Kardiologia Polska 2017; 75, 2: 157–163; DOI: 10.5603/KP.a2016.0147 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Basic laboratory parameters as predictors of in-hospital death in patients with acute decompensated heart failure: data from a large single-centre cohort

Małgorzata Ostrowska[1], Adam Ostrowski[2], Małgorzata Łuczak[3], Miłosz Jaguszewski[4], Piotr Adamski[1], Jerzy Bellwon[4], Andrzej Rynkiewicz[5], Marcin Gruchała[4]

[1]Department of Principles of Clinical Medicine, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, Poland
[2]Department of General and Oncological Urology, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, Poland
[3]Department of Anaesthesiology and Intensive Therapy, F. Ceynowa Specialist Hospital, Wejherowo, Poland
[4]1st Department of Cardiology, Medical University of Gdansk, Gdansk, Poland
[5]Department of Cardiology and Cardiosurgery, University of Warmia and Mazury, Olsztyn, Poland

Abstract

Background: Heart failure (HF) is a growing cause of hospitalisation worldwide, and despite significant progress in its treatment it is still associated with high mortality.

Aim: The aim of this study was to find factors predicting in-hospital death in acute decompensated HF by analysis of basic laboratory data and echocardiography, routinely collected on admission to the hospital.

Methods: To this single-centre retrospective study we involved 638 consecutive patients hospitalised in the years 2007–2008 due to acute decompensated HF. To the initial univariate analysis we included the results of echocardiography and 36 basic laboratory tests performed at hospital admission. Parameters significantly associated with in-hospital death in univariate analysis were taken to multivariate regression analysis.

Results: In-hospital death occurred in 119 cases (median age 75 years; 40.3% females). The multivariate analysis revealed significant association between in-hospital death and: higher leukocyte count (death [D]: 13.5 vs. survival [S]: 8.8 G/L, $p < 0.01$), higher neutrophil count (D: 10.5 vs. S: 5.9 G/L, $p < 0.01$), lower lymphocyte count (D: 1.3 vs. S: 1.7 G/L, $p < 0.05$), higher C-reactive protein concentration (D: 20.8 vs. S: 6.7 mg/dL, $p < 0.01$), higher serum glucose concentration (D: 167.0 vs. S: 116.0 mg/dL, $p < 0.00001$), higher serum creatinine concentration (D: 1.5 vs. S: 1.2 mg/dL, $p < 0.0001$), higher blood urea nitrogen concentration (D: 29.0 vs. S: 22.0 mg/dL, $p < 0.00001$), and higher aspartate aminotransferase (D: 72.0 vs. S: 27.0 U/L, $p < 0.0001$). Surprisingly, there was no significant association with echocardiographic parameters.

Conclusions: Analysis of basic laboratory data collected on admission to the hospital may help to identify patients with acute decompensated HF, who are at high risk of in-hospital death.

Key words: mortality predictors, decompensated, heart failure, basic laboratory parameters, in-hospital death

Kardiol Pol 2017; 75, 2: 157–163

Address for correspondence:
Małgorzata Ostrowska, MD, PhD, Department of Principles of Clinical Medicine, Collegium Medicum, Nicolaus Copernicus University, ul. Marii Skłodowskiej-Curie 9, 85–094 Bydgoszcz, Poland, e-mail: ostrowska.go@gmail.com
Received: 07.05.2016 **Accepted:** 08.09.2016 **Available as AoP:** 03.10.2016

INTRODUCTION

Heart failure (HF) is a significant cause of morbidity and mortality worldwide [1]. Approximately 14 million subjects in Europe currently suffer from HF, and this number is increasing [2]. It is estimated that in Poland this condition concerns approximately 800,000 patients. As compared with other European countries, in the Heart Failure Pilot Survey, Polish HF patients are usually hospitalised due to acute HF (73%), develop HF at a younger age, and are more severely ill, presenting with higher New York Heart Association class [3]. Natural history of HF demonstrates as chronic, progressive, complex disease characterised by recurrent hospitalisations for decompensation. It is the most common reason for hospitalisation in the over-65 age group [2]. It is associated with very poor prognosis; approximately 50% of patients diagnosed with HF are dead within five years of the diagnosis [4]. The proportion of patients with HF with preserved ejection fraction (EF) ranges from 22% to 73% [5]. Many models of risk stratification and mortality prediction have been developed for patients with HF during the past few years [6–12]. However, we are still looking for a clinically practical, user-friendly, simple, and universal method for HF patient assessment. Therefore, our objective was to find factors predicting in-hospital death in patients with acute decompensated HF by analysis of basic laboratory data and echocardiographic results, which are routinely collected on admission to the hospital.

