Original Article

Prognostic Importance of Acute Heart Failure Persistence in Patients with ST-elevation Myocardial Infarction

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Abstract

Introduction: Acute heart failure (AHF) is one of the most frequent complication of acute myocardial infarction (AMI). It is not only associated with a several-fold increase of in-hospital mortality but also, worsens the long-term survival in comparison to those without AHF. The AHF is observed to be more in AMI patients whose in-hospital stay is more than 3 days. The clinical implications and prognostic accuracy of the AHF term in the setting of AMI are yet unknown. **Methods:** We observed 1,104 consecutive cardiac care patients, who were admitted with ST-elevation AMI (STEMI). They were divided into groups according to the AHF presence {AHF(+) *n*=334 and AHF(-) *n*=764}. Among 334 AHF(+) patients: 252 patients were found to have a transient AHFt(+), whereas 82 of AHF(+) patients had persistent AHFp(+) during in-hospital period. Patients' baseline characteristics, blood analysis, left ventricle (LV) and renal function data were assessed and analyzed on the admission day and 10th day post-admission. The follow-up was conducted on the 30th day and after 2 years. **Results:** STEMI patients accompanied by AHF(+) were older, presented mostly with anterior AMI (p<0.01), had lower LV ejection fraction (EF) (p<0.01) and a higher heart rate (p<0.05). Their rates of comorbidities and of in-hospital complications such as recurrent angina, reinfarction, LV aneurism were higher in comparision to AHF(-) patients. AHFp(+) patients had the shortest time from symptoms onset before thrombolysis in comparision to AHFt(+) and AHF(-) groups. Partial recovery of cardiac function according to Left ventricular ejection fraction (LVEF) and end-systolic volume index, occurred mainly in AHF(-) and AHFt(+) patients on the 10th day post-admission, but not in AHFp(+). STEMI patients with AHFp(+) demonstrated a larger infarct size, higher C-reactive protein and VGEF level, fasting glucose and heart rate on admission, higher erythrocyte sedimentation rate, absence of heart rate normalization on the 10th day post-admission. All of these markers were the signs of severe myocardial damage and inflammation, which can reflect worse recovery in AHF patients despite optimal management. Patients with AHF(+) had renal dysfunction on admission while its creatinine clearance (CrCl) decreased during the in-hospital period which is the reflection of a poor prognosis. Cardiovascular mortality and non-fatal MI were significantly higher in the AHFp(+) group as compared to the AHFt(+) and the AHF(–) groups during the 30 days and 2 years of follow-up. **Conclusion:** The AHF is a frequent STEMI complication. AHF lasting >3 days had worse short- and long-term prognosis. Therefore, an aggressive strategy should be recommended particularly in patients who have clinical signs and symptoms of persistent AHF.

Keywords: Myocardial infarction, Acute heart failure, Infarct size, Survival.

Introduction

Over the last few decades, a significant progress has been achieved in the treatment of patients with acute myocardial infarction (AMI), primarily due to the use of pharmacological or mechanical reperfusion strategies; second, due to medical therapy optimization.[1,2] Advances in the treatment have led to survival rate improvement in AMI patients as a result of an infarct-related artery opening that minimize the size of the myocardial damage. As the results show, despite

of progress in management more than one third the AMI patients have complication - acute heart failure (AHF). It is clear that natural history of patients with coexisting AMI and AHF leads to poor survival.[3]

Heart cell damage is the main factor of AHF occurrence, caused by ischemia/reperfusion injury, which often leads to deterioration or even loss of heart function, limits the benefits of reperfusion after AMI and has negative impact on global outcome.[4,5]

Large number of predictive models and risk scores exist to stratify the management of AHF patients. However, there are only few studies which have assessed the utility of AHF duration in ST-elevation myocardial infarction (STEMI) patients.[6] The clinical implications and prognostic accuracy of AHF terms in the setting of AMI yet unknown.

Therefore, we aimed to assess the prevalence and prognostic implications of transient versus persistent AHF among patients hospitalized with acute STEMI and to evaluate the short- and long-term outcomes in patients treated according to contemporary recommendations.

Methods

Study population

Patients were admitted to the Emergency Cardiology Department of the National Scientific Center "The M.D. Strazhesko Institute of Cardiology," Kiev, Ukraine.

