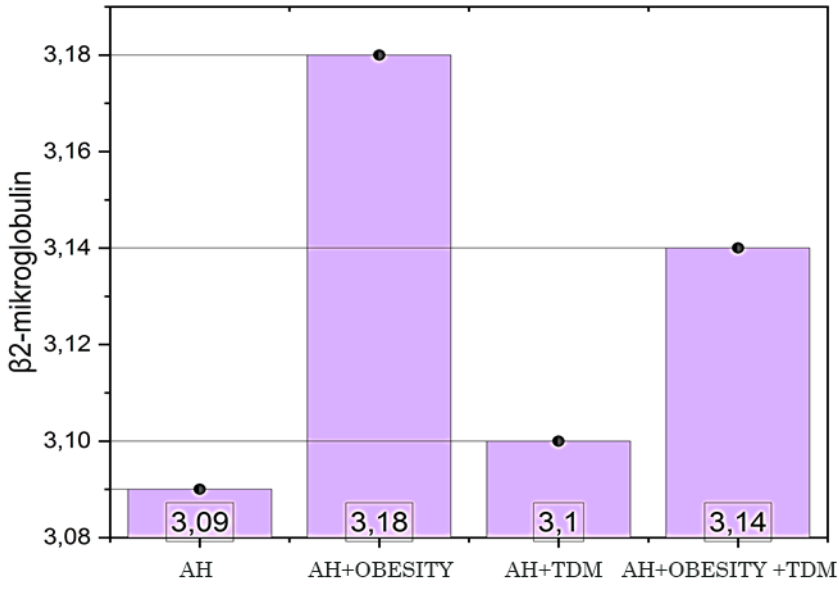

Fig. 1. ß2-M level (µg/mL) in patients with hypertension depending on the functional state of the kidneys. ß2-M values in the groups compared with healthy subjects are significant (p < 0.001).

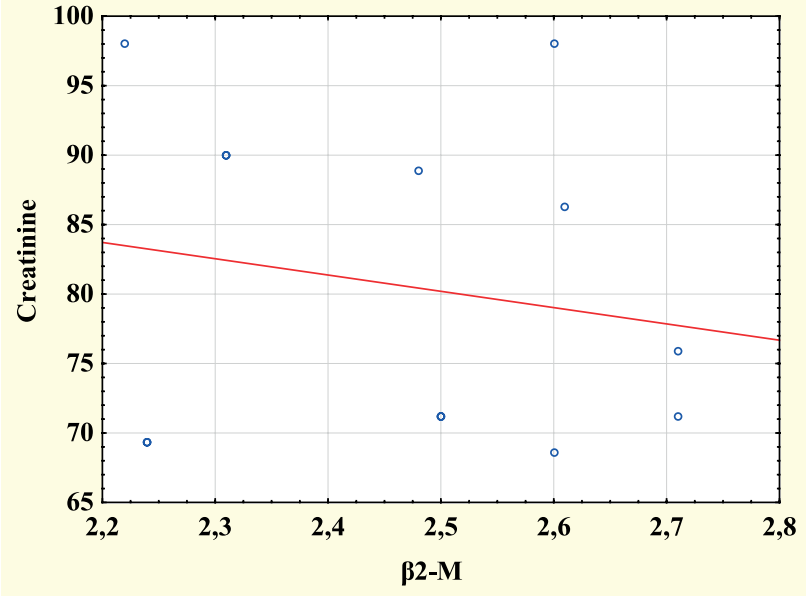

Fig. 2. Mathematical model of Spearman's rank correlation between creatinine and ß2-M (group of patients with ß2-M < 3.08 µg/mL), r = – 0.124; p = 0.016.

Thus, ß2-M level in patients with AH was 3.09 ± 1.08, in patients with AH and T2DM it was 3.10 ± 0.97, in patients with AH + T2DM + obesity it was 3.14 ± 0.84, in healthy subjects it was 1.98 ± 0.52. p < 0.001.

Therefore, a significant increase (p < 0.001) in the concentration of ß2-M in the blood serum was noted in patients of different groups compared with the control group. An increase in ß2-M concentration allowed us to confirm the presence of tubular renal dysfunction, which was not diagnosed by conventional methods. All examined patients were divided into 2 groups depending on the ß2-M level (Table 2). The first group included patients with a serum ß2-M level < 3.08 µg/mL, and the second group included patients with a ß2-M level > 3.08 µg/mL. Patients of both groups significantly differed in creatinine (p = 0.01), urea (p = 0.029), VLDL-C (p = 0.028), AC (p = 0.022), CTF-1 (p = 0.023), and CST (p = 0.018). Our data prove the role of ß2-M as an independent biomarker of renal dysfunction, as well as the development of early cardiovascular complications.

