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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

Jan Biegus[1], Robert Zymliński[1], Joanna Szachniewicz[1], Paweł Siwołowski[1], Aleksander Pawluś[3], Waldemar Banasiak[1], Ewa A. Jankowska[1, 2], Piotr Ponikowski[1, 2]

[1]Cardiology Department, 4th Military Hospital, Wroclaw, Poland; [2]Department of Heart Diseases, Wroclaw Medical University, Wroclaw, Poland; [3]Students' Scientific Association, Department of Heart Diseases, Wroclaw Medical University, Wroclaw, Poland

Abstract

Background: Acute heart failure (HF) is an emerging problem in clinical practice, associated with high in-hospital mortality and a high short-term readmission rate.

Aim: To describe the clinical characteristics and define predictors of in-hospital mortality in patients with acute HF.

Methods: We conducted a prospective registry of all consecutive patients hospitalised due to acute HF from October 2008 to November 2009 in a single cardiology centre. Clinical status and laboratory parameters were analysed on admission and after 48 h.

Results: We examined 270 patients (age 68 ± 13 years, 71% men, 27% with *de novo* acute HF, 55% with ischaemic aetiology, 56% with decompensated chronic HF, 80% with warm-wet haemodynamic profile). In-hospital mortality was 8.5% (n = 23). There were no differences between survivors *vs* non-survivors regarding age, gender, HF aetiology, prevalence of *de novo* acute HF, and baseline heart rate and body weight values and changes of these parameters during hospitalisation ($p > 0.2$ for all comparisons). Cardiogenic shock and isolated right-sided HF were more common in patients who died as compared to survivors (17% *vs* 1% and 22% *vs* 2%, respectively; $p < 0.001$), as were the cold-wet and cold-dry haemodynamic profiles (22% *vs* 2% and 17% *vs* 1%, respectively; $p < 0.001$). The most common factor precipitating decompensation in non-survivors was an acute coronary syndrome (17% *vs* 7%), while elevation of blood pressure and inadequate diuretic therapy were the most common causes of acute HF in survivors (26% *vs* 4% and 45% *vs* 22%, respectively; $p < 0.05$). Baseline mean blood pressure and serum Na^+ level were higher in survivors than in non-survivors (94 ± 20 *vs* 79 ± 19 mm Hg and 140 ± 4 *vs* 136 ± 5 mmol/L, respectively; $p < 0.001$) and both remained higher during follow-up. There were no differences in baseline haemoglobin and serum K^+ levels between these groups. Haemoglobin level decreased after 48 h of therapy only in patients who died (11.1 ± 2.4 *vs* 12.5 ± 2.1 g/dL; $p < 0.01$), whereas a reduction in serum K^+ level after 48 h was observed only in survivors (4.2 ± 0.6 *vs* 3.9 ± 0.5 mmol/L; $p < 0.05$), probably reflecting effective diuretic therapy. Baseline renal function was more impared in non-survivors (serum creatinine 1.7 [1, 2.5] *vs* 1.2 [1, 1.6] mg/dL, and blood urea nitrogen 40 [24, 65] *vs* 24 [19, 33] mg/dL; $p < 0.05$) and deteriorated further during hospitalisation (serum creatinine 2.0 [1.2, 2.5] *vs* 1.2 [0.9, 1.5] mg/dL, blood urea nitrogen 64 [45, 77] *vs* 27 [19, 36] mg/dL; $p < 0.01$). Baseline plasma N-terminal proB-type natriuretic peptide (NT-proBNP) level did not differentiate these two groups, but plasma NT-proBNP level measured after 48 h was lower in survivors compared to non-survivors (3560 [1711, 6738] *vs* 11780 [5371, 18912] pg/mL; $p < 0.01$); data are shown as medians [lower, upper quartile].

Conclusions: In our registry, in-hospital mortality in patients admitted due to acute HF was slightly higher compared to other reports. Baseline values of some parameters (e.g. blood pressure, serum Na^+, renal function) as well as their changes during hospitalisation (e.g. serum K^+, renal function, plasma NT-proBNP) can help identify acute HF patients at a higher risk of in-hospital mortality.

Key words: acute heart failure, registry, risk stratification, prognosis

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Address for correspondence:
Jan Biegus, MD, PhD, Cardiology Department, 4th Military Hospital, ul. Weigla 5, 50–540 Wrocław, Poland, e-mail: janbiegus@o2.pl
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INTRODUCTION

Acute heart failure (HF) is a major and increasing clinical problem associated with very poor outcome. In-hospital mortality is as high as 5–10% [1–4]. Among patients who are discharged, 15% die and 30% require readmission within 3 months of follow-up [2]. Despite increasing knowledge on acute HF, it is still very difficult to identify patients hospitalised with acute HF in whom prognosis is particularly grave. Based on large, multicentre registries, such as the Organised Program to Initiate Lifesaving Treatment in Hospitalised Patients with Heart Failure (OPTIMIZE-HF) and the Acute Decompensated Heart Failure National Registry (ADHERE), multiple risk factors for adverse outcomes in patients with acute HF were identified [5, 6]. Such data are still missing, however, for Poland and other central European countries.