Patients have been enrolled in our study according to the following criteria of inclusion:

- Age ≥21 years;
- Admission to the hospital due to STEMI. Diagnosis of AMI was based on serum markers level more than twice the upper limit of normal for MB fraction of creatine kinase and chest pain lasting for at least 20 mins and electrocardiogram (ECG) changes on at least 2 contiguous leads with significant ST-elevation;
- <24 h from symptoms onset of AMI;
- Written informed consent to participate in the study.

The exclusion criteria were as follows:

- Cardiogenic shock (Killip IV);
- Hepatic insufficiency (AST, ALT >3 times the ULN);
- Severe renal insufficiency (serum creatinine ≥200 μmol/l);
- Coagulopathy;
- Active cancer.

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 with measuring of blood pressure, heart rate, body mass index, and followed by standard diagnostic tests (ECG, biomarkers). Furthermore, we analyzed complications during in-hospital period, infarct size and laboratory data on the 1st and on the 10th days.

2-D echocardiography was performed on days 1, 3, 7 and 10 of in-hospital stay. End-diastolic volume (EDV) and end-systolic volume (ESV), and left ventricle ejection fraction (LVEF) were assessed by Simpson method. EDV index (EDVI) and ESV index (ESVI) were calculated as a ratio of EDV and ESV to body surface area.

Renal function was estimated using creatinine level and its clearance (CrCl) on admission and on the 10th day using the Cockcroft–Gault formula.

Endpoints and follow-up

Short- and long-term prognosis was analyzed according to the following events: Cardiovascular (CV) deaths and non-fatal MI. Follow-up was conducted at 30 days and after 2 years. Information regarding adverse events was obtained from patients or their relatives during phone calls.

Statistical methods

Statistical analysis was assessed with "SPSS 11.0" software using Student's *t*-test, χ^2-test, Wilcoxon non-parametric test, and Mann-Whitney test. Data were reported as means with M±m, the categorical variables were expressed as numbers with percentages. Survival rate was compared between groups using Kaplan–Meier plots and log-rank test. A value of $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics

A total of 1104 patients (54.1 ± 0.4 years) were hospitalized due to STEMI between 2000 and 2010. AHF was diagnosed on the criteria - signs and symptoms of AHF Killip class II/III (pulmonary congestion presented during physical examination and/or confirmed on chest X-ray).

STEMI patients were divided into two groups: Patients with AHF symptoms on the 1st day were classified as the AHF(+) group (n = 340) and those without heart failure - as the AHF(–) group (n = 764).

A total of 334 AHF(+) patients were randomized into two groups according to the AHF duration: Transient AHFt(+) - with AHF occurred on the 1st day and up to 3 days (n = 252) and persistent AHFp(+) - with AHF symptoms >3 days (n = 82) of in-hospital period. Six patients were excluded from the study due to cardiogenic shock during in-hospital stay.

The mean time from the onset of AMI symptoms was 4.8 ± 0.4 h and did not differ between groups.

Patients with AHFp(+) were older ($P < 0.05$), presented mostly with anterior AMI ($P < 0.01$), lower LVEF ($P < 0.01$) and higher heart rate ($P < 0.05$). They had significantly higher comorbidity rate such as previous history of angina pectoris ($P < 0.05$), hypertension ($P < 0.05$), chronic HF ($P < 0.01$), and diabetes ($P < 0.01$) compared to patients with AHF(-) and AHFt(+).

The shortest time from symptoms onset before thrombolysis ($P < 0.05$) was in the AHFp(+) group. The baseline characteristics of the patients are summarized in Table 1.

The patients received standard therapy according to the guidelines recommendations [Table 2].

It is generally accepted that the 3rd day following AMI is a critical period because of LV early remodeling and systolic function impairment due to reperfusion injury, myocardial stunning etc. Results of LV function showed significantly higher ESVI and lowered LVEF in the AHFp(+) group in comparison with those in AHFt(+) and AHF(-) patients on the 1st and 3rd day of in-hospital stay [Figure 1].

In our study, partial recovery of LV shape and contractile function occurred on the 7th and 10th days by EF increase and lower ESVI in AHF(-) and AHFt(+) patients in contrast to those in the AHFp(+) group.

Inflammation is a key pathogenic factor that plays an important role in the development of AHF. The analysis of the inflammation markers in peripheral blood during in-hospital period showed significantly higher CRP level on admission in AHFp(+) patients [Figure 2].