METHODS

This single-centre, retrospective analysis was performed in 638 consecutive patients hospitalised in the 1st Department of Cardiology, Medical University of Gdansk from January 1st, 2007 to December 31st, 2008 due to acute decompensated HF. The hospital electronic database was used to search for HF patients. The diagnosis of HF was based on the European Society of Cardiology Guidelines on the diagnosis and treatment of acute HF 2005 [13]. This retrospective analysis was approved by the Ethics Committee of the Medical University of Gdansk in accordance with the Declaration of Helsinki.

The results of 36 basic laboratory tests performed on admission to the hospital were collected, including: complete blood count, serum sodium, potassium, D-dimers, international normalised ratio, serum glucose, glomerular filtration rate, serum creatinine, blood urea nitrogen, C-reactive protein (CRP), aspartate aminotransferase, alanine aminotransferase, troponin I, B-type natriuretic peptide, thyroid-stimulating hormone, total bilirubin, and serum uric acid concentration. Echocardiographic parameters recorded on admission to the hospital included: EF, left ventricular end-systolic and end-diastolic dimensions, interventricular septal dimension, posterior wall dimension, left atrial dimension, and inferior vena cava diameters at end-expiration and end-inspiration. Altogether, 43 candidate predictor variables were collected to be considered in the analysis. In the next step a database of collected data was created. The studied group was divided into two subgroups: S — "survival" — patients who were discharged home, and D — "death" — patients who died during index hospitalisation. All candidate variables were taken to perform initial univariate analysis. Parameters significantly associated with in-hospital death in univariate analysis were taken to multivariate regression analysis.

Statistical analysis

Our data was checked for normal distribution by the use of the Shapiro-Wilk W test. The continuous variables were analysed by the use of either Student's t-test or non-parametric Mann-Whitney U test, depending on the presence or absence of normal distribution. Comparisons between categorical variables were performed with the use of χ^2 test, with Yate's correction if necessary. Parameters significant in univariate analysis were taken to multivariate regression analysis. We performed multivariate regression analysis on two models, the first one included leukocytes, neutrophils, and lymphocytes, while the second model included neutrophil-to-lymphocyte ratio (NLR). All continuous variables are presented as medians and inter-quartile ranges, and categorical variables are presented as numbers and per cent of patients. The reported p-values are two-sided and a p-value of less than 0.05 was considered to indicate statistical significance. All analyses were performed by STATISTICA, version 9.1. Statsoft, Inc. (2010).

RESULTS

The overall group consisted of 638 patients — 421 (66.0%) men, median age 69 years, and 217 (34.0%) women, median age 74 years. In-hospital death occurred in 119 (18.7%) patients, median age 75 years; 71 men, median age 73 years; 48 women, median age 79 years, while 519 (81.3%) patients were discharged home — median age 67 years; 350 men, median age 64 years; 169 women, median age 72 years. There were 30.3% of patients with preserved EF (> 40%). Among patients who died in the hospital 46.5% had preserved EF, while in patients who were discharged home EF was preserved in 28.4%.

The univariate analysis identified 32 parameters, from 43 variables evaluated, to be significantly ($p < 0.05$) associated with in-hospital death (Table 1). In multivariate regression analysis eight out of 32 candidate variables were identified to predict in-hospital mortality. The patients' characteristics that were associated with in-hospital mortality included: higher leukocytes count (death [D]: 13.5 vs. survival [S]: 8.8 G/L, $p < 0.01$), higher neutrophil count (D: 10.5 vs. S: 5.9 G/L, $p < 0.01$), lower lymphocyte count (D: 1.3 vs. S:1.7 G/L, $p < 0.05$), higher CRP concentration (D: 20.8 vs. S: 6.7 mg/dL, $p < 0.01$), higher serum glucose concentration (D: 167.0 vs. S: 116.0 mg/dL, $p < 0.00001$), higher serum creatinine concentration (D: 1.5 vs. S: 1.2 mg/dL, $p < 0.0001$), higher blood urea nitrogen concentration (D: 29.0 vs. S: 22.0 mg/dL,

Table 1. Laboratory and echocardiographic data on admission in regard to in-hospital death. Results of univariate analysis

