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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Acute heart failure in patients admitted to the emergency department with acute myocardial infarction

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Abstract

Background: Acute heart failure (AHF), occurring as a complication of ongoing acute myocardial infarction (AMI), is a common predictor of worse clinical outcome. Much less is known about the unique subpopulation of patients who present these two life-threatening conditions in the emergency department (ED).

Aim: The aim of the study was to establish the prevalence of coexistence of AHF with AMI in the ED, to identify clinical factors associated with the higher prevalence of AHF at very early onset of AMI, and to assess the prognostic impact of the presence of AHF with AMI.

Methods: A prospective study of 289 consecutive patients (mean age: 68 ± 11 years, 61% men) admitted to our institution (via the ED) with the diagnosis of AMI between May and October 2012 and followed-up for 2.5 years.

Results: Acute heart failure was diagnosed in 13% of patients in the ED. In multivariable analysis, female sex, chronic obstructive pulmonary disease, and chronic kidney disease significantly increased the risk of developing AHF together with AMI (all $p < 0.05$). Patients with AHF were hospitalised for longer (9.2 ± 6.1 vs. 6.3 ± 4.5 days, $p < 0.001$), had higher in-hospital cardiovascular mortality (8% vs. 0%, $p < 0.001$), and all-cause (34% vs. 15%, $p = 0.004$) and cardiovascular mortality (26% vs. 9%, $p = 0.002$) during long-term follow-up.

Conclusions: Despite good logistic- and evidence-based treatment, AHF is present in one in eight patients with AMI at the time of admission to the ED. Particularly poor outcomes characterise critically ill patients; therefore, great effort should be undertaken to improve their care.

Key words: acute cardiac care, acute decompensated heart failure, acute myocardial infarction, acute heart failure, heart failure

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INTRODUCTION

The natural history of patients with coexisting acute myocardial infarction (AMI) and acute heart failure (AHF) is extremely fatal [1–5]. Although the rates of heart failure (HF) occurring in the later phase of AMI have been reduced in recent decades due to the introduction of modern pharmacotherapy and primary percutaneous coronary intervention [2, 3, 6], it still remains unclear regarding AHF presenting together with the acute phase of AMI in the emergency department (ED) [2, 3, 7]. Some factors may potentially interfere with the prevalence of AHF in AMI, such as aging population, numerous comorbidities, episodes of recurrent AMI, and the presence of chronic HF [7]. However, there are no evidence-based data analyses of this phenomenon.

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Therefore, we aimed to assess prospectively the prevalence of AHF on admission to the ED among patients hospitalised due to AMI, to identify clinical factors that promote the occurrence of AHF, and to evaluate the long-term outcome in patients treated according to contemporary recommendations.

METHODS

Study population

Patients consecutively admitted to the Centre for Heart Diseases via the ED of the Clinical Military Hospital in Wroclaw, Poland between May and October 2012, eligible for enrolment to our study, had to fulfil the following criteria of inclusion:

- age ≥ 18 years;
- admission to the hospital due to AMI (ST-elevation myocardial infarction [STEMI] or non-ST-elevation myocardial infarction [NSTEMI]). Diagnosis of AMI was made according to the Third Universal Definition of Acute Myocardial Infarction. Therefore, all patients had to present dynamic changes (a rise and/or a fall) in high-sensitivity cardiac troponin I (hs-cTnI) values (including at least one value above 99th percentile upper reference limit) and at least one of the following symptoms: ischaemia (angina pectoris) and/or new ischaemic features in electrocardiogram (significant ST-segment-T wave changes or new left bundle branch block or development of pathological Q waves) [8];
- written informed consent to participate in the study.

The exclusion criterion was subsequent admission due to recurrent AMI during the enrolment period.

Two groups of patients were compared in this study: patients who presented AHF and AMI on admission to the ED were classified as the AMI-AHF(+) group and those without decompensation as the AMI-AHF(–) group. AHF was strictly diagnosed based on two obligatory criteria:

- signs and symptoms of AHF (dyspnoea at rest or on minimal exertion, pulmonary congestion presented during physical examination and confirmed on chest X-ray) or cardiogenic shock;
- appropriate therapy for AHF (loop diuretic IV and/or nitroglycerin IV administered within the first 24 h of hospitalisation).

The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee.