Using the Spearman's rank correlation coefficient to determine the closeness (strength) and direction of the correlation between two features using the IBM SPSS Statistics analysis package (group of patients with β2-M less than 3.08 μg/mL and patients with β2-M grea-

Table 2

Changes in laboratory parameters depending on the β2-M level in patients with AH

Parameter, unit	β2-M level less than 3.08, μg/mL	β2-M level greater than 3.08, μg/mL	p
Age, year	55.0 ± 1.31 [47.50; 60.50]	58.00 ± 1.24 [47.00; 62.00]	0.023
IMT, kg/m^2	32.28 ± 0.60 [28.62; 35.83]	30.59 ± 0.57 [27.36; 34.54]	0.041
SBP, mmHg	145.0 ± 1.58 [140.0; 155.0]	145.0 ± 1.63 [134.0; 156.0]	0.038
DBP, mmHg	90.0 ± 1.30 [82.0; 91.0]	90.0 ± 1.07 [85.0; 100.0]	0.020
Creatinine, μmol/L	88.90 ± 2.04 [79.42; 94.35]	93.99 ± 2.04 [86.72; 106.50]	0.010
Urea, mmol/L	5.64 ± 0.19 [4.81; 6.55]	5.94 ± 0.28 [4.45; 7.38]	0.029
TC, mmol/L	5.60 ± 0.21 [4.55; 6.70]	5.87 ± 0.16 [5.20; 6.60]	0.033
HDL-C, mmol/L	1.30 ± 0.05 [1.10; 1.50]	1.30 ± 0.04 [1.00; 1.50]	0.026
TG, mmol/L	1.75 ± 0.12 [1.30; 2.35]	1.70 ± 0.17 [1.50; 2.30]	0.015
VLDL-C, mmol/L	0.80 ± 0.05 [0.60; 1.00]	0.80 ± 0.06 [0.60; 1.00]	0.028
LDL-C, mmol/L	3.60 ± 0.19 [47.50; 60.50]	3.70 ± 0.15 [2.80; 4.30]	0.013
AC	3.31 ± 0.21 [2.16; 4.24]	3.86 ± 0.17 [2.82; 4.78]	0.022
CTF-1, pmol/L	1145.04 ± 33.64 [870.68; 1225.19]	1148.75 ± 32.33 [889.45; 1239.05]	0.023
CST, ng/mL	2.64 ± 0.16 [2.33; 3.44]	2.34 ± 0.14 [2.09; 2.83]	0.018
Cystatin C, mg/L	144.61 ± 5.64 [104.80; 167.73]	139.69 ± 6.49 [84.94; 175.61]	0.033
Leptin, ng/mL	27.40 ± 1.93 [18.55; 40.56]	25.55 ± 1.50 [16.58; 32.97]	0.041
NGAL, ng/mL	19.11 ± 0.68 [14.61; 23.34]	19.45 ± 0.85 [14.86; 23.15]	0.028
NT-proBNP, pg/mL	491.64 ± 22.01 [343.28; 602.11]	456.12 ± 23.41 [372.47; 625.00]	0.033
Insulin, pmol/L	15.29 ± 1.08 [9.70; 21.68]	13.27 ± 1.18 [8.61; 20.04]	0.016
Vitamin D3, ng/mL	39.68 ± 1.56 [30.59; 47.06]	39.96 ± 1.64 [31.82; 49.05]	0.014
HbA1c, %	6.05 ± 0.14 [5.45; 7.08]	6.04 ± 0.13 [5.40; 6.91]	0.022