Parameter	All patients (n = 638)	In–hospital death (n = 119)	Discharged home (n = 519)	p
Age [years]	69.0 (59.0–77.0)	75.0 (63.0–82.0)	67.0 (58.0–76.0)	< 0.001
Leukocytes [G/L]	9.2 (7.4–11.9)	13.5 (9.6–18.3)	8.8 (7.2–10.7)	< 0.001
Lymphocytes [G/L]	1.6 (1.2–2.2)	1.3 (0.8–2.0)	1.7 (1.3–2.2)	< 0.001
Neutrophils [G/L]	6.4 (4.9–9.1)	10.5 (7.2–15.0)	5.9 (4.7–9.0)	< 0.001
Neutrophil/Lymphocyte ratio	3.9 (2.4–6.7)	7.7 (4.0–15.3)	3.5 (2.3–5.4)	< 0.001
% lymphocytes	18.3 (11.9–26.3)	10.7 (5.8–19.1)	19.4 (14.3–26.6)	< 0.001
% neutrophils	71.3 (62.7–79.8)	83.1 (72.2–87.9)	69.7 (62.0–76.9)	< 0.001
Erythrocytes [T/L]	4.4 (4.0–4.8)	4.1 (3.6–4.6)	4.5 (4.1–4.8)	< 0.001
Haemoglobin [g/dL]	13.6 (12.2–14.8)	12.8 (11.1–14.0)	13.8 (12.4–14.9)	< 0.001
Haematocrit [%]	40.0 (36.0–43.0)	38.0 (33.0–41.0)	40.0 (37.0–44.0)	< 0.001
MCV [fL]	90.0 (87.0–94.0)	91.0 (87.0–93.0)	90.0 (87.0–94.0)	NS
RDW [%]	15.1 (14.1–16.6)	15.3 (14.4–17.9)	15.0 (14.1–16.4)	< 0.01
Platelets [G/L]	221.5 (179.0–275.6)	205.5 (154.0–263.3)	225.0 (183.0–279.3)	< 0.05
MPV [fL]	9.2 (8.5–10.1)	8.9 (8.3–10.0)	9.3 (8.6–10.2)	< 0.05
MCHC [g/dL]	34.0 (33.0–35.0)	34.0 (33.0–34.0)	34.0 (33.0–35.0)	< 0.01
% monocytes	7.3 (5.4–9.1)	5.4 (3.6–7.8)	7.6 (5.9–9.3)	< 0.001
% eosinophils	1.0 (0.2–2.1)	0.1 (0.03–0.5)	1.3 (0.5–2.3)	< 0.001
% basophils	0.4 (0.3–0.6)	0.3 (0.1–0.5)	0.4 (0.3–0.6)	< 0.001
Monocytes [G/L]	0.7 (0.5–0.8)	0.7 (0.5–1.0)	0.7 (0.5–0.8)	NS
Eosinophils [G/L]	0.09 (0.02–0.18)	0.02 (0–0.07)	0.10 (0.04–0.19)	< 0.001
Basophils [G/L]	0.09 (0.02–0.18)	0.03 (0.01–0.06)	0.04 (0.02–0.06)	< 0.05
INR	1.1 (1.0–1.4)	1.3 (1.2–1.8)	1.1 (1.0–1.3)	< 0.001
Serum sodium [mmol/L]	140.0 (137.0–142.0)	137.0 (134.0–141.0)	140.0 (138.0–142.0)	< 0.001
Serum potassium [mmol/L]	4.3 (3.9–4.7)	4.3 (3.8–4.8)	4.2 (3.9–4.7)	NS
Serum glucose [mg/dL]	120.5 (100.0–169.0)	167.0 (127.0–273.0)	116.0 (99.0–147.0)	< 0.001
D-dimers [μG/L]	265.8 (187.8–443.5)	376.1 (228.6–1028.0)	249.5 (177.8–402.9)	< 0.001
GFR [mL/min]	56.7 (41.8–60.0)	40.1 (29.6–54.0)	59.9 (45.5–60.0)	< 0.001
Serum creatinine [mg/dL]	1.2 (1.0–1.6)	1.5 (1.2–2.1)	1.2 (1.0–1.5)	< 0.001
Blood urea nitrogen [mg/dL]	23.0 (18.0–33.0)	29.0 (21.0–48.5)	22.0 (17.0–30.0)	< 0.001
Aspartate aminotransferase [U/L]	30.0 (21.0–58.0)	72.0 (30.8–260.3)	27.0 (20.0–43.5)	< 0.001
C-reactive protein [mG/L]	8.0 (3.0–26.9)	20.8 (7.0–64.1)	6.7 (2.6–19.6)	< 0.001
Alanine aminotransferase [U/L]	26.0 (16.0–48.3)	39.5 (19.0–111.0)	25.0 (16.0–42.0)	< 0.001
Troponin I [ng/mL]	0.06 (0.01–0.97)	2.4 (0.1–24.0)	0.04 (0.01–0.31)	< 0.001
BNP [pg/mL]	936.5 (355.0–1901.7)	1243.0 (566.5–2603.8)	878.0 (322.0–1754.0)	< 0.01
TSH [mIU/L]	1.0 (0.5–1.8)	1.0 (0.6–1.8)	1.0 (0.5–1.8)	NS
Total serum bilirubin [mg/dL]	1.2 (0.7–1.7)	1.1 (0.8–1.9)	1.2 (0.7–1.7)	NS
Serum uric acid [mg/dL]	7.2 (5.6–9.5)	8.6 (4.7–12.2)	7.2 (5.7–9.3)	NS
Ejection fraction [%]	35.0 (25.0–45.0)	35.0 (24.5–45.0)	35.0 (25.0–45.0)	NS
LV end-systolic dimension [mm]	44.0 (35.0–54.0)	43.0 (34.5–50.5)	45.0 (36.0–54.0)	< 0.01
LV end-diastolic dimension [mm]	57.0 (50.0–64.0)	56.0 (48.0–62.0)	58.0 (50.0–64.0)	< 0.05
Interventricular septal dimension [mm]	11.0 (10.0–13.0)	11.0 (10.0–13.0)	11.0 (10.0–13.0)	NS
Posterior wall dimension [mm]	10.0 (9.0–12.0)	11.0 (10.0–12.0)	10.0 (9.0–12.0)	NS
Left atrial dimension [mm]	46.0 (42.0–51.0)	45.0 (40.8–50.3)	46.0 (42.0–52.0)	NS
Inferior vena cava dimension at end-expiration [mm]	21.0 (18.0–24.3)	20.0 (18.0–24.0)	22.0 (18.0–25.0)	< 0.05
Inferior vena cava dimension at end-inspiration [mm]	15.0 (10.0–19.0)	16.0 (8.0–19.0)	15.0 (10.0–19.3)	NS