Study protocol

Each patient underwent a physical examination followed by standard diagnostic tests. In our study we not only analysed patients' hospital admission laboratory data, but also observed the maximum level of serum hs-cTnI during their hospitalisation. Plasma N-terminal pro-B type natriuretic peptide (NT-proBNP [pg/mL]) was measured using electrochemiluminescence on the Elecsys 1010/2010 System (Roche Diagnostics GmbH, Mannheim, Germany). Renal function was assessed using the estimated glomerular filtration rate (eGFR [mL/min/1.73 m^2]), calculated from the Modification of Diet in Renal Disease equation. Hs-TnI (ng/mL) was measured using chemiluminescence (technology LOCI) on the Dimension EXL System (Siemens Healthcare Diagnostics, Erlangen, Germany). Echocardiography was performed at the discretion of the treating physician, typically as soon as it was possible, in the vast majority of patients in the first 48 h from admission to hospital.

Follow-up

The following events were analysed: all-cause deaths, cardiovascular deaths, urgent HF hospitalisations, non-fatal myocardial infarctions, and non-fatal strokes. Additionally, the length of index hospitalisation was also observed. Follow-up was censored at 30 days, six months, and 2.5 years. Information regarding the aforementioned events was obtained from the Polish National Health Fund, the hospital computer system, and confirmed by patients or their relatives during phone calls.

Statistical analysis

Normally distributed continuous variables were presented as means ± standard deviations. Variables with a skewed distribution were expressed as medians with lower and upper quartiles, and were log transformed in order to normalise their distributions. The intergroup differences were tested using Student's t-test. The categorical variables were expressed as numbers with percentages. The inter-group differences were tested using the χ^2 test.

Univariable and multivariable linear regression models were constructed in order to identify the patient's clinical profile (baseline parameters) that could favour the development of AHF in the course of AMI. All variables that had been shown to be significant predictors of AHF development in AMI in the univariable analyses were included into the multivariable model.

A value of $p < 0.05$ was considered statistically significant.

Statistical analyses were performed using the STATISTICA 10 data analysis software system (StatSoft, Inc.).

RESULTS

Between May and October 2012 a total of 337 patients were hospitalised in the Cardiology Department of the Clinical Military Hospital in Wroclaw due to AMI. A cohort of 289 (86%) patients fulfilled the inclusion criteria of our study. Forty-eight patients were not included into the study: eight (2%) patients because of recurrent AMI during the study period, 13 (4%) patients were not able to give written informed consent to participate in the study due to severe clinical status at admission to the hospital, and 27 (8%) patients refused to participate in the study. Among the patients enrolled into the study 38 (13%) experienced AHF or cardiogenic shock on admission to the ED and made up the study AMI-AHF(+) group; the remaining 251 patients made up the AMI-AHF(–) group.

Patients' population and pre-admission predictors of AHF

Patients with AMI accompanied by AHF were more likely to be women, with a history of hypertension, chronic obstructive pulmonary disease (COPD), and chronic kidney disease (CKD) (all $p < 0.01$; Table 1).

Patients with AMI and AHF on admission were characterised by: increased heart rate, higher diameter of left atrium, higher plasma concentration of NT-proBNP, glucose, thrombocytes, white blood cells, including neutrophils, and lower eGFR, serum urea, and haemoglobin level (all $p < 0.05$; Table 2).

In a multivariate analysis the presence of COPD and also female sex were associated with a ca. five-fold increased risk of developing AHF together with AMI. Similarly, coexisting CKD increased the risk of developing AHF with the acute phase of AMI by 2.5 times, whereas the presence of hypertension was associated with a greater risk of developing AHF only in univariate analysis (Table 1).

Treatment patterns

Treatment of AMI and AHF was performed according to the European guidelines.

In both studied groups the major diagnosis of AMI was NSTEMI (84% vs. 69%), and serum hs-cTnI was on similar maximum level in each group (6.7 vs. 4.0; all p non-significant). Coronarography was performed in almost all patients with AMI (96%). Primary revascularisation was done in 95% of patients with STEMI, and revascularisation was performed in 82% of patients with NSTEMI, who had coronary artery lesions assessed as significant (no difference between studied groups according to the presence of AHF on admission was observed). Left main coronary artery and left anterior descending artery was more than 1.5 times more often the cause of angina symptoms in patients with AHF than in those without decompensation (58% vs. 34%, $p = 0.008$) (Table 3).

Patients with AMI and AHF were more likely to be prescribed the following at discharge: beta-blockers, mineralocorticoid receptor antagonists, loop diuretics, and digoxin, but less likely to be prescribed angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (all $p < 0.05$, Fig. 1).

Hospital outcomes

Patients from the AMI-AHF(+) group were hospitalised for longer and experienced higher in-hospital all-cause and cardiovascular mortality than patients without AHF on admission (Table 4).

