Armağan Kaya[1], Mustafa Gökçe[2]

[1] Trabzon Kanuni Training and Research Hospital, Cardiology Clinic, Trabzon, Turkey

[2] Karadeniz Technical University Faculty of Medicine, Department of Cardiology, Trabzon, Turkey

The Relationship between Phenotypic Classification and Inflammatory Parameters in Patients Hospitalized with Acute Heart Failure

Aim	To evaluate the effects of inflammatory parameters on mortality and prognosis in patients who were hospitalized with acute heart failure (AHF) and phenotypically classified.
Material and methods	Between December 2020 and August 2021, 240 patients, who were newly diagnosed with acute heart failure (AHF) or those with heart failure and who developed decompensation, were prospectively included in the study. The patients composed four equal groups of 60 patients each according to the phenotypical class of AHF: warm-wet, warm-dry, cold-wet, and cold-dry. Acute phase reactants, namely C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and plasma albumin, were examined at hospitalization, discharge, and 30±7 days after discharge. The reactants were compared between the groups in terms of mortality and prognosis.
Results	Univariate analyses showed that, at the time of initial hospitalization, a one-unit increase in albumin decreased the mortality risk 0.794-fold, while a one-unit increase in CRP increased the mortality risk 1.013-fold and a one-unit increase in ESR increased the mortality risk 1.026-fold ($p<0.001$, $p=0.003$, and $p=0.002$, respectively). At discharge, a one-unit increase in albumin decreased the mortality risk 0.85-fold ($p=0.043$). However, multivariate analyses showed that, at the time of initial hospitalization, a one-unit increase in albumin decreased the mortality risk 0.803-fold, while a one-unit increase in the ESR value increased the mortality risk 1.021-fold ($p<0.001$ and $p=0.049$, respectively). Although a statistically significant difference was observed between the warm-dry group and the other groups in terms of in-hospital mortality distributions ($p=0.032$), there was no statistically significant difference between the groups in terms of out-of-hospital mortality ($p>0.050$).
Conclusion	In AHF patients, low albumin values at initial hospitalization and discharge, high CRP and ESR values at initial hospitalization predict increased mortality.

Keywords	Acute heart failure; phenotypical classification; acute phase reactants
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Corresponding author	Armağan Kaya. E-mail: daleesamsun@gmail.com

Introduction

Heart failure (HF) is a clinical syndrome characterized by the emergence of acute or chronic symptoms and signs that result from the inability of the ventricles to adequately pump blood due to structural and/or functional heart diseases [1]. Acute heart failure (AHF) is a clinical condition characterized by dyspnea associated with rapid accumulation of fluid in the interstitial and alveolar spaces of the lung. AHF may be the result of acute decompensation of chronic HF, or it may be the result of acute cardiac damage, i.e., de novo AHF. Patients with acute decompensated heart failure (ADHF) are categorized according to the presence of congestion ("wet") or absence of congestion ("dry") and the presence of adequate peripheral perfusion ("warm") or hypoperfusion ("cold"). This categorization can help guide treatment during the initial phase and provides valuable prognostic information [2].

C-reactive protein (CRP) is an acute phase reactant secreted in acute inflammation and infection states. Many cardiovascular studies have investigated the role of CRP in cardiovascular diseases [3, 4]. Most studies have found that the evaluation of CRP together with pro-brain natriuretic peptide provides better prognostic information [5, 6]. However, there is still no cut-off value for CRP in predicting the occurrence of adverse effects in HF. Although CRP measurements are suitable for clinical use, their utilization for prognosis and screening in HF is not yet recommended in the relevant guidelines [7].

Albumin is the most abundant plasma protein in humans. It has many functions, including the regulation of plasma oncotic pressure, binding and transportation of many