Data are presented as medians and interquartile ranges; BNP — B-type natriuretic peptide; GFR — glomerular filtration rate; INR — international normalised ratio; LV — left ventricular; MCHC — mean corpuscular haemoglobin concentration; MCV — mean corpuscular volume; MPV — mean platelet volume; NS — not significant; RDW — red blood cell distribution width; TSH — thyroid-stimulating hormone

Table 2. Results of the first and the second model of multivariate analysis

Parameter	In-hospital death (D) (n = 119)	Discharged home (S) (n = 519)	The first model			The second model		
			BETA	B	p	BETA	B	p
Leukocytes [G/L]	13.5 (9.6–18.3)	8.8 (7.2–10.7)	–1.582	–0.113	< 0.01			
Lymphocytes [G/L]	1.3 (0.8–2.0)	1.7 (1.3–2.2)	0.891	0.113	< 0.05			
Neutrophils [G/L]	10.5 (7.2–15.0)	5.9 (4.7–9.0)	1.447	0.129	< 0.01			
NLR	7.7 (4.0–15.3)	3.5 (2.3–5.4)				0.042167	0.008526	< 0.001
Serum glucose [mg/dL]	167.0 (127.0–273.0)	116.0 (99.0–147.0)	0.225	0.001	< 0.001	0.041409	0.001078	< 0.001
Serum creatinine [mg/dL]	1.5 (1.2–2.1)	1.2 (1.0–1.5)	–0.694	–0.033	< 0.001			
Blood urea nitrogen [mg/dL]	29.0 (21.0–48.5)	22.0 (17.0–30.0)	0.728	0.005	< 0.001	0.041985	0.004468	< 0.001
Aspartate aminotransferase [U/L]	72.0 (30.8–260.3)	27.0 (20.0–43.5)	0.173	0.0003	< 0.001	0.054824	0.000558	< 0.001
Alanine aminotransferase [U/L]	39.5 (19.0–111.0)	25.0 (16.0–42.0)				0.054301	–0.000307	< 0.01
C-reactive protein [mg/L]	20.8 (7.0–64.1)	6.7 (2.6–19.6)	0.136	0.001	< 0.01	0.040089	0.001147	< 0.001
Intercept				–0.123	< 0.05		0.205563	< 0.001

Data are presented as the median value and interquartile ranges; B — unstandardised regression coefficient; BETA — standardised regression coefficient; NLR — neutrophil-to-lymphocyte ratio

p < 0.00001), and higher aspartate aminotransferase concentration (D: 72.0 vs. S: 27.0 U/L, p < 0.0001) (Table 2). Surprisingly, there was no significant association with echocardiographic parameters. The second model of multivariate analysis with parameters significant in univariate analysis was performed, but this time NLR was included, while the number of neutrophils, lymphocytes, and leukocytes was excluded from the analysis. It occurred that higher NLR (D: 7.7 vs. S: 3.5, p < 0.001) was also predictive of in-hospital death in multivariate analysis. Comparing results of both models of multivariate analysis we can notice that the results are quite similar — increased inflammatory parameters, increased plasma glucose concentration, and increased parameters of impaired renal and liver function — with an exception: serum creatinine was no longer significant, while alanine aminotransferase became significant in a second model of multivariate analysis (Table 2).