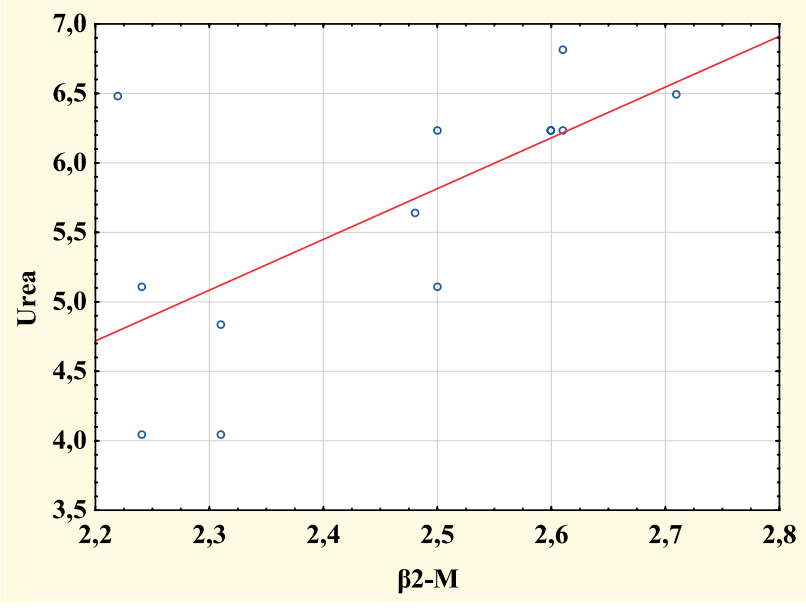

Fig. 3. Mathematical model of Spearman's rank correlation between urea and ß2-M (group of patients with ß2-M < 3.08 µg/ml), r = 0.620; p = 0.024.

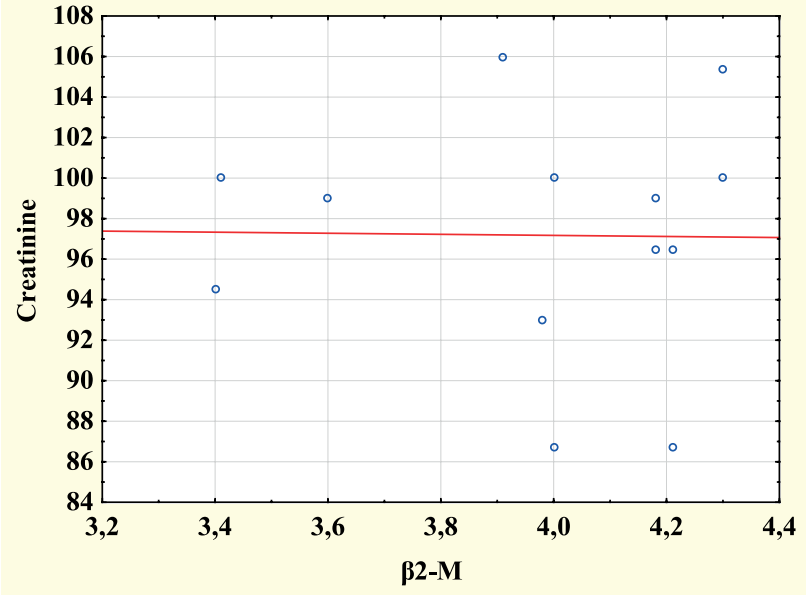

Fig. 4. Mathematical model of Spearman's rank correlation between creatinine and ß2-M (group of patients with ß2-M > 3.08 µg/mL), r = – 0.571; p = 0.010.

ter than 3.08 µg/mL), mathematical models were built between creatinine and ß2-M, urea and ß2-M (Fig. 2, 3, 4, 5).

The data obtained prove a highly significant correlation between ß2-M and renal function and demonstrate that ß2-M is an independent factor in the prediction of renal dysfunction in patients with AH and concomitant T2DM.

Nowadays, researchers acknowledge the role of ß2-M in the development of cardiometabolic conditions. For example, a systematic meta-analysis [20] demonstrated the correlation of ß2-M levels with cardiovascular disease and overall mortality.

National scientists have concluded that the factors associated with an increased risk of developing CRS in patients with T2DM and chronic heart failure include the age of over 55 years and a body mass index over 32 kg/m^2, respectively ($p < 0.05$). A significant inverse correlation was found between the functional renal condition and body weight in patients with heart failure ($r = – 0.32$; $p < 0.05$) [21].