Post-discharge outcomes

All-cause mortality, including cardiovascular, was significantly higher in the AMI-AHF(+) group as compared to the AMI-AHF(–) group during short- and long-term follow-up (Table 4, Fig. 2). Detailed causes of deaths are presented in Figure 3. Strokes occurred similarly often in both studied groups. Patients from the AMI-AHF(+) group were 2.5-times more often hospitalised due to HF and more than two-times more often experienced recurrent myocardial infarction during a 2.5-year follow-up (Table 4).

DISCUSSION

Despite the fact that several studies describing the problem of AHF in the setting of AMI have already been published, most of them have three main limitations: (a) they analysed the whole period of hospitalisation (both on admission and during hospital stay); (b) some important groups of patients were excluded, e.g. those with chronic HF; and (c) they over-diagnosed decompensation because the definition of AHF was based only on one criteria (the presence of pulmonary rales or alveolar/interstitial oedema by radiograph) [2, 6, 9, 10]. Therefore, there was a need for a study that would focus only on the time of the admission to the ED, to exclude AHF possibly connected with in-hospital management or caused iatrogenically. In our paper we presented an AHF group consisting of all patients admitted firstly to the ED with AMI and signs and symptoms of AHF (all criteria of AHF had to be met: clinical, radiological, and therapeutic), and later to the cardiology ward. We hope that this study will be found particularly useful in daily clinical practice in ED.

In contemporary treatment, the prevalence of AHF occurring together with AMI at the same time of admission to the ED remains unknown. Our study demonstrates that currently about 13% of patients with AMI present circulatory decompensation on admission to hospital. The majority of them (76%) experience AHF de novo, whereas one-fourth of the patients were previously diagnosed with chronic HF.

Taking into account all our patients ($n = 289$), AHF that developed during the whole hospitalisation period was diagnosed in 15% of patients, and in 86% of them already on admission. We observed that the majority of AHF in patients with AMI was present already on admission to the ED. The reasons for this profile of the prevalence of AHF may be attributed to the considerable advances in AMI treatment, including early pharmacotherapy and revascularisation, whereas the management of patients presenting simultaneously AHF and AMI on the ED remains an urgent challenge for modern medicine [2, 10].

We are familiar with many HF long-term predictive factors of AMI, but we still have very poor knowledge how to predict the occurrence of AHF in an acute phase of manifestation of ischaemia [11–16]. Some clinical features and symptoms prior to hospital admission have a prognostic value [17–19]. Therefore, it is highly important to establish the predictive factors of AHF in the presence of AMI and identify patient groups at high risk.

Multivariable analysis in our study revealed that the female sex has a greater risk of AHF occurrence on admission due to AMI. Our finding is consistent with previous reports, but most studies refer again to AHF developed during the whole hospitalisation period [9, 15, 20–22]. Our multivari-

Table 1. Baseline characteristics of the studied population — comparison between group of patients admitted due to acute myocardial infarction (AMI) with acute heart failure (AHF) and group of patients with AMI without AHF on admission to the hospital

Parameters	All recruited patients with AMI (n = 289)	AMI-AHF(+) N = 38 (13%)	AMI-AHF(–) N = 251 (87%)	Logistic regression models: Patients who developed AHF on admission vs. patients without circulatory decompensation			
				Univariable models		Multivariable model	
				OR (95% CI)	χ^2	OR (95% CI)	χ^2
Age (years)	68 ± 11	69 ± 11	67 ± 11	1.02	0.99–1.05	–	–
Age > 75 years	86 (30%)	12 (38%)	74 (29%)				
Women	114 (39%)	27 (71%)	87 (35%)[c]	4.6	2.2–9.8[c]	5.01	2.3–11.1[c]
Current or previous smoker	171 (59%)	18 (47%)	153 (61%)	0.6	0.3–1.2	–	–
Hypertension	235 (81%)	37 (97%)	198 (79%)[b]	9.9	1.3–74.5[a]	7.1	0.9–54.5
Dyslipidaemia	228 (79%)	33 (87%)	195 (78%)	1.9	0.7–5.1	–	–
Diabetes mellitus	76 (26%)	14 (37%)	62 (25%)	1.8	0.9–3.7	–	–
History of atrial fibrillation	62 (21%)	9 (24%)	53 (21%)	1.2	0.5–2.6	–	–
Previous stroke/TIA	27 (9%)	4 (11%)	23 (10%)	1.2	0.4–3.6	–	–
Previous AMI	98 (34%)	13 (34%)	85 (34%)	1.02	0.5–2.1	–	–
History of CHF	49 (17%)	9 (24%)	40 (16%)	1.6	0.7–3.7	–	–
Anaemia	30 (10%)	3 (8%)	27 (11%)	0.7	0.2–2.5	–	–
Chronic obstructive pulmonary disease	19 (7%)	7 (18%)	12 (5%)[b]	4.5	1.6–12.3[b]	4.9	1.6–15.3[b]
Chronic kidney disease	52 (18%)	13 (34%)	39 (16%)[b]	2.8	1.3–6.02[b]	2.5	1.1–5.8[a]