For this reason, we designed a prospective observational study (a registry) of patients admitted due to acute HF to our department. Our aims were to describe the clinical characteristics of these patients and to identify factors associated with increased in-hospital mortality.

METHODS

Study group

We included consecutive patients hospitalised due to acute HF in our department, from October 2008 to November 2009. The only inclusion criterion was the diagnosis of acute HF as defined according to the current European Society of Cardiology (ESC) guidelines [7]. There were no exclusion criteria.

In all patients, researchers completed a questionnaire regarding the presenting clinical problem, concomitant diseases, medications, aetiology of HF, the cause of decompensation, acute HF type, and the haemodynamic profile. On admission, the following laboratory tests were performed: complete blood count — haemoglobin (HGB; g/dL); haematocrit (%), leukocyte count (G/L), platelets (G/L), plasma sodium (Na^+; mmol/L), potassium (K^+; mmol/L) and creatinine level (mg/dL), blood urea nitrogen (BUN; mg/dL), glucose (mg/dL), glutamic-oxaloacetic aminotransferase (GOT; IU/L), glutamic-pyruvic aminotransferase (GTP; IU/L), γ-glutamylotranspeptidase (GGTP; IU/L), bilirubin (mg/dL), coagulation parameters, plasma N-terminal pro-B-type natriuretic peptide (NT-proBNP; pg/mL; immunoenzymatic assay, Siemens, Marburg, Germany), serum troponin I (TnI; ng/mL; one-step immunoenzymatic assay, Dimension RxLMax, Siemens, Marburg, Germany), total protein (mg/dL), albumin (mg/dL), thyroid-stimulating hormone (TSH; μIU/mL; chemiluminescent assay, Roche, Cobas 411, Mannhein, Germany), C-reactive protein (CRP; mg/L; immunonephelometric assay, Siemens CardioPhase, Marburg, Germany), and arterial blood gases. Based on serum creatinine, age, and gender, estimated glomerular filtration rate (eGFR; mL/min/1.73 m^2) was calculated using the simplified modification of diet in renal disease (MDRD) equation [8]. On the third hospital day, we re-evaluated complete blood count, plasma Na^+ and K^+, creatinine, BUN, and NT-proBNP.

During hospitalisation, standard transthoracic echocardiography was performed, and the following parameters were analysed: left ventricular ejection fraction (LVEF, %); left ventricular end-diastolic dimension (LVEDD, mm), right ventricular diastolic diameter (RVDD, mm), and left atrial dimension (LA, mm).

The patients were treated in accordance with the current ESC guidelines [7].

Statistical analysis

Normally distributed continuous variables are shown as mean values ± SD, continuous variables with a skewed distribution as median values with upper and lower quartiles, and categorical variables as absolute numbers and percentages

To analyse in-hospital survival, the study population was divided into two groups: (a) patients who died during the index hospitalisation; and (b) patients who survived until discharge; and the analysed parameters were compared between the two groups. Differences between normally distributed continuous variables were tested using the Student *t* test for unpaired samples, continuous variables with a skewed distribution using the Mann-Whitney U test, and categorical variables using the χ^2 test.

Changes of parameters with time were tested using the Student *t* test and the sign test for continuous variables with a normal or skewed distribution, respectively. Statistical analyses were performed using the Statistica 8.0 software. A p value < 0.05 was considered statistically significant.

RESULTS

Clinical characteristics and laboratory parameters in patients admitted due to acute HF

During one-year period in our centre, 270 patients were hospitalised due to acute HF and all these patients were included into the registry. About 4% of patients were referred from other hospitals in the same voivodeship. Table 1 shows the characteristics of the study group, which consisted mostly of men. The mean age was 68 ± 12 years, and three fourths of the admissions were due to acute decompensation of chronic HF. The most prevalent aetiology of HF was ischaemic heart disease, and the predominant haemodynamic pattern was the wet-warm profile. The cause of acute HF was mostly attributed to inadequate diuretic therapy. The most common concomitant diseases were arterial hypertension and ischaemic heart disease. Mean LVEF was 36 ± 14%, and LVEF ≤ 45% was noted in 63% of patients. Before admission, most commonly used medications were diuretics (73%), beta-blockers (66%) and angiotensin-converting enzyme inhibitors (ACEI, 56%). Among patients with HF diagnosed before the index hospitalisation, proportions of patients taking these drugs were higher — 87%, 78%, and 62%, respectively.