The severity and persistence of inflammation in patients with AHFp(+) was confirmed additionally by the increased ESR level on the 10th day [Table 3].

Results of the studies involving AMI in experimental models have shown that VEGF promotes angiogenesis in infarct zone and reduces MI area. Marked increase in VEGF indicates a protective effect on patients as a result of angiogenesis and endothelial cell proliferation. According to its biological effects VEGF may improve the long-term prognosis of patients with AMI.[7] In the present study, VEGF level increased twice on the 10th day in AHF(-) and AHFt(+) patients. In the AHFp(+) group VEGF was high on admission but has been not risen up to the 10th day of in-hospital period. To our opinion, this could be a marker of greater myocardial damage, inflammation, and worse survival.

Table 1: Baseline characteristics of the studied patients - comparison of AMI patients groups with AHFt(+) and AHFp(+) and group of patients with AMI without AHF – AHF(-)

Variable	AHF(-)	AHFt(+)	AHFp(+)
Patients, n	764	252	82
Male sex, %	84.6	91.7	89.0
Age, years, M ± m	53.1 ± 0.4*	55.3 ± 0.6	59.3 ± 1.0
Smokers, %	55.4	58.3	50.0
BMI, kg/m^2, M ± m	27.0 ± 0.2	27.2 ± 0.3	27.9 ± 0.4
BMI ≥ 30 kg/m^2, %	22.1	20.2	25.6
Previous history of (%)			
Stable angina, %	36.8*	46.4	51.2
Unstable angina, %	38.5	41.7	41.5
MI, %	14.4	20.2	18.3
Chronic HF, %	16.1**	15.5**	31.7
Hypertension, %	45.4*	49.6*	60.9
Diabetes, %	7.8**	6.0**	18.3
Time from onset of AMI before admission, hours	4.8 ± 0.2	5.1 ± 0.5	4.3 ± 0.4
Time from onset of AMI before thrombolysis, hours	3.9 ± 0.4*	3.5 ± 0.2*	2.9 ± 0.2
Anterior AMI, %	49.4**	62.7	74.4
LVEF, %	44.6 ± 0.4**	42.8 ± 0.6**	39.1 ± 1.0
HR, bpm	72.9 ± 0.6*	75.0 ± 1.3*	81.8 ± 2.6
SBP, mm Hg	123.2 ± 1.6	121.1 ± 2.4	129.3 ± 2.7
DBP, mm Hg	79.3 ± 1.0	76.8 ± 1.9	81.4 ± 1.5

*Significant in compare with AHFp(+) group $P < 0.05$, **Significant in compare with AHFp(+) group $P < 0.01$, BMI: Body mass index, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, AMI: Acute myocardial infarction, AHF: Acute heart failure, LVEF: Left ventricle ejection fraction

Table 2: Treatment of patients with AHFt(+), AHFp(+) and group of patients with AMI without AHF– AHF(-) during in-hospital course

Type of treatment/ medication	AHF(-)	AHFt(+)	AHFp(+)
Thrombolysis, %	45.1*	54.4	51.3
PCI, %	16.9	15.8	13.4
UF Heparin, %	66.5	69.0	65.8
LMW Heparin, %	35.2	44.8	36.6
Acetylsalicylic acid, %	80.4	80.1	75.6
Thienopyridines, %	17.8	16.3	15.9
Beta-blockers, %	96.2	96.0	97.6
ACEI, %	60.2**	77.0	82.9
ARB, %	8.1*	12.7	11.1
Diuretics, i.v., %	11.1**	24.6**	67.1
Aldosteroneblockers,%	5.5**	14.7*	25.6

*Significant in compare with AHFp(+) group $P < 0.05$, **significant in compare with AHFp(+) group $P < 0.001$, ACEI: Angiotensin enzyme converting inhibitor, ARB: Angiotensin receptors blocker, LMW Heparin - low molecular weight heparin, PCI: Percutaneous intervention, AMI: Acute myocardial infarction, AHF: Acute heart failure