[a]$p < 0.05$; [b]$p < 0.01$; [c]$p < 0.001$. Data are presented as a number of patients (with percentage); AMI-AHF(+) — a group of patients admitted to the hospital due to AMI with AHF; AMI-AHF(–) — a group of patients admitted to the hospital due to AMI without AHF; CI — confidence interval; CHF — chronic heart failure; OR — odds ratio; TIA — transient ischaemic attack

Table 2. Different clinical pattern of patients admitted with acute myocardial infarction (AMI) with versus without acute heart failure (AHF)

Parameters	AMI-AHF(+) N = 38 (13%)	AMI-AHF(–) N = 251 (87%)
Clinical status on admission		
Systolic BP [mm Hg]	143 ± 35	138 ± 24
Diastolic BP [mm Hg]	78 ± 16	77 ± 13
Heart rate [bpm]	83 [75–100]	75 [65–83][b]
Peripheral oedema	9 (24%)	14 (6%)[c]
Killip class: I/II/III/IV	0/18 (46%)/ /18 (46%)/2 (5%)	–
Echocardiography		
LVEF [%]	39 ± 10	50 ± 11[c]
LVEF < 40%	20 (54%)	39 (16%)[c]
LVEDD [mm]	52 ± 7	50 ± 7
IVSD [mm]	13 [11–14]	13 [12–14]
Left atrium [mm]	43 ± 5	40 ± 6[b]
RVEDD [mm]	26 ± 4	26 ± 4
Laboratory results		
Hs-TnI* [ng/mL]	6.7 [0.9–25.2]	4.0 [0.7–24.7]
NT-proBNP [pg/mL]	4128 [2397–9261]	742 [259–2043][c]
Haemoglobin [g/dL]	13.0 ± 1.6	13.6 ± 1.8[a]
PLT count [$10^3/\mu L$]	234 [200–290]	214 [179–254][a]
WBC count [thousands/mm^3]	10.9 [8.3–14.3]	8.5 [6.9–10.6][b]
Neutrophiles [cells/μL]	8.1 [5.2–11.0]	5.8 [4.3–7.8][c]
Monocytes [cells/μL]	0.6 [0.5–0.8]	0.6 [0.5–0.8]
Lymphocytes [cells/μL]	1.9 [1.2–2.3]	1.7 [1.3–2.2]
eGFR [mL/min/1.73 m^2]	62 ± 27	76 ± 26[b]
Urea [mg/dL]	44 [31–64]	37 [30–48][a]
Sodium [mmol/L]	140 ± 3	140 ± 3
Glucose [mg/dL]	155 [109–246]	125 [109–156][a]
GGPT [IU/L]	41 [29–66]	37 [27–59]
Bilirubin [mg/dL]	0.6 [0.4–1.1]	0.6 [0.4–0.9]
AlAT [IU/L]	25 [18–46]	24 [17–36]
AspAT [IU/L]	38 [23–70]	29 [20–48]
Implantable devices		
Pacemaker	0 (0%)	6 (2%)
ICD	2 (5%)	7 (3%)
CRT	0 (0%)	3 (1%)

*The highest level during hospitalisation; [a]$p < 0.05$; [b]$p < 0.01$; [c]$p < 0.001$. Data are presented as a mean ± standard deviation of the mean, a median [with lower and upper quartiles] or number of patients (percentage), where appropriate; AMI-AHF(+) — a group of patients admitted to the hospital due to AMI with AHF; AMI-AHF(–) — a group of patients admitted to the hospital due to AMI without AHF; AlAT — alanine aminotransferase; AspAT — aspartate aminotransferase; BP — blood pressure; CRT — cardiac resynchronisation therapy; eGFR — estimated glomerular filtration rate; GGTP — gamma-glutamyl transpeptidase; hs-TnI — high-sensitivity troponin I; ICD — implantable cardioverter defibrillator; IVSD — interventricular septal thickness at diastole; LVEDD — left ventricular end diastolic diameter; LVEF — left ventricular ejection fraction; NT-proBNP — N-terminal prohormone of B-type natriuretic peptide; PLT — platelets; RVEDD — right ventricular end diastolic diameter; WBC — white blood cells