DISCUSSION

Our analysis of 638 decompensated HF patients demonstrates that the risk of in-hospital mortality can be reliably estimated using routinely available laboratory data obtained at hospital admission. We observed similarities and differences between our findings and those of other published risk stratification studies. In our population the in-hospital mortality rate was very high — approximately 18%. According to other papers, in-hospital mortality in HF patients varies greatly, ranging from 2% to 20% [6–8, 14]. At first, we used univariate analysis. Because multiple risk factors can exist in the same patient, death predictor analysis must consider factors in combination rather than in isolation. Therefore, parameters significantly associated with in-hospital death in univariate analysis were taken to multivariate regression analysis. In the OPTIMIZE-HF (Organized Program to Initiate Lifesaving Treatment in Hospitalised Patients with Heart Failure) study logistic regression analysis was performed, while in the ADHERE (Acute Decompensated Heart Failure National Registry) study the classification analysis/risk tree was used [7, 8].

The three factors most predictive of mortality in the ADHERE trial were increased blood urea nitrogen, increased serum creatinine, and decreased systolic blood pressure [8]. Likewise, Biegus et al. [14] identified increased baseline serum creatinine and blood urea nitrogen to be predictive of in-hospital death in 270 acute HF patients. We also determined parameters of impaired renal function, blood urea nitrogen, and serum creatinine, as significant predictors. Acute decompensated HF often causes acute pre-renal kidney failure due to decreased blood flow through the kidneys. Chronic HF also often co-exists with chronic kidney failure, which is associated with increased activity of the renin–angiotensin–aldosterone system, oxidative stress, inflammation, and increased activity of the sympathetic nervous system [15]. These are the cornerstones of cardiorenal syndrome patho-

physiology, a condition in which combined cardiac and renal dysfunction amplifies progression of failure of the individual organ [16]. In the OPTIMIZE-HF study liver disease was an important death predictor, while in the EFFECT (Enhanced Feedback for Effective Cardiac Treatment) study liver cirrhosis was predictive of mortality [7, 9]. In our analysis we identified increased aspartate aminotransferase concentration, which is a good marker of liver dysfunction, to be characteristic for HF patients who died in the hospital. Congestive HF is associated with increased central venous pressure, which is transmitted to the liver via the inferior vena cava and hepatic veins, causing venous congestion and eventually congestive hepatopathy, presenting as a liver dysfunction accompanied by a peak of the liver enzymes — especially aminotransferases.

It is known that inflammation plays an important role in the progression of coronary atherosclerosis, which is the main cause of HF [17]. CRP seems to be increased in HF patients [18–20]. Anand et al. [19] suggested that higher CRP concentration is associated with more severe HF, and independently with increased morbidity and mortality. Alonso-Martinez et al. [20] indicated that higher CRP concentration is related to higher rate of readmission and mortality. We found a significant association in multivariate analysis between the occurrence of in-hospital death and increased parameters of inflammation, including higher concentration of CRP, but also higher leukocyte count, as well as higher neutrophil count. Many researches highlighted that NLR is associated with poor outcomes in patients with acute coronary syndromes [21, 22]. However, its role for risk stratification in HF has not been well described. In a paper by Uthamalingam et al. [22] they found that higher NLR is associated with an increased risk of long-term mortality in patients admitted with acute decompensated HF. We also observed the NLR to be predictive of in-hospital mortality in the second model of multivariate analysis.

According to Berry et al. [23], admission blood glucose concentration and diabetes are prognostically important in HF patients. They noticed abnormal glucose tolerance, but not diabetes, to be predictive of in-hospital mortality. Barsheshet et al. [24] found elevated admission blood glucose concentration to be associated with increased in-hospital and 60-day mortality. Furthermore, mortality risk was correlated with admission glucose concentration and each 18 mg/dL increase in glucose concentration was associated with a 31% increase of in-hospital mortality. On the other hand, Kosiborod et al. [25] found no significant association between admission glucose levels and mortality in large cohort of patients hospitalised due to HF. Their findings suggest that the relationship between hyperglycaemia and adverse outcomes seen in acute myocardial infarction cannot be automatically extended to patients hospitalised with other cardiovascular conditions [26]. Our HF patients who died in-hospital had higher admission blood glucose concentration, which we identified as a significant death predictor in multivariate analysis.

Many studies have shown that increased levels of specific cardiac markers are associated with bad outcomes. It is emphasised that increased admission troponins and B-type natriuretic peptide are good in-hospital death predictors in HF patients [27–29]. In our observation we identified them to be predictive of death only in univariate analysis. Our conclusions are in line with findings by Biegus et al. [14], who did not define baseline plasma N-terminal pro-B-type natriuretic peptide as an in-hospital mortality predictor because its concentration did not differ between survivors and non-survivors.

Surprisingly, we did not identify any echocardiographic parameter to be significant in multivariate analysis, while in univariate analysis left ventricular end-systolic and end-diastolic dimensions, but not left ventricular EF, were predictive of death.