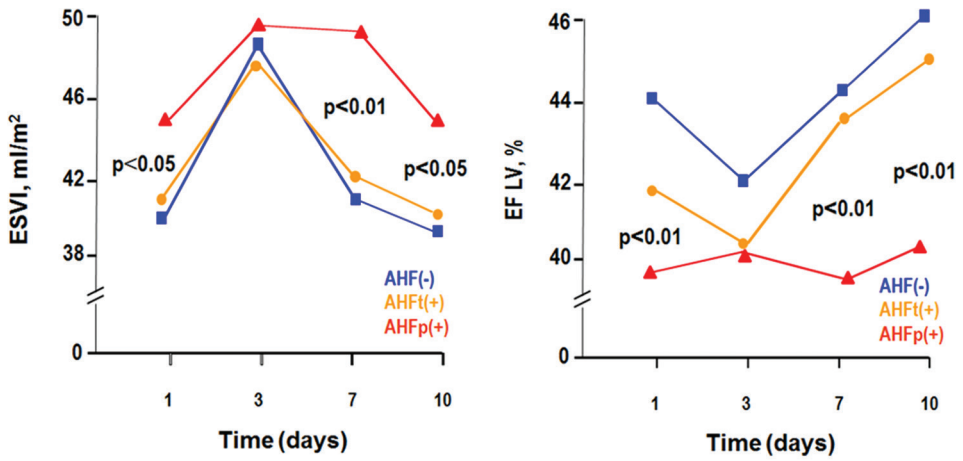

Figure 1: Changes of end-systolic volume index and left ventricle ejection fraction in acute myocardial infarction patients

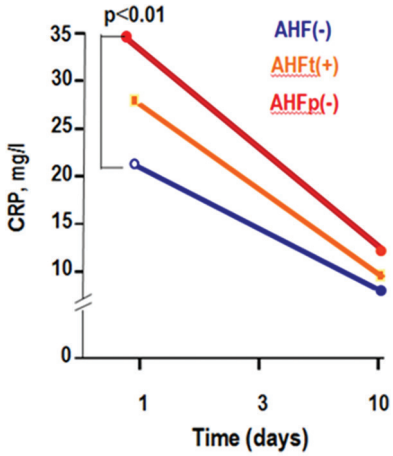

Figure 2: C-reactive protein levels in acute myocardial infarction patients

AHFt(+) and AHFp(+) patients have significantly greater infarct size according to the levels of serial MB-CK in compare to those in AHF(-) patients [Figure 3].

Both AHFt(+) and AHFp(+) patients demonstrated increased glucose level on admission [Figure 4].

In AMI normal hormonal control of blood glucose concentration is disturbed by the stress. Irrespective of diabetes status is quite common for blood glucose to be raised in the immediate period following AMI.[8]

Several reports have evaluated an association between hyperglycemia and mortality following MI.[8]

Renal impairment on admission in patients presented with AHF is common sign, occurred approximately in half of the patients and associated with high mortality.[9]

In our study CrCl rate was significantly lower on admission in patients with AHFp(+) in compare to both AHF(-) ($P < 0.05$) and AHFt(+) patients ($P < 0.05$). On the 10th day, CrCl level was decreased in all groups of patients, but significantly lower it was only in AHFp(+) group [Figure 5].

Hospital and post-discharge outcomes

Patients from both AHFt(+) and AHFp(+) groups had higher in-hospital complications rate than patients without AHF(-). Detailed analysis is presented in Table 4.

During the following 30 days AHFp(+) patients had higher incidence rate of CV death and non-fatal MI in compare with those in AHF(-) (7.3 % vs 1.2%, P < 0.01 and 12.2% vs 5.8%, P < 0.01) and AHFt(+) (7.3% vs 2.1%, P < 0.05 and 12.2 vs 3.2%, P < 0.01) groups.

CV mortality and non-fatal MI were significantly higher in the AHFp(+) group as compared to AHFt(+) and AHF(–) group during long-term follow-up [Figure 6].