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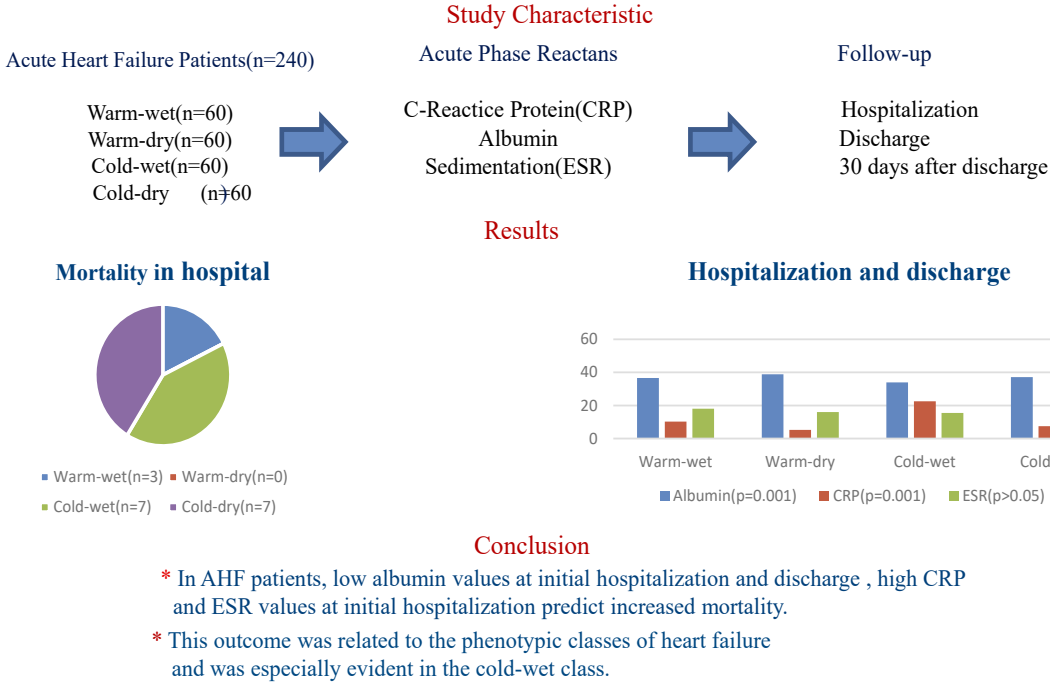
Central illustration. The Relationship between Phenotypic Classification and Inflammatory Parameters in Patients Hospitalized with Acute Heart Failure



molecules in the blood, clearance of free radicals, inhibition of platelet functions, and regulation of capillary membrane permeability [8]. Regardless of the underlying disease, there is an inverse correlation between serum albumin concentrations and mortality. Hypoalbuminemia has been found to be associated with increased mortality and morbidity and a prolonged hospital stay [8]. Many mechanisms have been proposed to explain the relationship between low albumin and increased cardiovascular mortality. For example, it has been reported that albumin is strongly associated with infection and inflammation [9, 10], nutritional status [11], platelet aggregation [10], hemostasis and fibrinolysis factors [10], and increased vascular permeability due to underlying diseases [9], as well as acting as an antioxidant [12].

The erythrocyte sedimentation rate (ESR) is a simple and inexpensive test used to detect inflammatory activity. An increased ESR is used to determine the presence of a disease in clinic settings, monitor the course of a known disease, and evaluate the patient's response to treatment [13, 14]. In this study, we aimed to investigate the relationship between inflammatory parameters and prognosis and mortality among patients who were hospitalized with AHF and phenotypically classified, a topic that has not been previously addressed in the literature.

Materials and methods

Study population

Between December 2020 and August 2021, 240 patients with a new diagnosis of AHF or HF with decompensation were prospectively enrolled. Phenotypical classification was performed by two cardiologists according to the physical examination findings. Congestion findings included pulmonary edema, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, neck venous engorgement, hepatomegaly, ascites, and hepatojugular reflux. The signs of hypoperfusion were cold and sweaty extremities, oliguria, mental confusion, narrowed pulse pressure, and increased lactate concentrations. The patients comprised four groups according to the phenotypical class of AHF: warm-wet, warm-dry, cold-wet, and cold-dry [15]. Acute phase reactants, i.e., inflammation markers, namely CRP, ESR, and albumin, were measured in these patient groups at the time of hospitalization, at discharge, and at 30±7 days after discharge. The values of these parameters were compared between the groups in terms of mortality and prognosis.