Table 3. Comparison of major diagnosis and applied coronary interventions in patients admitted with acute myocardial infarction with [AMI-AHF(+)] versus without acute heart failure [AMI-AHF(+)]

Parameters	AMI-AHF(+) N = 38 (13%)	AMI-AHF(–) N = 251 (87%)
Major diagnosis		
STEMI/NSTEMI	16/84	31/69
Multivessel CAD	13	12
Coronary interventions		
Coronarography	92	97
PCI/CABG/no revascularisation	68/3/29	70/4/27
Culprit lesions		
Left main coronary artery	6	0.5[b]
Left anterior descending artery	52	33[a]
Right coronary artery	19	33
Circumflex artery	6	18
Unknown	16	17

[a]$p < 0.05$; [b]$p < 0.01$. Data are presented as a percentage of patients; AMI-AHF(+) — a group of patients admitted to the hospital due to AMI with AHF; AMI-AHF(–) — a group of patients admitted to the hospital due to AMI without AHF; CABG — coronary artery bypass graft; CAD — coronary artery disease; NSTEMI — non-ST-elevation myocardial infarction; PCI — percutaneous coronary intervention STEMI — ST segment elevation myocardial infarction

able analysis has indicated that chronic HF in a patient's history is not an independent predictive factor of developing AHF in the presence of AMI. This fact may confound, especially regarding very poor and contradictory data in this area [15]. Shah et al. [15] in their study indicate history of CHF as a predictor of AHF in AMI; however, AHF in this report has been analysed during the whole in-hospital period, thus it cannot be counterpoint to our result. In summary, this problem requires further attention from researchers and a much more thorough investigation.

There is a lack of data assessing COPD and CKD as an independent factor promoting development of AHF at the same time as the occurrence of AMI; therefore, it is difficult to critically discuss our results. However, there are some studies revealing that coexisting COPD and CKD in HF patients is associated with higher mortality. Macchia et al. [23] in an observational study on 1020 patients with CHF aged ≥ 60 years found that COPD was an independent short-term prognostic indicator of their mortality and cardiovascular comorbidity. According to the ADHERE registry, more than half of patients with all-cause AHF had at least moderate renal insufficiency (eGFR < 60 mL/min/1.73 m^2) on admission, which was associated with increased mortality [24].

Although there has been a significant decrease in the incidence rates of HF following AMI (almost threefold in

Table 4. Comparison of in-hospital and postdischarge outcomes in patients admitted with acute myocardial infarction with [AMI-AHF(+)] versus without acute heart failure [AMI-AHF(–)]

Parameters	AMI-AHF(+) N = 38 (13%)	AMI-AHF(–) N = 251 (87%)
Hospital outcomes		
Length of hospitalisation [days]	7 [6–10]	5 [4–7][c]
In-hospital all-cause mortality	3 (8%)	0 (0%)[c]
In-hospital cardiovascular mortality	3 (8%)	0 (0%)[c]
Outcomes at 30-day follow up		
All-cause mortality	5 (13%)	2 (1%)[c]
Cardiovascular mortality	4 (11%)	1 (0.4%)[c]
Outcomes at 6-month follow up		
All-cause mortality	10 (26%)	10 (4%)[c]
Cardiovascular mortality	6 (16%)	4 (2%)[a]
Outcomes at 2.5-year follow up		
All-cause mortality	13 (34%)	38 (15%)[b]
Cardiovascular mortality	10 (26%)	23 (9%)[b]
Non-fatal AMI	9 (24%)	28 (11%)[a]
Heart failure urgent hospitalisations	12 (32%)	33 (13%)[b]
Stroke	2 (5%)	9 (4%)

[a]$p < 0.05$; [b]$p < 0.01$; [c]$p < 0.001$. Data are presented as median (lower and upper quartile) or number of patients (with percentage); AMI-AHF(+) — a group of patients admitted to the hospital due to AMI with AHF; AMI-AHF(–) — a group of patients admitted to the hospital due to AMI without AHF