Table 1. Clinical characteristics of 270 patients with acute heart failure included into the registry

Parameter	Acute HF group (n = 270)
De novo HF decompensation	72 (27)
Gender (women)	78 (29)
Age (years)	68 ± 12
Left ventricular ejection fraction [%]	36 ± 14
Left ventricular ejection fraction ≤ 45 [%]	171 (63)
Left ventricular end-diastolic dimension [mm]	62 ± 11
Interventricular septum thickness [mm]	12 ± 3
Right ventricular dimension [mm]	28 ± 7
Left atrial dimension [mm]	49 ± 8
Aetiology of HF:	
Ischaemic	148 (55)
Arterial hypertension	65 (24)
Valvular heart disease	57 (21)
Myocarditis/infection	23 (8)
Toxic	12 (4)
Acute HF type:	
Decompensation of chronic HF	151 (56)
Pulmonary oedema	53 (20)
Acute HF with blood pressure elevation	30 (11)
Acute right ventricular HF	30 (11)
Acute HF in an acute coronary syndrome	22 (8)
Cardiogenic shock	6 (2)
Haemodynamic profile:	
Warm-wet	215 (80)
Warm-dry	37 (14)
Cold-wet	11 (4)
Cold-dry	7 (2)
Most likely cause of acute HF:	
Inadequate diuretic therapy	117 (43)
Blood pressure elevation	65 (24)
Acute coronary syndrome	22 (8)
Tachyarrhythmia	49 (18)
Infection	25 (9)
Concomitant diseases and procedures:	
Ischaemic heart disease	148 (54)
History of myocardial infarction	99 (37)
Previous coronary angioplasty	59 (22)
Previous coronary artery bypass grafting	32 (12)
Hypertension	161 (60)
Atrial fibrillation	119 (44)
Diabetes	106 (39)
History of renal failure	106 (39)
History of anaemia	46 (17)
Thyroid disease	32 (12)
History of stroke and/or TIA	36 (13)
Cancer	8 (3)
Medications used before hospitalisation:	
ACEI	150 (56)
ARB	30 (11)
Beta-blocker	178 (66)
Aldosterone antagonist	100 (37)
Diuretic	198 (73)
Digoxin	59 (22)
Statin	130 (48)
ASA	130 (48)
Oral anticoagulant	79 (29)

Reported values are means ± SD or n (%); ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor antagonist; ASA — acetylsalicylic acid; HF — heart failure; TIA — transient ischaemic attack

Overall, mean body weight of all patients admitted due to acute HF was 80 ± 16 kg and decreased within 2 days of treatment by 1.7 (3.2, 0.4) kg. Mean systolic/diastolic blood pressure (BP) and heart rate were 130 ± 33/75 ± 17 mm Hg and 90 ± 22 bpm on admission, and reduced to 114 ± 20/ /69 ± 10 mm Hg and 79 ± 15 bpm, respectively ($p < 0.001$). During the first 48 h of treatment, a reduction was also observed in the following parameters: HGB, haematocrit, leukocyte count, platelet count, plasma NT-proBNP, and serum K^+ ($p < 0.05$ for all). No changes of other laboratory parameters were found during the first 48 h of hospitalisation (Table 2).

Comparison of patients who died and survivors

During the index hospitalisation, 23 patients with acute HF died (8.5% of all treated patients). Progression of HF was the cause of death in all these cases.

The study population was divided into two groups: those who survived until discharge and those who died. The two groups did not differ in regard to basic demographic characteristics, echocardiographic parameters, and the aetiology of HF (Table 3). The most common forms of acute HF in survivors were acute decompensation of chronic HF and pulmonary oedema. Cardiogenic shock and right ventricular HF were more common among those patients who died ($p < 0.001$). Cold-wet and cold-dry haemodynamic profiles were also more common in this group ($p < 0.001$). In patients who survived until discharge, decompensation was more frequently attributed to inadequate diuretic therapy and BP elevation ($p < 0.05$). We did not find any differences between these groups in regard to the prevalence of concomitant disease, except for more frequent previous diagnoses of anaemia ($p < 0.05$). Treatment used before the index hospitalisation also did not differ between the groups (Table 3).

Patients who died in hospital had lower BP (systolic, diastolic, and mean) and lower Na^+ level both on admission and in the subsequent days of follow-up (Table 4). On admission, HGB level did not differ between the groups, but on the third day of treatment HGB level and haematocrit were found to be lower in patients who died in hospital. Baseline K^+ levels were also identical in both groups, but during further follow-up, K^+ level decreased in patients who survived but remained unchanged in those who died. Renal function (as evaluated based on creatinine level, eGFR and BUN) was initially worse in patients who died in hospital and deteriorated in this group during the first 48 h of hospitalisation. We did not find a baseline difference in plasma NT-proBNP level between the two groups. Among the patients who survived, plasma NT-proBNP level decreased on the third day of treatment, while a rising trend for plasma NT-proBNP level was noted among those who died. On the third day of hospitalisation, plasma NT-proBNP level in patients who died in hospital was higher than in those who survived until discharge.