Patients with cancer, active infection, acute coronary syndrome, and acute renal failure were excluded from the study. Consent for the study was obtained from the ethics committee of the Karadeniz Technical University Faculty of Medicine (approval number 24237859–685). Patients were included in the study in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

Data were analyzed with the SPSS version 21 for Windows (SPSS Inc, Chicago, IL, USA). program. The Kolmogorov-Smirnov test was used to understand whether the data had a normal distribution.test. A chi-square

Table 1. Demographic and clinical characteristics of the patients grouped by phenotypic class

Variable	Total, n=240	Warm-wet, n=60	Warm-dry, n=60	Cold-wet, n=60	Cold-dry, n=60	p
Male	131 (54.6)	26 (43.3)[a]	29 (48.3)[a]	36 (60)[ab]	40 (66.7)[b]	0.041
Female	109 (45.4)	34 (56.7)	31 (51.7)	24 (40)	20 (33.3)	
Hypertension	195 (81.3)	53 (88.3)	49 (81.7)	48 (80)	45 (75)	0.310
Diabetes	111 (46.3)	33 (55)[a]	19 (31.7)[b]	26 (43.3)[ab]	33 (55)[a]	0.029
CAD	142 (59.2)	42 (70)[a]	34(56.7)[ab]	39 (65)[a]	27 (45)[b]	0.031
PAD	4 (1.7)	1 (1.7)	0 (0)	1 (1.7)	2 (3.3)	0.565
Chronic heart Failure		44 (73.3)[ab]	23 (38.3)[a]	52(86.7)[b]	39 (65)[a]	<0.001
AF	110 (45.8)	36 (60)[a]	17 (28.3)[b]	32 (53.3)[a]	25(41.7)[ab]	0.003
CVD	26 (10.8)	7 (11.7)	3 (5)	9 (15)	7 (11.7)	0.351
CKD	55 (22.9)	14 (23.3)	12 (20)	11 (18.3)	18 (30)	0.438
COPD	46 (19.2)	12 (20)	10 (16.7)	14 (23.3)	10 (16.7)	0.757
ACE-i/ARB	106 (44.2)	21 (35)	32 (53.3)	25 (41.7)	28 (46.7)	0.222
Beta blockers	157 (65.4)	45 (75)	32 (53.3)	41 (68.3)	39 (65)	0.088
MRA	58 (24.2)	20 (33.3)[a]	3 (5)[b]	19 (31.7)[a]	16 (26.7)[a]	0.001
Diuretics	177 (73.8)	49 (81.7)[ab]	37 (61.7)[b]	51 (85)[a]	40(66.7)[ab]	0.008
Digoxin	17 (7.1)	6 (10)	3 (5)	6 (10)	2 (3.3)	0.358
OAC/NOAC	96 (40)	33 (55)[a]	15 (25)[b]	27 (45)[ab]	21 (35)[ab]	0.006
ASA	89 (37.1)	19 (31.7)	28 (46.7)	24 (40)	18 (30)	0.201
Clopidogrel	33 (13.8)	7 (11.7)	9 (15)	12 (20)	5 (8.3)	0.289
Statin	74 (30.8)	23 (38.3)	16 (26.7)	18 (30)	17 (28.3)	0.519
O2 required	114 (47.5)	27 (45)[a]	5 (8.3)[b]	51 (85)[c]	31 (51.7)[a]	0.001
iv diuretic required	122 (50.8)	60 (100)[a]	2 (3.3)[b]	60 (100)[a]	0 (0)[b]	0.001
iv nitrate required	56 (23.3)	12 (20)[a]	44 (73.3)[b]	0 (0)[c]	0 (0)[c]	0.001
Positive inotrope or vasodepressor	124 (51.7)	1 (1.7)[a]	5 (8.3)[a]	58 (96.7)[b]	60 (100)[b]	0.001

Data are number (percentage). p values from Chi-square test. [a-c] No significant difference between the groups with the same letter.
CAD, coronary artery disease; PAD, peripheral artery disease; AF, atrial fibrillation; CVD, cerebrovascular disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; ACE-i, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blocker; MRA, mineralocorticoid receptor antagonist; OAC, oral anticoagulant; NOAC, new oral anticoagulant; ASA, acetylsalicylic acid; iv, intravenous.