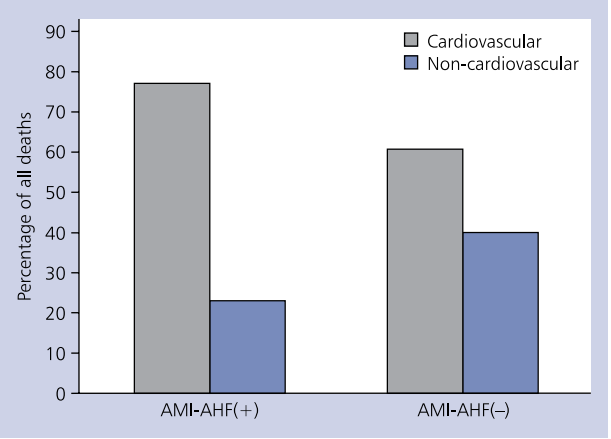

Figure 2. The percentage of cardiovascular and non-cardiovascular mortality rates between patients with acute heart failure on admission to the hospital due to acute myocardial infarction [AMI-AHF(+), number of all deaths = 13] and patients with AMI and without AHF [AMI-AHF(–) group, number of all deaths = 38] during the 2.5-year follow-up

the last four decades) due to significant achievement in treatment of AMI and HF, the prognosis of patients with AMI complicated by HF still remains extremely fatal [1–5]. McManus et al. [2] reported a decline in the prevalence of 30-day deaths in patients with AHF during index hospitalisation due to AMI between 1975 (29%) and 2003 (16%). In our study (2012) all-cause mortality on 30-day follow-up amounted to 13%, and after one year every third patient died of decompensation in the acute phase of AMI. The persistently high mortality rate indicates an urgent need for

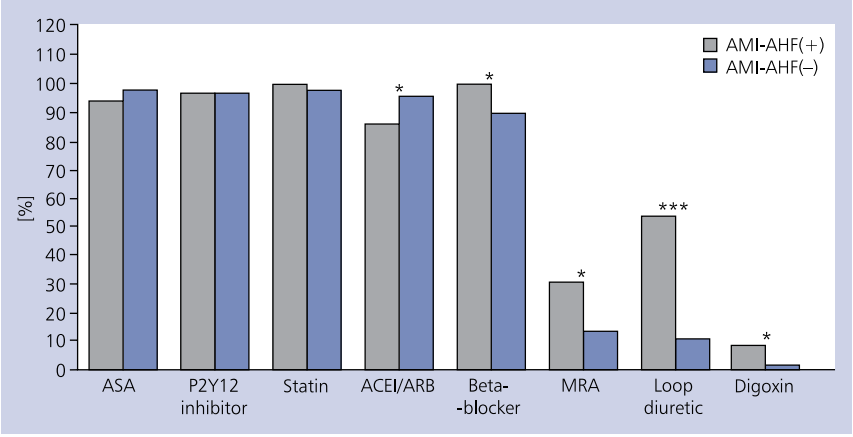

Figure 1. Comparison of pharmacotherapy applied at discharge from hospital between patients with acute heart failure on admission to the hospital due to acute myocardial infarction [AMI-AHF(+)] and patients with AMI and without AHF [AMI-AHF(–)]; ACEI — angiotensin converting enzyme inhibitors; ARB — angiotensin receptor blockers; ASA — acetylsalicylic acid; MRA — mineralocorticoid receptor antagonists; *$p < 0.05$; ***$p < 0.001$

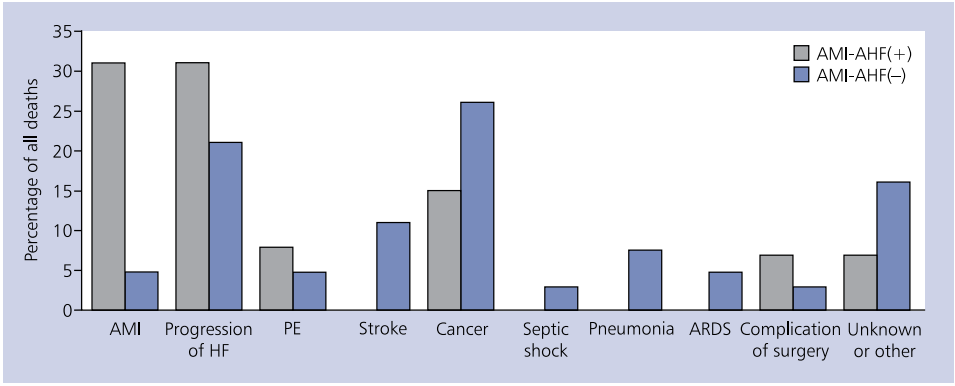

Figure 3. The percentage of detailed causes of death in patients with acute heart failure on admission to the hospital due to acute myocardial infarction [AMI-AHF(+), number of all deaths = 13] and patients with AMI and without AHF [AMI-AHF(–) group, number of all deaths = 38] during the 2.5-year follow-up; AMI — acute myocardial infarction; ARDS — acute respiratory distress syndrome; PE — pulmonary embolism

development of successful treatment and effective prevention of AHF, due to the fact that currently available management is undeniably insufficient.