Table 2. Laboratory parameters and vital signs in 270 patients with acute heart failure at the first and third day of hospitalisation

Parameter	Day 1	Day 3	Difference (day 3 – day 1)
Body weight [kg]	80.3 ± 16.3	79.2 ± 16.5**	–1.7 (–3.2; –0.4)
Systolic blood pressure [mm Hg]	130 ± 33	114 ± 20**	–15 (–30; 0)
Diastolic blood pressure [mm Hg]	75 ± 17	69 ± 10**	–5 (–16; 0)
Mean blood pressure [mm Hg]	93 ± 21	84 ± 13**	–8 (–20; 0)
Heart rate [bpm]	90 ± 22	79 ± 15**	–8 (–22; 4)
Haemoglobin [g/dL]	13.0 ± 2.1	12.4 ± 2.1**	–0.4 (–1.1; 0.2)
Haematocrit [%]	39.2 ± 6.4	37.2 ± 6.3**	–1.3 (–3.8; 0.7)
Leukocyte count [G/L]	10.2 ± 9.5	8.2 ± 3.3*	–1 (–2.5; 0)
Platelet count [G/L]	225 ± 90	202 ± 72**	–16 (–42; 5)
Serum Na^+ [mmol/L]	139 ± 5	139 ± 4	0 (–2; 2)
Serum K^+ [mmol/L]	4.2 ± 0.6	3.9 ± 0.5**	–0.3 (–0.6; 0.2)
Blood urea nitrogen [mg/dL]	25 (19; 35)	28 (20; 39)	1.2 (–4.1; 6.0)
Serum creatinine [mg/dL]	1.2 (0.98; 1.62)	1.2 (1.0; 1.5)	0.0 (–0.2; 0.1)
Glomerular filtration rate (MDRD equation) [mL/min/1.73 m^2]	60 ± 24	62 ± 36	1.1 (–5.2; 7.4)
Random serum glucose [mg/dL]	160 ± 77		
GOT [IU/L]	29 (21; 43)		
GPT [IU/L]	27 (16; 48)		
GGTP [IU/L]	74 (41; 119)		
Serum bilirubin [mg/dL]	1.3 (0.8; 2.0)		
Plasma NT-proBNP [pg/mL]	5307 (2845; 11509)	3774 (1774; 7575)**	–1306 (–4214; 12)
Change in plasma NT-proBNP in comparison to day 1 [%]		68 (48; 101)	
Serum troponin I [ng/mL]	0.06 (0.0; 0.2)		
Serum total protein [mg/dL]	7.3 ± 4.6		
Serum albumin [mg/dL]	3.8 ± 0.6		
Serum TSH [μIU/mL]	1.6 (0.6; 2.8)		
Serum CRP [mg/L]	12.5 (6; 34)		
pO_2 [mm Hg]^x	66.9 ± 20.4		
pCO_2 [mm Hg]^x	37.9 ± 11.4		
sO_2 [%]^x	91 ± 6		
pH^x	7.4 ± 0.1		

Reported values are means ± SD or medians (lower and upper quartile); ^{x}capillary blood gases; *$p < 0.01$; **$p < 0.001$ for the difference between day 1 and day 3; MDRD — modification of diet in renal disease; GOT — glutamic oxaloacetic aminotransferase; GPT — glutamic pyruvic aminotransferase; GGTP — γ-glutamylotranspeptidase; TSH — thyroid-stimulating hormone; CRP — C-reactive protein; NT-proBNP — N-terminal pro-B-type natriuretic peptide; pO_2 — oxygen tension; pCO_2 — carbon dioxide tension; sO_2 — oxygen blood saturation

DISCUSSION

In our report, we presented the characteristics of patients hospitalised due to acute HF in one Polish centre specialising in the treatment of HF patients. The patients were mostly elderly men (mean age 68 years) with multiple concomitant diseases. Most of them were previously diagnosed with HF and had LV systolic dysfunction. A similar patient profile was reported in large international registries. Patients included into the ADHERE and OPTIMIZE-HF registries were older (about 72 years), with equal proportion of both sexes [2, 6]. Patient characteristics similar to what we found in our study was reported in the pilot findings of the ESC-HF Pilot study, and in the EuroHeart Failure Survey II (EHFS II) [9, 10].

It should be stressed that drug treatment before admission was suboptimal, which have predisposed to more frequent episodes of decompensation. Only 67% of all patients (77% among patients with established chronic HF) were treated with ACEI or angiotensin receptor antagonists (ARB), 66% of patients received beta-blockers (78% among patients with established chronic HF), and only 37% of pa-

Table 3. Comparison of clinical characteristics of patients with acute heart failure who were discharged alive versus those who died in hospital