Limitations of the study

There are some limitations of the study that deserve attention. First of all, we found predictors of in-hospital mortality only, so we cannot extrapolate these results to foresee post-discharge outcomes. Secondly, our study was not a prospective, randomised trial. Thirdly, the echocardiographic examination and the blood drawing for laboratory analysis were not performed in a standardised manner or time after admission. Fourthly, there was a significant group of patients who did not have an echocardiographic examination at hospital admission. Fifthly, due to the lack of clinical characteristics of the study population there might have been other factors prognostic for in-hospital death, which were not included in the analysis.

CONCLUSIONS

Analysis of basic laboratory test results, performed on admission to the hospital, may help to identify patients with acute decompensated HF, who are at high risk of in-hospital death.

Conflict of interest: *none declared*

References

1. Roger VL, Weston SA, Redfield MM, et al. Trends in heart failure incidence and survival in a community-based population. JAMA. 2004; 292(3): 344–350, doi: 10.1001/jama.292.3.344, indexed in Pubmed: 15265849.
2. SHAPE Survey Results to the General Public, Annual Congress of the European Society of Cardiology in Vienna, September 2003.
3. Sosnowska-Pasiarska B, Bartkowiak R, Wożakowska-Kapłon B, et al. Population of Polish patients participating in the Heart Failure Pilot Survey (ESC-HF Pilot). Kardiol Pol. 2013; 71(3): 234–240, doi: 10.5603/KP.2013.0034, indexed in Pubmed: 23575777.
4. Ho KK, Anderson KM, Kannel WB, et al. Survival after the onset of congestive heart failure in Framingham Heart Study subjects. Circulation. 1993; 88(1): 107–115, doi: 10.1161/01.cir.88.1.107, indexed in Pubmed: 8319323.
5. Ponikowski P, Voors AA, Anker SD, et al. Authors/Task Force Members. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J.