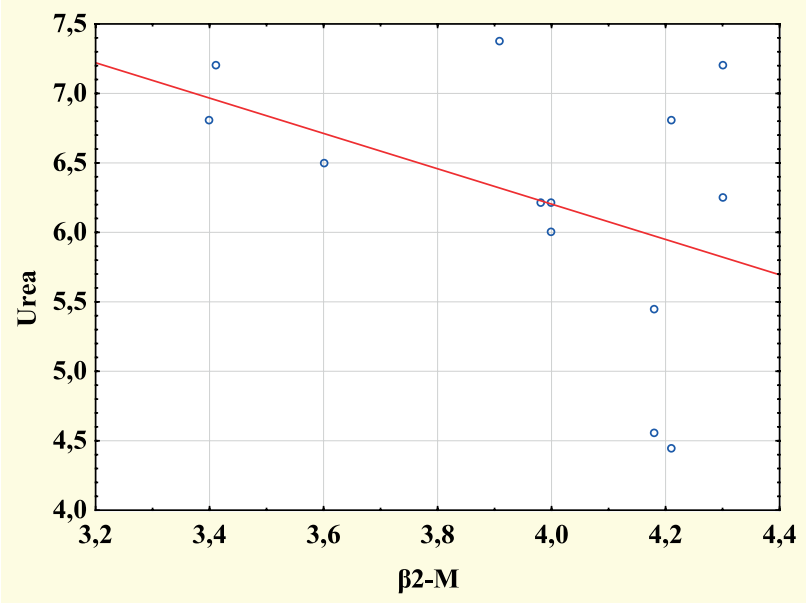

Fig. 5. Mathematical model of Spearman's rank correlation between urea and ß2-M (group of patients with ß2-M > 3.08 μg/mL), r = 0.506; p = 0.018.

Moreover, there is evidence of an association between sodium metabolism disorders and the development of cardiorenal syndrome in patients with chronic heart failure [22].

In summary, our study revealed the diagnostic and prognostic significance of assessing the ß2-M level in the blood serum, regardless of the presence of renal complications. Studying ß2-M in patients with a high risk of developing cardiorenal syndrome may delay the onset and progression of chronic kidney disease, as well as stratify nephrological and cardiovascular risks.

CONCLUSIONS

1. ß2-microglobulin is an early independent biomarker of renal dysfunction, namely, renal tubular dysfunction in patients with hypertension and type 2 diabetes mellitus.
2. A significant increase in the level of ß2-microglobulin in patients with hypertension and concomitant type 2 diabetes mellitus and obesity is accompanied by a notable increase in markers of early cardiovascular disorders, which suggests its involvement in these processes.
3. It is advisable to determine ß2-microglobulin in the diagnosis and prognosis of early manifestations of cardiorenal syndrome.

REFERENCES

1. Chung ES, Meyer TE. *J Card Fail* 2015;21(2): 116-118. doi: 10.1016/j.cardfail.2014.11.011.
2. De Vecchis R, Baldi C. *Ther Clin Risk Manag* 2014;10: 949-961. doi: 10.2147/TCRM.S63255.
3. Demesova E, Goncalvesova E, Slezak P, Pontuch P. *Bratisl Lek Listy* 2015;116(2): 83-87. doi: 10.4149/bll_2015_015.
4. Iacoviello M, Leone M, Antoncecchi V, Ciccone MM. *World J Clin Cases* 2015;3(1): 10-19. doi: 10.12998/wjcc.v3.i1.10.
5. Ricci Z, Romagnoli S, Ronco C. *Crit Care Clin* 2021; 37(2): 335-347. doi: 10.1016/j.ccc.2020.11.003.
6. Zhang Y, Jiang Y, Yang W, et al. *Front Cardiovasc Med* 2021;8: 639959. doi: 10.3389/fcvm.2021.639959.
7. Núñez-Marín G, Martín-Sánchez FJ, Bonanad C. *Rev Esp Geriatr Gerontol* 2021;56(1): 1-2. doi: 10.1016/j.regg.2020.10.003.
8. García-Carrasco A, Izquierdo-Lahuerta A, Medina-Gómez G. *Nephron* 2021;145(6): 604-608. doi: 10.1159/000515419.
9. Wikstrom J, Liu Y, Whatling C, et al. *J Diabetes Complications* 2021;35(4): 107881. doi: 10.1016/j.jdiacomp.2021.107881.
10. Berggård I, Bearn AG. *J Biol Chem* 1968;243(15): 4095-4103.
11. Feder JN, Penny DM, Irrinki A, et al. *Proc Natl Acad Sci U S A* 1998;95(4): 1472-1477. doi: 10.1073/pnas.95.4.1472.