Parameter	Patients discharged alive (n = 247)	Patients who died in hospital (n = 23)
De novo HF decompensation	66 (27)	6 (26)
Gender (women)	72 (29)	6 (26)
Age [years]	68 ±12	68 ±13
Left ventricular ejection fraction [%]	36 ± 14	35 ± 16
Left ventricular ejection fraction ≤ 45 [%]	160 (65)	11 (48)
Left ventricular end-diastolic dimension [mm]	62 ± 11	63 ± 14
Interventricular septum thickness [mm]	12 ± 3	12 ± 3
Right ventricular dimension [mm]	28 ± 7	30 ± 8
Left atrial dimension [mm]	49 ± 8	52 ± 13
Aetiology of HF:		
Ischaemic	137 (55)	11 (48)
Arterial hypertension	61 (25)	4 (17)
Valvular heart disease	49 (20)	8 (35)
Myocarditis/infection	21 (8)	2 (9)
Toxic	12 (5)	0 (0)
Acute HF type:		**
Decompensation of chronic HF	140 (57)	11 (48)
Pulmonary oedema	51 (21)	2 (9)
Acute HF with blood pressure elevation	30 (12)	0 (0)
Acute right ventricular HF	24 (10)	6 (26)
Acute HF in an acute coronary syndrome	18 (7)	4 (17)
Cardiogenic shock	2 (1)	4 (17)
Haemodynamic profile:		**
Warm-wet	204 (83)	11 (48)
Warm-dry	34 (14)	3 (13)
Cold-wet	6 (2)	5 (22)
Cold-dry	3 (1)	4 (17)
Most likely cause of acute HF:		
Inadequate diuretic therapy	112 (45)	5 (22)*
Blood pressure elevation	64 (26)	1 (4)*
Acute coronary syndrome	18 (7)	4 (17)
Tachyarrhythmia	45 (18)	4 (17)
Infection	23 (9)	2 (9)
Concomitant diseases and procedures:		
Ischaemic heart disease	136 (55)	12 (52)
History of myocardial infarction	90 (36)	9 (39)
Previous coronary angioplasty	56 (23)	3 (13)
Previous coronary artery bypass grafting	29 (12)	3 (13)
Hypertension	151 (62)	10 (46)
Atrial fibrillation	108 (44)	11 (48)
Diabetes	97 (39)	9 (39)
History of renal failure	94 (38)	12 (52)
History of anaemia	38 (15)	8 (35)*
Thyroid disease	28 (12)	4 (17)
History of stroke and/or TIA	35 (14)	1 (4)
Cancer	6 (2)	2 (9)
Medications used before hospitalisation:		
ACEI	142 (58)	8 (36)
ARB	25 (10)	5 (22)
Beta-blocker	166 (67)	12 (55)
Aldosterone antagonist	90 (37)	8 (36)
Diuretic	181 (74)	17 (77)
Digoxin	51 (21)	8 (36)
Statin	120 (49)	10 (46)
ASA	120 (49)	10 (46)
Oral anticoagulant	72 (29)	7 (32)

Reported values are means ± SD or n (%); *$p < 0.05$; **$p < 0.001$; abbreviations as in Tables 1 and 2

Table 4. Comparison of laboratory parameters and vital signs between patients with acute heart failure (n = 270) who were discharged home or died in hospital