2016; 37(27): 2129–2200, doi: 10.1093/eurheartj/ehw128, indexed in Pubmed: 27206819.
6. Aronson D, Mittleman MA, Burger AJ. Elevated blood urea nitrogen level as a predictor of mortality in patients admitted for decompensated heart failure. Am J Med. 2004; 116(7): 466–473, doi: 10.1016/j.amjmed.2003.11.014, indexed in Pubmed: 15047036.
7. Abraham WT, Fonarow GC, Albert NM, et al. OPTIMIZE-HF Investigators and Coordinators. Predictors of in-hospital mortality in patients hospitalized for heart failure: insights from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF). J Am Coll Cardiol. 2008; 52(5): 347–356, doi: 10.1016/j.jacc.2008.04.028, indexed in Pubmed: 18652942.
8. Adams KF, Fonarow GC, Emerman CL, et al. ADHERE Scientific Advisory Committee and Investigators. Characteristics and outcomes of patients hospitalized for heart failure in the United States: rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). Am Heart J. 2005; 149(2): 209–216, doi: 10.1016/j.ahj.2004.08.005, indexed in Pubmed: 15846257.
9. Lee DS, Austin PC, Rouleau JL, et al. Predicting mortality among patients hospitalized for heart failure: derivation and validation of a clinical model. JAMA. 2003; 290(19): 2581–2587, doi: 10.1001/jama.290.19.2581, indexed in Pubmed: 14625335.
10. Balsam P, Tymińska A, Kapłon-Cieślicka A, et al. Predictors of one-year outcome in patients hospitalised for heart failure: results from the Polish part of the Heart Failure Pilot Survey of the European Society of Cardiology. Kardiol Pol. 2016; 74(1): 9–17, doi: 10.5603/KP.a2015.0112, indexed in Pubmed: 26101021.
11. Gromadziński L, Targoński R. Impact of clinical and echocardiographic parameters assessed during acute decompensation of chronic heart failure on 3-year survival. Kardiol Pol. 2006; 64(9): 951–958, indexed in Pubmed: 17054026.
12. Bjurman C, Holmström A, Petzold M, et al. Assessment of a multi-marker risk score for predicting cause-specific mortality at three years in older patients with heart failure and reduced ejection fraction. Cardiol J. 2015; 22(1): 31–36, doi: 10.5603/CJ.a2014.0017, indexed in Pubmed: 24526512.
13. Nieminen MS, Böhm M, Cowie MR, et al. ESC Committe for Practice Guideline (CPG). Executive summary of the guidelines on the diagnosis and treatment of acute heart failure: the Task Force on Acute Heart Failure of the European Society of Cardiology. Eur Heart J. 2005; 26(4): 384–416, doi: 10.1093/eurheartj/ehi044, indexed in Pubmed: 15681577.
14. Biegus J, Zymliński R, Szachniewicz J et al. Clinical characteristics and predictors of in-hospital mortality in 270 consecutive patients hospitalised due to acute heart failure in a single cardiology centre during one year. Kardiol Pol. 2011; 69(10): 997–1005. indexed in Pubmed: 22006596.
15. Gronda E, Genovese S, Padeletti L, et al. Renal function impairment predicts mortality in patients with chronic heart failure treated with resynchronization therapy. Cardiol J. 2015; 22(4): 459–466, doi: 10.5603/CJ.a2015.0019, indexed in Pubmed: 26325259.
16. Ronco C, Haapio M, House AA, et al. Cardiorenal syndrome. J Am Coll Cardiol. 2008; 52(19): 1527–1539, doi: 10.1016/j.jacc.2008.07.051, indexed in Pubmed: 19007588.
17. Libby P. Inflammation and Atherosclerosis. Circulation. 2002; 105(9): 1135–1143, doi: 10.1161/hc0902.104353.
18. Pye M, Rae AP, Cobbe SM. Study of serum C-reactive protein concentration in cardiac failure. Br Heart J. 1990; 63(4): 228–230, doi: 10.1136/hrt.63.4.228, indexed in Pubmed: 2337494.
19. Anand IS, Latini R, Florea VG, et al. Val-HeFT Investigators. C-reactive protein in heart failure: prognostic value and the effect of valsartan. Circulation. 2005; 112(10): 1428–1434, doi: 10.1161/CIRCULATIONAHA.104.508465, indexed in Pubmed: 16129801.
20. Alonso-Martínez JL, Llorente-Diez B, Echegaray-Agara M, et al. C-reactive protein as a predictor of improvement and readmission in heart failure. Eur J Heart Fail. 2002; 4(3): 331–336, doi: 10.1016/s1388-9842(02)00021-1, indexed in Pubmed: 12034159.
21. Azab B, Zaher M, Weiserbs KF, et al. Usefulness of neutrophil to lymphocyte ratio in predicting short- and long-term mortality after non-ST-elevation myocardial infarction. Am J Cardiol. 2010; 106(4): 470–476, doi: 10.1016/j.amjcard.2010.03.062, indexed in Pubmed: 20691303.
22. Uthamalingam S, Patvardhan EA, Subramanian S, et al. Utility of the neutrophil to lymphocyte ratio in predicting long-term outcomes in acute decompensated heart failure. Am J Cardiol. 2011; 107(3): 433–438, doi: 10.1016/j.amjcard.2010.09.039, indexed in Pubmed: 21257011.
23. Berry C, Brett M, Stevenson K, et al. Nature and prognostic importance of abnormal glucose tolerance and diabetes in acute heart failure. Heart. 2008; 94(3): 296–304, doi: 10.1136/hrt.2006.110999, indexed in Pubmed: 17664189.
24. Barsheshet A, Garty M, Grossman E, et al. Admission blood glucose level and mortality among hospitalized nondiabetic patients with heart failure. Arch Intern Med. 2006; 166(15): 1613–1619, doi: 10.1001/archinte.166.15.1613, indexed in Pubmed: 16908794.
25. Kosiborod M, Inzucchi SE, Spertus JA, et al. Elevated admission glucose and mortality in elderly patients hospitalized with heart failure. Circulation. 2009; 119(14): 1899–1907, doi: 10.1161/CIRCULATIONAHA.108.821843, indexed in Pubmed: 19332465.
26. Tomaszuk Kazberuk A, Kożuch M, Małyszko J, et al. What level of hyperglycaemia on admission indicates a poor prognosis in patients with myocardial infarction treated invasively? Kardiol Pol. 2012; 70(6): 564–572, indexed in Pubmed: 22718372.
27. Perna ER, Macín SM, Cimbaro Canella JP, et al. Minor myocardial damage detected by troponin T is a powerful predictor of long-term prognosis in patients with acute decompensated heart failure. Int J Cardiol. 2005; 99(2): 253–261, doi: 10.1016/j.ijcard.2004.01.017, indexed in Pubmed: 15749184.
28. Maisel A, Hollander JE, Guss D, et al. Rapid Emergency Department Heart Failure Outpatient Trial investigators. Primary results of the Rapid Emergency Department Heart Failure Outpatient Trial (REDHOT). A multicenter study of B-type natriuretic peptide levels, emergency department decision making, and outcomes in patients presenting with shortness of breath. J Am Coll Cardiol. 2004; 44(6): 1328–1333, doi: 10.1016/j.jacc.2004.06.015, indexed in Pubmed: 15364340.
29. Diez M, Talavera ML, Conde DG, et al. High-sensitivity troponin is associated with high risk clinical profile and outcome in acute heart failure. Cardiol J. 2016; 23(1): 78–83, doi: 10.5603/CJ.a2015.0058, indexed in Pubmed: 26412605.

Cite this article as: Ostrowska M, Ostrowski A, Łuczak M, et al. Basic laboratory parameters as predictors of in-hospital death in patients with acute decompensated heart failure: data from a large single-centre cohort. Kardiol Pol. 2017; 75(2): 157–163. doi: 10.5603/KP.a2016.0147.