Limitations of the study

Certain limitations must be considered during interpreting our study. Firstly, our study represents a small number of patients with AHF in the presence of AMI. Secondly, to participate in this study all patients had to give written informed consent, which may have introduced unexpected selection bias of participants. Further studies are required to confirm our findings on larger populations of patients with AHF at the time of admission to the ED due to AMI.

CONCLUSIONS

In our study we found that, despite contemporary treatment of AMI, the prevalence of AHF that develops in the course of AMI at the time of admission to the ED is still high and remains a significant clinical problem. Women and patients with coexisting COPD and CKD more often than other patients present AHF together with AMI during urgent hospital admission.

We have shown that chronic HF and diabetes mellitus are not predictive factors of AHF in the presence of AMI.

Despite optimal revascularisation, pharmacological therapy, and early rehabilitation, patients with AHF and AMI on admission to the ED have poorer prognosis during hospitalisation and after discharge from hospital: one third of these patients die within 2.5 years after discharge, most of them due to cardiovascular events, and more than one forth experiences new AMI and/or AHF during this time. A tenacious effort should be undertaken by modern medicine to improve care and outcomes in patients presenting AHF together with AMI during admission to the ED, starting with the pre-hospital management, via the ED, and continuing during the whole index hospitalisation.

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Conflict of interest: *none declared*

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Ostra niewydolność serca u chorych przyjmowanych z ostrym zawałem serca na szpitalny oddział ratunkowy

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Streszczenie

Wstęp: Ostra niewydolność serca (AHF) występująca jako powikłanie ostrego zawału serca (AMI) jest znanym czynnikiem istotnie pogarszającym rokowanie u osób z chorobą niedokrwienną serca. Dużo mniej jednak wiadomo o szczególnej subpopulacji chorych, u których już w trakcie przyjmowania na szpitalny oddział ratunkowy (SOR) współwystępują objawy tych dwóch zagrażających życiu stanów. Częstość występowania niewydolności serca występującej w późniejszej fazie zawału serca została wprawdzie znacznie zredukowana w ciągu ostatnich dziesięcioleci dzięki wprowadzeniu nowoczesnej farmakoterapii i przezskórnych zabiegów angioplastyki wieńcowej, jednak częstość współwystępowania AHF w ostrej fazie zawału serca pozostaje nieznana. Zwykle bowiem w badaniach klinicznych dotyczących leczenia AMI współwystępowanie AHF w trakcie rekrutacji do badania jest traktowane jako kryterium wykluczające — m.in. z tego powodu, brakuje aktualnych i wiarygodnych danych dotyczących tej grupy chorych.

Cel: Celem pracy było ustalenie częstości współwystępowania AHF z AMI u chorych przyjmowanych na SOR, identyfikacja klinicznych czynników związanych z częstszym występowaniem AHF wspólnie z AMI oraz ocena rokowania w badanej grupie pacjentów.

Metody: Przeprowadzono prospektywne badanie 289 kolejnych chorych z rozpoznaniem AMI, przyjmowanych podczas ostrego dyżuru do szpitala, w którym pracują autorzy niniejszej pracy, w okresie od maja do października 2012 r., a następnie poddanych 2,5-rocznej obserwacji (*follow-up*). Ostry zawał serca (z uniesieniem odcinka ST lub bez) rozpoznawano na podstawie trzeciej uniwersalnej definicji AMI. U wszystkich chorych stwierdzono dynamiczne zmiany w stężeniu wysokoczułej troponiny I (w tym co najmniej jedna wartość przekraczała 99. centyl górnej granicy przedziału wartości referencyjnych) i co najmniej jeden z wymienionych objawów: dławicę piersiową i/lub nowe lub przypuszczalnie nowe niedokrwienne zmiany w EKG (istotne zmiany odcinka ST i załamka T lub nowy blok lewej odnogi pęczka Hisa lub wystąpienie patologicznych załamków Q). W badaniu porównano populację pacjentów z AMI, która została podzielona na dwie grupy w zależności od obecności objawów AHF przy przyjęciu do szpitala. Pacjenci, którzy zostali zakwalifikowani do grupy badanej, musieli spełniać dwa kryteria rozpoznania AHF: 1) objawy podmiotowe i przedmiotowe AHF (duszność spoczynkowa lub przy minimalnym wysiłku, cechy zastoju w krążeniu płucnym podczas badania fizykalnego i potwierdzone na zdjęciu rentgenowskim klatki piersiowej) lub wstrząs kardiogenny; 2) stosowanie typowego leczenia AHF (diuretyki pętlowe i.v. i/lub nitrogliceryna i.v. podane w ciągu pierwszych 24 h hospitalizacji). Pacjenci byli obserwowani przez kolejne 2,5 roku (*follow-up*) i w tym czasie w analizie rokowania brano pod uwagę następujące zdarzenia: zgony z jakiejkolwiek przyczyny, zgony z przyczyn sercowo-naczyniowych, pilne hospitalizacje z powodu pogorszenia niewydolności serca, zawały serca i udary niezakończone zgonem. Ponado analizowano czas pobytu w szpitalu podczas pierwszej hospitalizacji (włączenia do badania). Wyniki z *follow-up* analizowano po upływie 30 dni, 6 miesięcy i 2,5 roku od czasu wystąpienia zawału serca i włączenia do badania.