Parameter	Patients discharged home (n = 247)			Patients who died in hospital (n = 23)		
	Day 1 (D1)	Day 3 (D3)	D3–D1	Day 1 (D3)	Day 3 (D3)	D3–D1
Body weight [kg]	80.7 ± 16	79.9 ± 16	–1,7 (–3.3; –0.5)	76.5 ± 15	74.1 ± 13	–1.1 (–1.8; 0.4)
Systolic blood pressure [mm Hg]	132 ± 32	115 ± 20	–18 (–32; 0)	105 ± 29***	100 ± 19**	–5 (–14; 0)
Diastolic blood pressure [mm Hg]	76 ± 16	69 ± 10	–5 (–16; 0)	65 ± 16**	62 ± 6**	1 (–8; 10)
Mean blood pressure [mm Hg]	94 ± 20	85 ± 12	–9 (–20; 0)	79 ± 19***	75 ± 8**	1 (–10; 12)
Heart rate [bpm]	90 ± 23	79 ± 15	–8 (–24; 4)	87 ± 19	81 ± 11	0 (–6; 5)
Haemoglobin [g/dL]	13.1 ± 2.1	12.5 ± 2.1	–0.4 (–1.1; 0.2)	12.7 ± 2.5	11.1 ± 2.4**	–0.7 (–1.5; –0.3)
Haematocrit [%]	39 ± 6	37.5 ± 6.1	–1.3 (–3.7; 0.8)	38 ± 7	33.7 ± 7.4*	–1.8 (–4.5; –0.9)
Leukocyte count [G/L]	10.2 ± 9.7	8.1 ± 3.0	–1 (–2.4; –0.1)	11.1 ± 5.7	9.7 ± 5.4*	–0.6 (–3.4; 1.8)
Platelet count [G/L]	225 ± 87	203 ± 71	–16 (–41; 5)	222 ± 114	185 ± 80	–17 (–63; 14)
Serum Na^+ [mmol/L]	140 ± 4	140 ± 4	0 (–2; 2)	136 ± 5***	136 ± 6***	0 (–2; 2)
Serum K^+ [mmol/L]	4.2 ± 0.6	3.9 ± 0.5	0 (–1; 0)	4.2 ± 0.7	4.2 ± 0.6*	0 (0; 1)
Blood urea nitrogen [mg/dL]	24 (19; 33)	27 (19; 36)	1 (–4; 6)	40 (24; 65)**	64 (45; 77)***	1 (–1; 4)
Serum creatinine [mg/dL]	1.2 (1; 1.6)	1.2 (0.9; 1.5)	0 (–0.2; 0.1)	1,7 (1; 2.5)*	2.0 (1.2; 2.5)**	0 (–0.3; 0.2)
Glomerular filtration rate (MDRD equation) [mL/min/1.73 m^2]	57 ± 22	60 ± 34	1 (–6; 9)	45 ± 22**	38 ± 19**	–1 (–4; 4)
Random serum glucose [mg/dL]	158 ± 78			172 ± 70		
GOT [IU/L]	29 (21; 44)			25 (20; 34)		
GPT [IU/L]	27 (17; 46)			16 (14; 58)		
GGTP [IU/L]	75 (41; 120)			54 (46; 110)		
Serum bilirubin [mg/dL]	1.2 (0.8; 2)			1.8 (1.1; 2.9)		
Plasma NT-proBNP [pg/mL]	5217 (2818; 11431)	3560 (1711; 6738)	–1513 (–4514; –220)	6038 (3768; 18284)	11780 (5371; 18912)**	1951 (–493; 4860)***
Change in plasma NT-proBNP in comparison to day 1 [%]	x	66 (46; 97)		x	123 (100; 138)***	
Serum troponin I [ng/mL]	0.1 (0; 0.2)			0.1 (0.1; 2.3)		
Serum total protein [mg/dL]	7.4 ± 4.7			6.4 ± 1.1		
Serum albumin [mg/dL]	3.9 ± 0.6			3.4 ± 0.5***		
Serum TSH [μIU/mL]	2.4 ± 4.2			3.4 ± 2.8		
Serum CRP [mg/L]	27.0 ± 35.3			30.4 ± 26.6		
pO_2 [mm Hg]^x	75 ± 15			86 ± 46		
pCO_2 [mm Hg]^x	38 ± 11			39 ± 13		
sO_2 [%]^x	91 ± 5			91 ± 10		
pH^x	7.4 ± 0.1			7.4 ± 0.1		

Reported values are means ± SD or medians (lower and upper quartile); ^{x}capillary blood gases; *p < 0.05; **p < 0.01; ***p < 0.001 for the difference between day 1 values in patients who died in hospital *vs* survivors; day 3 values in patients who died in hospital *vs* survivors; and changes at day 3 compared to day 1 in patients who died in hospital *vs* survivors; abbreviations as in Table 2

tients received an aldosterone antagonist (48% among patients with established chronic HF). Much more patients received a diuretic — 73% overall and 87% among those with established chronic HF. Of note, nearly three fourths of patients in our study were previously diagnosed with HF. In previously published studies, these proportions were even more unfavourable. In the ADHERE and OPTIMIZE-HF registries, only 52% of patients were receiving renin–angiotensin system inhibitor, and only 7% of patients were treated with aldosterone antagonist in the OPTIMIZE-HF registry [2, 6]. In the EHFS II study, proportion of patients treated with ACEI/ARB was similar to that in our study (63%), while fewer patients were treated with beta-blocker and aldosterone antagonist (43% and 28%, respectively) [10]. In the Finnish Acute Heart Failure Study FINN-AKVA, as many as 76% patients were on beta-blocker tre-

atment on admission, and the rate of ACEI/ARB use was similar to that in our group [11]. It should be noted that in addition to optimal drug management, the current ESC guidelines also highlight the role of educating patients with HF to prevent decompensation and improve quality of life [7]. This is an important issue also in view of our findings indicating inappropriate diuretic treatment as the most common cause of decompensation, as this aspect of the management is significantly affected by properly educated patients

In-hospital mortality in our study was 8.5%, slightly higher than reported by other authors [1–4]. In large US registries, in-hospital mortality was about 4%, and in the European registries it ranged from 3.8% (ESC-HF Pilot) to 6.7% (EHFS II) and 7.1% (FINN-AKVA) [5, 6, 9–11]. Higher mortality in our patient population may reflect the fact that our centre, specialising in the treatment of HF, is a tertiary referral centre for most severe cases in our region (about 4% of patients were referred from other hospitals in the same voivodeship).