Ocena wyników podstawowych testów laboratoryjnych jako czynników predykcyjnych wystąpienia zgonu wewnątrzszpitalnego wśród pacjentów z zaostrzeniem niewydolności serca: dane z dużego jednoośrodkowego badania

Małgorzata Ostrowska[1], Adam Ostrowski[2], Małgorzata Łuczak[3], Miłosz Jaguszewski[4], Piotr Adamski[1], Jerzy Bellwon[4], Andrzej Rynkiewicz[5], Marcin Gruchała[4]

[1]Zakład Podstaw Medycyny Klinicznej, *Collegium Medicum*, Uniwersytet Mikołaja Kopernika, Bydgoszcz
[2]Klinika Urologii Ogólnej i Onkologicznej, *Collegium Medicum*, Uniwersytet Mikołaja Kopernika, Bydgoszcz
[3]Oddział Anestezjologii i Intensywnej Terapii, Szpital Specjalistyczny im. F. Ceynowy, Wejherowo
[4]I Klinika Kardiologii, Gdański Uniwersytet Medyczny, Gdańsk
[5]Klinika Kardiologii i Kardiochirurgii, Uniwersytet Warmińsko-Mazurski, Olsztyn

Streszczenie

Wstęp: Liczba hospitalizacji spowodowanych zaostrzeniem niewydolności serca (HF) ciągle wzrasta i mimo znaczącego postępu w leczeniu nadal wiąże się z dużą śmiertelnością.

Cel: Celem pracy była ocena zależności między wynikami podstawowych badań laboratoryjnych i echokardiografii, wykonywanych przy przyjęciu do szpitala, a występowaniem zgonu wewnątrzszpitalnego, wśród chorych z zaostrzeniem HF.

Metody: Analizie jednoczynnikowej poddano wyniki 36 testów laboratoryjnych i echokardiografii wszystkich 638 kolejnych pacjentów hospitalizowanych z powodu zaostrzenia HF w I Klinice Kardiologii Gdańskiego Uniwersytetu Medycznego w latach 2007–2008. Czynniki istotnie związane z wystąpieniem zgonu wewnątrzszpitalnego w analizie jednoczynnikowej ($p < 0,05$) zostały wzięte do analizy regresji wieloczynnikowej.

Wyniki: Zgon wewnątrzszpitalny wystąpił w 119 przypadkach (mediana wieku 75 lat; 40,3% kobiet). Wieloczynnikowa analiza uzyskanych wyników wykazała statystycznie istotny związek wystąpienia zgonu wewnątrzszpitalnego: z wyższą liczbą leukocytów (zgon (Z): 13,5 *vs*. wypis (W): 8,8 G/l, $p < 0,01$), wyższą liczbą neutrocytów (Z: 10,5 *vs*. W: 5,9 G/l, $p < 0,01$), niższą liczbą limfocytów (Z: 1,3 *vs*. W: 1,7 G/l, $p < 0,05$), wyższym stężeniem białka C-reaktywnego (Z: 20,8 *vs*. W: 6,7 mg/dl, $p < 0,01$), wyższym stężeniem glukozy (Z: 167,0 *vs*. W: 116,0 mg/dl, $p < 0,00001$), wyższym stężeniem kreatyniny (Z: 1,5 *vs*. W: 1,2 mg/dl, $p < 0,0001$), wyższym stężeniem azotu mocznika (Z: 29,0 *vs*. W: 22,0, $p < 0,00001$) oraz wyższym stężeniem aminotransferazy asparaginianowej (Z: 72,0 *vs*. W: 27,0 U/l, $p < 0,0001$). Nie znaleziono istotnego związku między parametrami echokardiograficznymi a wystąpieniem zgonu wewnątrzszpitalnego.

Wnioski: Analizując wyniki podstawowych badań laboratoryjnych, wykonywanych przy przyjęciu do szpitala, można zidentyfikować pacjentów z zaostrzeniem HF, którzy są obarczeni wysokim ryzykiem wystąpienia zgonu wewnątrzszpitalnego.

Słowa kluczowe: czynniki predykcyjne, zaostrzenie niewydolności serca, zgon wewnątrzszpitalny, podstawowe badania laboratoryjne

Kardiol Pol 2017; 75, 2: 157–163

Adres do korespondencji:
dr n. med. Małgorzata Ostrowska, Zakład Podstaw Medycyny Klinicznej, *Collegium Medicum*, Uniwersytet Mikołaja Kopernika, ul. Marii Skłodowskiej-Curie 9, 85–094 Bydgoszcz, e-mail: ostrowska.go@gmail.com
Praca wpłynęła: 07.05.2016 r. **Zaakceptowana do druku:** 08.09.2016 r. **Data publikacji AoP:** 03.10.2016 r.