DISCUSSION

AHF is a complex clinical syndrome characterized by the rapid symptom occurrence due to LV contractile function failure: Inadequate tissue perfusion, high lung capillary pressure, and tissue congestion. Patients with AMI complicated by AHF have worse short- and long-term survival rate.[10,11]

According to the US National Registry of Myocardial Infarction data AHF occurrence in AMI patients was more than 20% on admission and additionally in 9% during in-hospital stay.[12] In the French study, AHF was presented in 38% patients during 5 days after AMI onset. AHF symptoms developed mostly within the first 24 h of AMI with next worsening on the 4th day.[13] In our study, signs and symptoms of AHF in AMI patients were

Table 3: Comparison of biochemical parameters in patients with AHFt(+), AHFp(+) and group of patients with AMI without AHF – AHF(-)

Variable	AHF(-)	AHFt(+)	AHFp(+)
Hemoglobin, g/l (on 1st day)	138.3 ± 0.5	139.2 ± 0.8	137.7 ± 1.5
Hemoglobin, g/l (on 10th day)	131.6 ± 0.7	131.5 ± 1.0	128.9 ± 1.9
Platelets, ×10^9/l (on 1st day)	233.5 ± 7.0	230.8 ± 9.7	238.0 ± 17.0
Platelets, ×10^9/l (on 10th day)	255.0 ± 7.4	247.4 ± 9.3	224.5 ± 12.5
VGEF, pg/ml (on 1st day)	95.3 ± 17.8**	123.3 ± 37.6*	391.2 ± 147.5
VGEF, pg/ml (on 10th day)	209.6 ± 28.8	223.4 ± 44.9	428.1 ± 99.0
ESR, mm/h (on 1st day)	8.5 ± 0.4	8.5 ± 0.5	9.0 ± 0.9
ESR, mm/h (on 10th day)	16.9 ± 0.8	17.2 ± 1.1*	22.0 ± 1.9
WBC, ×10^9/l (on 1st day)	9.2 ± 0.1	9.4 ± 0.2	9.8 ± 0.4
WBC, ×10^9/l (on 10th day)	8.5 ± 0.2	7.8 ± 0.2	8.4 ± 0.3
CRP, mg/l, (on 1st day)	22.1 ± 1.4**	26.9 ± 2.3	35.6 ± 3.9
CRP, mg/l, (on 10th day)	10.7 ± 0.8	11.1 ± 1.0	13.3 ± 2.5

*Significant in compare with AHFp(+) group $P < 0.05$, **significant in compare with AHFp(+) group $P < 0.01$, CRP: C-reactive protein, WBC: White blood cells, ESR: Erythrocyte sedimentation rate, VGEF: Vascular endothelial growth factor, AMI: Acute myocardial infarction, AHF: Acute heart failure

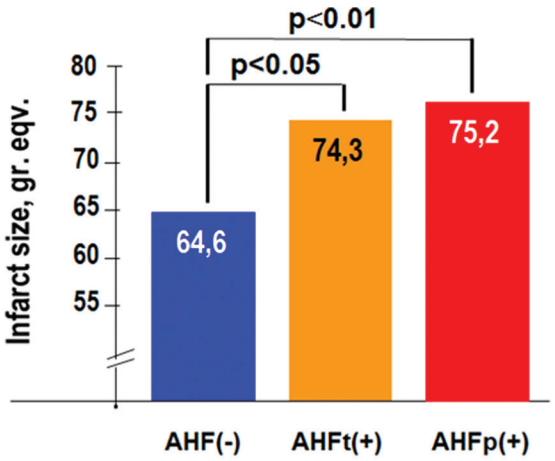

Figure 3: Infarct size by serial measurement of MB-CK in acute myocardial infarction patients Both AHFt(+) and AHFp(+) patients demonstrated increased glucose level on admission [Figure 4]

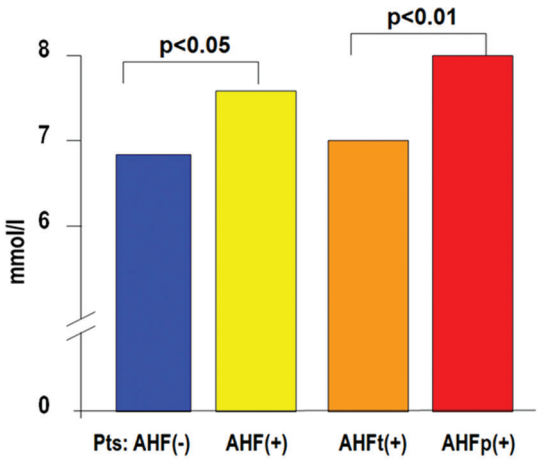

Figure 4: Levels of glucose on admission in acute myocardial infarction patients

presented in 30.8% on the 1st day and in 9% - occurred during in-hospital stay.