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Wyniki: W trakcie trwania badania 337 pacjentów było hospitalizowanych w ośrodku, w którym pracują autorzy niniejszej pracy, z powodu AMI. 289 (86%) chorych spełniło kryterium włączenia do badania. Wśród nich 38 (13% — grupa badana) pacjentów prezentowało objawy AHF lub wstrząsu kardiogennego w momencie przyjmowania na SOR, pozostałych 251 chorych stanowiło grupę kontrolną. U osób z grupy badanej stwierdzono: wyższą częstość akcji serca przy przyjęciu, większy wymiar lewego przedsionka w badaniu ultrasonograficznym serca, wyższe stężenia NT-proBNP, glukozy i mocznika w surowicy, wyższe stężenia trombocytów, leukocytów, w tym neutrofili, niższy współczynnik przesączania kłębuszkowego i niższe stężenia hemoglobiny w porównaniu z pacjentami z grupy kontrolnej (dla wszystkich wymienionych parametrów: $p < 0,05$). W grupie badanej zmiana miażdżycowa odpowiedzialna za zawał znajdowała się 1,5-krotnie częściej w gałęzi zstępującej i pniu głównym lewej tętnicy wieńcowej niż u osób z grupy kontrolnej (58% *vs*. 34%, $p = 0,008$). W analizie wieloczynnikowej płeć żeńska, przewlekła obturacyjna choroba płuc i przewlekła choroba nerek znacząco zwiększały ryzyko wystąpienia AHF wspólnie z AMI (dla wszystkich: $p < 0,05$). Chorzy z AHF byli istotnie dłużej hospitalizowani (9,2 ± 6,1 *vs*. 6,3 ± 4,5 dnia; $p < 0,001$) i charakteryzowali się wyższą wewnątrzszpitalną śmiertelnością sercowo-naczyniową (8% *vs*. 0%, $p < 0,001$). Nie zaobserwowano różnic w częstości występowania udarów mózgu między analizowanymi grupami. Pacjenci z grupy AHF byli 2,5-krotnie częściej hospitalizowani z powodu niewydolności serca, 2-krotnie częściej doświadczali ponownego zawału serca, cechowali się wyższą śmiertelnością ogólną (34% *vs*. 15%; $p = 0,004$) i sercowo-naczyniową (26% vs. 9%; $p = 0,002$) w ciągu 2,5-letniego okresu obserwacji.

Wnioski: Mimo dobrej logistyki i stosowania medycyny opartej na faktach w praktyce klinicznej AHF występuje u jednego na ośmiu pacjentów przyjmowanych na SOR z rozpoznaniem AMI. Ci krytycznie chorzy pacjenci charakteryzują się wyjątkowo złym rokowaniem. Dlatego też w nowych wytycznych Europejskiego Towarzystwa Kardiologicznego dotyczących leczenia niewydolności serca ogromny nacisk położono na wczesne rozpoznanie i szybkie wdrożenie odpowiedniej terapii u chorych z AHF. Sugerowane jest nawet postępowanie zbliżone do strategii „złotej godziny" w zawale serca z uniesieniem odcinka ST. W niniejszej pracy wykazano, że jest to bardzo istotny problem w polskiej populacji, dlatego wszelkie działania mające na celu poprawę jakości opieki nad tymi chorymi powinny być wytrwale podejmowane.

Słowa kluczowe: dekompensacja układu sercowo-naczyniowego, intensywna terapia kardiologiczna, ostra niewydolność serca, niewydolność serca, zawał serca

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