12. Argyropoulos CP, Chen SS, Ng YH, et al. *Front Med (Lausanne)* 2017;4: 73. doi: 10.3389/fmed.2017.00073.

13. Chen H, Li H. *Clin Lab* 2017;63(2): 241-247. doi: 10.7754/Clin.Lab.2016.160719.

14. Ekrikpo UE, Effa EE, Akpan EE, et al. *Int J Nephrol* 2017;2017: 4093171. doi: 10.1155/2017/4093171.

15. Zhang WR, Parikh CR. *Annu Rev Physiol* 2019;81: 309-333. doi: 10.1146/annurev-physiol-020518-114605.

16. Han X, Zhang S, Chen Z, et al. *Clin Chim Acta* 2020; 510: 298-310. doi: 10.1016/j.cca.2020.07.040.

17. Yılmaz A, Yılmaz B, Küçükseymen S. *Anatol J Cardiol* 2016;16(11): 844-849. doi: 10.14744/AnatolJCardiol.2015.6691.

18. Rysz J, Gluba-Brzózka A, Franczyk B, et al. *Int J Mol Sci* 2017;18(8): 1702. doi: 10.3390/ijms18081702.

19. Yanai H, Adachi H, Hakoshima M, Katsuyama H. *Int J Mol Sci* 2021;22(17): 9221. doi: 10.3390/ijms22179221.

20. Shi F, Sun L, Kaptoge S. *Atherosclerosis* 2021;320: 70-78. doi: 10.1016/j.atherosclerosis.2021.01.018.

21. Lazidi EL, Rudyk IuS. UZhMBS 2019;4(5): 142-148. doi: 10.26693/jmbs04.05.142

22. Kovalevskaya LA, Zagorodnyaya LI. *Topical issues of pharmaceutical and medical science and practice* 2015(2). doi: 10.14739/2409-2932.2015.2.45201

DIAGNOSTIC AND PROGNOSTIC VALUE OF β2-MICROGLOBULIN IN THE DEVELOPMENT OF CARDIORENAL SYNDROME IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND ARTERIAL HYPERTENSION

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Arterial hypertension (AH) is the largest pandemic in the history of mankind, which defines the structure of cardiovascular morbidity and mortality. Currently, the correlation between hypertension and various pathological conditions and diseases has been proven, which largely determines its progression and contributes to the development of cardiovascular and renal complications. These diseases include type 2 diabetes mellitus, which is spreading globally as a pandemic. The history of arterial hypertension in combination with type 2 diabetes mellitus is often accompanied by the development of cardiorenal syndrome, so it is crucial to diagnose renal dysfunction and prevent cardiovascular complications in such patients. One of these biomarkers is ß2-microglobulin, which depends on the glomerular filtration rate and tubular reabsorption. This study was **aimed** at evaluating the role of ß2-microglobulin in the development of renal complications in patients with type 2 diabetes mellitus and arterial hypertension.

Materials and methods. 90 patients with AH (men/women — 48/42) and 20 control subjects were examined. During a thorough examination and follow-up of patients, they were classified into 3 groups: patients with AH — group 1 — 31 people; group 2 — AH in combination with T2DM — 31 people; group 3 — patients with AH, T2DM, and obesity — 28 people. Body weight and height were measured in all patients, and BMI was calculated. The ß2-microglobulin (ß2-M) levels in the patients' serum, cardiotrophin-1, catestatin, leptin, cystatin C, neutrophil gelatinase-associated lipocalin, and N-terminal pro-brain natriuretic peptide, 25-OH total vitamin D, serum insulin levels, glycosylated hemoglobin, lipid metabolism, and systolic and diastolic blood pressure were measured. Statistical data analysis was performed using Statistica, v.12 (Stat Soft Inc, USA) and Microsoft Office Excel 2013. The data are presented as mean (M) and standard deviation (δ). Differences between groups of mean values were evaluated using the Student's t-test.

Results and conclusions. The level of ß2-M in the observed groups of patients differed significantly from that of healthy individuals. An increase in ß2-M concentration allowed us to confirm the presence of tubular renal dysfunction, which was not diagnosed by conventional methods. All examined patients were divided into 2 roups depending on the ß2-M level. Our data prove the role of ß2-microglobulin as an independent biomarker of renal dysfunction, as well as the development of early cardiovascular complications. The data obtained from the mathematical models between creatinine and ß2-microglobulin, urea and ß2-microglobulin levels show a highly significant correlation between ß2-microglobulin and renal function and demonstrate that ß2-microglobulin is an independent factor in the prediction of renal dysfunction in patients with hypertension and concomitant type 2 diabetes mellitus. ß2-microglobulin is a biomarker that should be used in the diagnosis and prognosis of early manifestations of cardiorenal syndrome.