As indicated by data from the US registries, the most important prognostic factors among patients with acute HF include simple clinical parameters (BP) and basic laboratory parameters (creatinine level, BUN) [5, 6]. In our study, we also noted a relationship between in-hospital outcomes in patients with acute HF and BP on admission, with much higher in-hospital mortality among patients with low BP on admission [5, 6]. Another very important factor affecting in-hospital mortality was renal function assessed based on serum creatinine, eGFR, and BUN [6]. Patients who died in hospital, had poorer baseline renal function that worsened even further during the first 48 h of treatment, which may be explained by haemodynamic disturbances present on admission (these patients more frequently had lower BP and showed a "cold" haemodynamic profile indicating organ hypoperfusion), and probably also by use of higher diuretic doses. Of interest, patients with serum K^+ level reduction after 48 h of treatment fared better and it may be speculated that this was related to a better response to diuretics, leading to hypokalemia. In contrast, we did not find any serum Na^+ level changes during standard in-hospital treatment, although similarly to other authors, we observed much worse outcomes in patients with hyponatremia [12, 13]. Of note, BP and serum Na^+ on admission were the strongest factors differentiating between patients with good or poor in-hospital outcomes in our study population.

In our centre, we routinely measure plasma NT-proBNP level on admission in patients with acute HF, and later we monitor changes of this parameter during the treatment (in this case, with initial follow-up measurement at 48 h). This short period of time seems sufficient to evaluate initial response to treatment, particularly in view of a short half-time of this natriuretic peptide (1–2 h) and previous studies evaluating its dynamics in patients with acute HF (with changes in its level noted already after 24 h) [14]. Until now, the effects of drug therapy on circulating NT-proBNP and outcomes were the subject of intensive research, but mostly in ambulatory patients with chronic HF [15, 16]. However, the results are mixed [15, 16]. Only a few studies examined the relationship between plasma NT-proBNP level and outcomes and indicated that higher levels of this natriuretic peptide on discharge are a risk factor for cardiovascular events during a short-term follow-up [14, 17]. In the ADHERE registry, in which more than half of patients had BNP level measured during the first 24 h of hospitalisation, a nearly linear relationship was found between this single measurement and in-hospital mortality [18]. The BNP level on admission was an independent prognostic factor for in-hospital mortality [18]. In addition, lack of its reduction during hospitalisation was a risk factor for poor outcomes in patients with acute HF [19]. In a retrospective analysis of the Survival of Patients with Acute Heart Failure in Need of Intravenous Inotropic Support (SURVIVE) study, the effects of BNP level changes during hospitalisation on outcomes were studied [20]. Patients with BNP level reduction on the fifth day of hospital stay had much lower mortality at 31 and 180 days [20].

In our registry, baseline plasma NT-proBNP level did not differentiate between patients with good or poor short-term outcomes, and only dynamics of changes of this parameter identified patients at particularly mortality high risk. Among patients who survived, a reduction in plasma NT-proBNP level was observed, probably reflecting the response of the cardiovascular system to initial treatment and the degree of cardiac overload [14]. Based on our findings, only the dynamics of plasma NT-proBNP level changes but not its baseline level is an important parameter identifying patients at risk of in-hospital death. Of note, similarly to other authors, we observed a dynamics of plasma NT-proBNP level changes within just 48 h of follow-up [14].

CONCLUSIONS

The population of patients hospitalised due to acute HF in a single cardiology centre in Poland was similar to analogous patient populations characterised in international multicentre registries. In addition to established predictors of poor prognosis, such as BP, renal function and serum Na^+ level, the dynamics of changes of some other parameters that was observed during the hospitalisation (serum K^+ level, renal function, plasma NT-proBNP level) may also be useful in identification of acute HF patients at a higher risk of in-hospital mortality.

Conflict of interest: *none declared*

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Charakterystyka kliniczna i czynniki prognozujące śmiertelność wewnątrzszpitalną u 270 kolejnych chorych hospitalizowanych z powodu ostrej niewydolności serca w jednym ośrodku kardiologicznym w ciągu roku

Jan Biegus[1], Robert Zymliński[1], Joanna Szachniewicz[1], Paweł Siwołowski[1], Aleksander Pawluś[3], Waldemar Banasiak[1], Ewa A. Jankowska[1, 2], Piotr Ponikowski[1, 2]

[1]Ośrodek Chorób Serca, Klinika Kardiologii, 4. Wojskowy Szpital Kliniczny, Wrocław; [2]Klinika Chorób Serca, Akademia Medyczna, Wrocław; [3]Studenckie Koło Naukowe przy Klinice Chorób Serca, Akademia Medyczna, Wrocław

Streszczenie

Wstęp: Ostra niewydolność serca (HF) stanowi poważny problem kliniczny i wiąże się z wysoką śmiertelnością wewnątrzszpitalną i częstymi rehospitalizacjami w obserwacji krótkoterminowej.

Cel: Celem rejestru było określenie charakterystyki klinicznej chorych hospitalizowanych z powodu ostrej HF oraz wyodrębnienie czynników złego rokowania wśród tych pacjentów.