AHF in AMI patients has been associated with an increase of in-hospital mortality from 6% (with preserved EF) up to 80% (with cardiogenic shock) during the 1st year following AMI – it can reach 30%. In agreement with modern concepts, systolic LV dysfunction is a predictive factor of adverse outcomes.[14]

Reperfusion therapy is very important because both systemic TLT and percutaneous coronary interventions limit myocardial damage. Infarct size is the key determinant for stratification patients after MI. In our study, myocardial revascularization was performed in 64% patients, and infarct size was significantly higher in ST-elevation AMI patients with AHF.[15]

AHF as an AMI complication is a result of complex interaction of structural, hemodynamic, neurohumoral and genetic changes. Sudden myocytes loss leads to contractile dysfunction resulting in AHF manifestation where the level of myocardial damage biomarkers closely correlates with the degree of LV function recovery and prognosis.[16]

Myocardial infarction size and postischemic LV systolic dysfunction that lead to AHF, can be the result of myocardial necrosis, stunning and/or hibernation, which in turn depend on coronary perfusion.[17] In our study, AHFt(+) and AHFp(+) patients had significantly greater infarct size according to the levels of serial MB-CK in compare to those in AHF(-) patients.

Table 4: Comparison of in-hospital complications in patients with AHFt(+), AHFp(+) and group of patients with AMI without AHF – AHF(-)

Variable	AHF(-)	AHFt(+)	AHFp(+)
Recurrent angina, %	5.9*	8.3	14.6
Reinfarction, %	5.8*	3.2**	12.2
LV aneurism, %	8.5***	15.9**	25.7
Ventricular fibrillation/tachycardia, %	4.8	4.2	4.8
A-V block, %	3.1*	3.2	8.5
Bundle branch block, %	-	5.2**	19.5
Atrial fibrillation, %	2.2**	2.0**	19.5
Cardiogenic shock, %	-	0.6*	4.9

*Significant in compare with AHFp(+) group $P < 0.05$, **significant in compare with AHFp(+) group $P < 0.01$, ***significant in compare with AHFp(+) group $P < 0.001$, AMI: Acute myocardial infarction, AHF: Acute heart failure

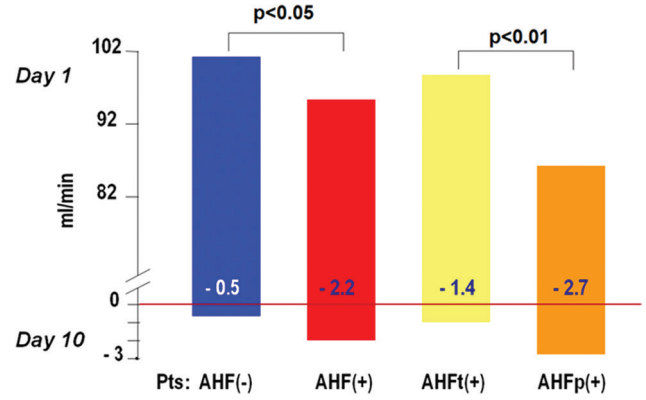

Figure 5: Changes of CrCl on the 1st and 10th days in acute myocardial infarction patients

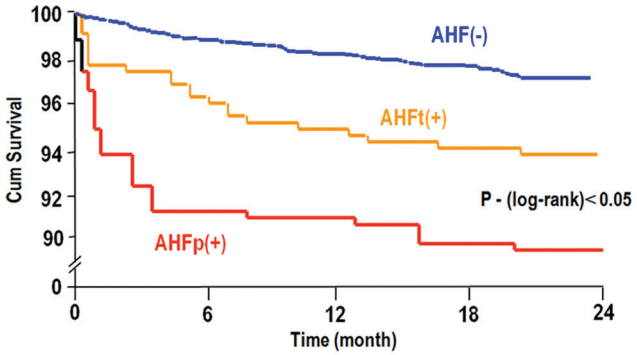

Figure 6: Kaplan-Meier survival analysis in acute myocardial infarction patients

Heart remodeling includes genes expression, molecular, cellular and interstitial alterations that are characterized by changes of ventricular size, shape and function after myocardial injury. Pathological LV remodeling occurs in AMI patients with greater regional contractility disorders that are also mediated by coronary perfusion. Abnormal myocardium relaxation due to ischemia cause impairment of LV filling and its global systolic function and as a consequence – lead to AHF. In addition, ischemia may be the cause acute mitral regurgitation, and thus to promote pulmonary congestion.[18]