Keywords: arterial hypertension, type 2 diabetes mellitus, ß2-microglobulin, cardiorenal syndrome.

ДІАГНОСТИЧНЕ І ПРОГНОСТИЧНЕ ЗНАЧЕННЯ β2-МІКРОГЛОБУЛІНУ В РОЗВИТКУ КАРДІОРЕНАЛЬНОГО СИНДРОМУ У ХВОРИХ НА ЦУКРОВИЙ ДІАБЕТ 2 ТИПУ З АРТЕРІАЛЬНОЮ ГІПЕРТЕНЗІЄЮ

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Найбільшою пандемією в історії людства, що визначає структуру серцево-судинної захворюваності і смертності, є артеріальна гіпертензія. На сьогоднішній час доведено взаємозв'язок артеріальної гіпертензії з різними патологічними станами і захворюваннями, які багато в чому визначають її прогресування та сприяють розвитку серцево-судинних та ниркових ускладнень. До таких захворювань належить цукровий діабет 2 типу (ЦД2), розповсюдження на який в світі натепер також має пандемічний характер. Перебіг артеріальної гіпертензії в сполученні з ЦД2 часто супроводжується розвитком кардіоренального синдрому, тому дуже важливим є рання діагностика порушень функції нирок і попередження ускладнень серцево-судинної системи у таких пацієнтів. Одним з таких біомаркерів є β2-мікроглобулін (β2-М), показник якого залежить від швидкості клубочкової фільтрації і канальцевої реабсорбції. Метою даного дослідження було оцінити роль β2-мікроглобуліну в розвитку ниркових ускладнень у хворих на цукровий діабет 2 типу та артеріальну гіпертензію.

Матеріали і методи. Обстежено 90 хворих на АГ (чоловіків/жінок — 48/42) та 20 осіб контрольної групи. В процесі ретельного обстеження і нагляду за пацієнтами вони були розподілені на 3 групи: хворі на АГ — 1 група — 31 особа; 2 група — АГ в сполученні з ЦД2 — 31 особа; 3 група — пацієнти з АГ, ЦД 2 типу та ожирінням — 28 осіб. У всіх пацієнтів вимірювали масу тіла, зріст, розраховували ІМТ. У сироватці крові пацієнтів визначали рівні β2-мікроглобуліну, кардіотрофіну-1, катестатину, лептину, цистатину С, ліпокаліну, асоційованого з желатіназою нейтрофілів, N-термінального мозкового натрійуретичного пептиду, 25-ОН загального вітаміну D, інсуліну, глікозильованого гемоглобіну, показники ліпідного обміну, вимірювали рівень систолічного та діастолічного артеріального тиску. Статистичний аналіз даних виконано за допомогою пакету статистичних програм Statistica, 12 (Stat Soft Inc, США), Microsoft Office Excel 2013. Дані представлені у вигляді середнього значення (М) та стандартного відхилення (δ). Відмінності між групами середніх величин оцінювали за допомогою критерію Стьюдента.

Результати та висновки. У спостережених груп хворих рівень β2-М значуще відрізнявся від показників здорових осіб. Збільшення концентрації β2-М дозволило констатувати наявність тубулярної дисфункції нирок, яку не було діагностовано за допомогою загальноприйнятих методів. Окремо всіх обстежених пацієнтів було розподілено на 2 групи залежно від рівня β2-мікроглобуліну. Отримані нами дані доводять роль β2-мікроглобуліну як незалежного біомаркеру порушень ренальної функції, а також розвитку ранніх серцево-судинних ускладнень. Отримані дані внаслідок побудування математичних моделей між рівнем креатиніну та β2-мікроглобуліну, сечовини та β2-мікроглобуліну засвідчують високо значущий зв'язок β2-мікроглобуліну з показниками ниркової функції, а також демонструють, що β2-мікроглобулін є незалежним фактором прогнозу порушень ренальної функції у хворих на артеріальну гіпертензію з супутнім цукровим діабетом 2 типу. β2-мікроглобулін є біомаркером, який доцільно використовувати в діагностиці та прогнозуванні ранніх проявів розвитку кардіоренального синдрому.

Ключові слова: артеріальна гіпертензія, цукровий діабет 2 типу, β2-мікроглобулін, кардіоренальний синдром.