Metody: Prospektywny rejestr objął wszystkich pacjentów hospitalizowanych z powodu ostrej HF od października 2008 do listopada 2009 r. w jednym ośrodku kardiologicznym. Analizie poddano stan kliniczny chorych i badania laboratoryjne przy przyjęciu i po 48-godzinnej hospitalizacji.

Wyniki: Badaniem objęto 270 chorych (wiek: 68 ± 13 lat, mężczyźni: 71%, *de novo* ostra HF: 27%, etiologia niedokrwienna HF: 55%, dekompensacja przewlekłej HF: 56%, profil ciepły-mokry: 80%). Śmiertelność wewnątrzszpitalna wyniosła 8,5% (n = 23). Nie stwierdzono różnic między chorymi, którzy przeżyli, i pacjentami, którzy zmarli w trakcie hospitalizacji w zakresie: wieku, płci, etiologii HF, częstości występowania ostrej HF *de novo*, wartości wyjściowych oraz zmian w trakcie hospitalizacji częstości rytmu serca i masy ciała (wszystkie $p > 0,2$). Wśród pacjentów, którzy zmarli, w porównaniu z chorymi, którzy zostali wypisani ze szpitala, częściej występował wstrząs kardiogenny i prawokomorowa HF (17% *v.* 1%; 22% *v.* 2%; $p < 0,001$), profile: zimny-mokry i zimny-suchy (22% *v.* 2% i 17% *v.* 1%; $p < 0,001$). Najczęstszym bezpośrednim czynnikiem sprawczym ostrej HF w grupie pacjentów, którzy zmarli, był ostry zespół wieńcowy (17% *v.* 7%), natomiast wzrost ciśnienia tętniczego i nieodpowiednie leczenie diuretyczne dominowały wśród osób, które przeżyły (26% *v.* 4% i 45% *v.* 22%, wszystkie $p < 0,05$). Wyjściowe średnie ciśnienie tętnicze i stężenia Na^+ w surowicy były wyższe u pacjentów, którzy przeżyli, w porównaniu z chorymi, którzy zmarli (94 ± 20 *v.* 79 ± ± 19 mm Hg, 140 ± 4 *v.* 136 ± 5 mmol/l; $p < 0,001$) i pozostawały wyższe w 3. dobie obserwacji. Nie stwierdzono różnic w wyjściowym stężeniu hemoglobiny i K^+ w surowicy między obiema grupami. U pacjentów, którzy zmarli, stężenie hemoglobiny spadło po 48 h leczenia (12,7 ± 2,5 *v.* 11,1 ± 2,4 g/dl; $p < 0,01$), podczas gdy tylko u pacjentów, którzy przeżyli, obserwowano zmniejszenie stężenia K^+ w surowicy po 48 h (4,2 ± 0,6 *v.* 3,9 ± 0,5 mmol/l; $p < 0,05$), co może odzwierciedlać skuteczność leczenia moczopędnego. Chorzy, którzy zmarli w szpitalu, w porównaniu z tymi, co przeżyli, charakteryzowali się gorszą wyjściową funkcją nerek [stężenie kreatyniny w surowicy: 1,7 (1; 2,5) *v.* 1,2 (1;1,6) mg/dl, stężenie azotu mocznikowego w surowicy: 40 (24; 65) *v.* 24 (19; 33) mg/dl; $p < 0,05$); funkcja ta ulegała dalszemu pogorszeniu w trakcie hospitalizacji [do 2,0 (1,2; 2,5) *v.* 1,2 (0,9; 1,5) mg/dl i 64 (45; 77) *v.* 27 (19; 36) mg/dl; $p < 0,01$]. Wyjściowe stężenie NT-proBNP w osoczu nie różniło się między grupami, ale po 48 h stężenie NT-proBNP w osoczu było niższe w grupie chorych, którzy przeżyli, w porównaniu z pacjentami, którzy zmarli — 3560 (1711; 6738) *v.* 11780 (5371; 18912) pg/ml; $p < 0,01$; dane podane jako mediana (dolny, górny kwartyl).

Wnioski: Śmiertelność wewnątrzszpitalna obserwowana w badanej populacji jest nieznacznie większa od opisywanej w innych rejestrach. Wyjściowe wartości wybranych parametrów (ciśnienie tętnicze krwi, stężenie Na^+ w surowicy, funkcja nerek) oraz zmiany wybranych parametrów po 48 h leczenia (stężenie K^+ w surowicy, parametry funkcji nerek i stężenie NT-proBNP) mogą pomóc w identyfikacji pacjentów o wyższym ryzyku zgonu w trakcie hospitalizacji.

Słowa kluczowe: ostra niewydolność serca, rejestr, stratyfikacja ryzyka, rokowanie

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Adres do korespondencji:
dr hab. n. med. Jan Biegus, Klinika Kardiologii, 4. Wojskowy Szpital Kliniczny, ul. Weigla 5, 50–540 Wrocław, e-mail: janbiegus@o2.pl
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