The remodeling process mainly depends on the hemodynamic load, neurohormonal and inflammation activation. It has been proven that the process of LV remodeling begins during the 1st hours of AMI (expansion of the infarct zone) and may continue for a long time (time-dependent dilatation, the distortion of ventricular shape), which results in EDVI and ESVI increasing with LVEF decline.[19] Our findings showed features of pathological remodeling in AHFp(+) by the higher meanings of ESVI and lower LVEF on the 1st and 3rd day of AMI in compare with those in AHFt(+) and AHF(-) patients.

Inflammation markers could reflect the myocardium healing. In our study high CRP level on admission was in STEMI patients with AHF(+), especially in AHFp(+) patients (P<0.01) comparatively to AHF(-) group. Results of clinical trials underline that high CRP level has been associated with higher mortality rate in patients with AMI.[20]

Patients with AMI have elevated circulating VEGF levels in comparison with healthy subjects. VEGF enhances vascular permeability, accelerates collateral formation in ischemic myocardium and promotes tissue repair after wound healing.[7]

In our study, VEGF level increased twice on the 10th day in AHF(-) and AHFt(+) patients, whereas in the AHFp(+) group VEGF was high on admission, but did not increase up to the 10th day of in-hospital period. To our opinion, this could be a marker of greater myocardial damage, inflammation and worse survival.

Despite of a strong evidence linking decreased glomerular filtration rate to worse outcomes, the impact of CrCl on mortality and morbidity in patients with AMI and AHF is not well defined.[21] According to our results, CrCl level was associated with severity and duration of AHF symptoms that connected with higher rate of complications and poor survival.

Therefore, according to guidelines LV systolic function should be routinely assessed by echocardiography in all AMI patients on the 10th day for individualized management depending on the LV EF. In our opinion, it is necessary to use more aggressive therapy to improve the prognosis of AMI patients with AHF. This category includes elderly patients, those with diabetes mellitus, history of coronary artery disease, anterior MI, and newly emerged bundle branch blocks.

Optimal medical therapy in patients with AHF based on guidelines should include beta-blockers, ACE inhibitors or angiotensin II receptor blockers and aldosterone receptor antagonist.[22]

However, according to real clinical practice, patients with AHF are less likely to receive aspirin, heparin, beta-blockers and reperfusion therapy.[12]

In our study we demonstrated dependence on the outcomes on the time of onset of AHF and its duration. AMI patients with AHFp(+) during in-hospital stay presented more frequent complications such as recurrent MI and angina, LV aneurysm, and ventricular arrhythmias.

AHFp(+) patients had also renal dysfunction (RD) on admission. RD is an independent risk factor of adverse outcomes both in AMI and AHF patients despite of age, gender and glucose intolerance degree.[9] At the same time, RD leads to fluid retention, activation of the renin-angiotensin system, increasing of proinflammatory cytokines and endothelial dysfunction. Reduced glomerular filtration rate (<60 ml/min/1.73 m^2) is not associated with the level of cardiac markers, infarct size, or ST segment elevation but is strongly related to CV history and initial LV function.[23]

In our study, AHF(+) was associated with increase of 30 days and 2 years mortality rates in compare with those observed in patients without AHF(-). Particularly, AMI patients with persistent AHF(+) had significantly higher complications rate during in-hospital period and long-term outcome.

Study limitations

Certain limitations must be considered during interpreting our study. First, this study represents patients with AHF in the presence of AMI only from one center. Second, in presented study, AMI patients had low percent of PCI performing. Further studies are required to confirm our findings on larger populations of patients with AHF terms in the setting of AMI. Incidence of high mortality rate in patients with AHF requires both more aggressive treatment on admission and effective AHF prevention.

Conclusion

Our study shows that AHF is a frequent complication of ST-segment elevation AMI. The AHF incidence in the first 24 h after admission and during in-hospital stay is similar to data reported in previous studies.

AHF on admission and up to 3 days has not important impact on MACE incidence in AMI patients.

STEMI patients with AHF lasting >3 days have worse short- and long-term prognosis. Therefore, an aggressive strategy should be recommended, particularly in patients who have clinical signs and symptoms of persistent AHF.

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