Table 2. Comparison of the groups in terms of mortality and rehospitalization

Variable	Total, n=240	Warm-wet, n=60	Warm-dry, n=60	Cold-wet, n=60	Cold-dry, n=60	p
Rehospitalization	24 (10)	7 (11.7)	5 (8.3)	8 (13.3)	4 (6.7)	0.604
Mortality	24 (10)	4 (6.7)[ab]	1 (1.7)[a]	9 (15)[b]	10 (16.7)[b]	0.019
In hospital mortality	17 (7.1)	3 (5)[ab]	0 (0)[b]	7 (11.7)[a]	7 (11.7)[a]	0.032
Out of hospital mortality	7 (2.9)	1 (1.7)	1 (1.7)	2 (3.3)	3 (5)	0.655

Data are number (percentage). p values are from Chi-square tests. [a-c] No significant difference between the groups with the same letter

test was used to compare categorical variables according to the phenotypical class. One-way analysis of variance was used to compare normally distributed data with groups of three or more, and multiple comparisons were performed with the Duncan test. The Kruskal-Wallis test was conducted to compare non-normally distributed data with groups of three or more. Repeated measures analysis of variance was employed for the comparison of normally distributed data from the same patient, and the Friedman test was utilized for the comparison of non-normally distributed data. Univariate and multivariate binary logistic regression analyses were performed to determine dependent and independent predictors of the mortality. The results of the analysis are presented as mean±standard deviation (SD) or median (minimum-maximum) values for quantitative data and as numbers (percentages) for categorical data. A p value <0.05 was considered statistically significant.

Results

The study included 240 patients hospitalized with the diagnosis of AHF. The demographic and clinical characteristics and comparisons of the four phenotypic groups are given in Table 1.

The warm-dry HF group differed significantly from the other phenotypical groups in terms of mortality distribution (p=0.019; Table 2). Mortality occurred in four patients (6.7%) in the warm-wet group, one patient (1.7%) in the warm-dry group, nine patients (15%) in the cold-wet

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Table 3. Comparison of acute phase reactant values between and within the groups

Variable	Total, n=240	Warm-wet, n=60	Warm-dry, n=60	Cold-wet, n=60	Cold-dry, n=60	p
Albumin hospitalization(g/l)	36.6±4.8	36.6±5.2^{bA}	38.9±4.3^{bA}	33.9±4.4^{aA}	37.1±4.0^{bA}	0.001
Albumin at discharge(g/l)	35.1±4.5	35.1±4.4^{abB}	36.5±4.4^{bB}	33.2±4.6^{aB}	35.5±3.9^{bB}	0.001
Albumin during follow-up(g/l)	37.9±4.7	37.6±4.8^{abA}	38.8±4.4^{bA}	36.4±4.3^{aA}	38.5±5.1^{abA}	0.038
p		<0.001	<0.001	<0.001	<0.001	
CRP at hospitalization(mg/l)	9.8 (0.6 -350.80)	10.25 (1.2-350.8)^{caB}	5.2 (0.6-102.2)^{a}	22.5 (1.4-166.7)^{bB}	7.55 (0.6-213.8)^{acAB}	0.001
CRP at discharge(mg/l)	10.9 (1.2-208.4)	11.4 (1.3-162.4)^{abB}	7.7 (1.2-80.2)^{a}	17.0 (2.1-208.4)^{bB}	11.6 (1.7-141.1)^{abB}	0.001
CRP during follow-up(mg/l)	5.7 (0.5-158.2)	5.9 (0.8-110.3)^{A}	4.35 (0.5-149.0)	7.9 (0.5-158.2)^{A}	4.9 (0.9-51.1)^{A}	0.177
p		0.017	0.183	<0.001	0.011	
ESR at Hospitalization(mm/h)	18.0 (2.0-100.0)	16.0 (4.0-87.0)^{A}	15.5 (3.0-81.0)^{B}	20.0 (3.0-100.0)^{B}	19.5 (2.0-90.0)	0.940
ESR at discharge(mm/h)	24.0 (3.0-121.0)	24.0 (3.0-121.0)^{B}	19.5 (3.0-84.0)^{AB}	26.5 (4.0-103.0)^{A}	24.0 (3.0-97.0)	0.594
ESR during follow-up(mm/h)	19.0 (3.0-112.0)	19.5 (3.0-112.0)^{AB}	21.0 (5.0-88.0)^{A}	19.0 (3.0-84.0)^{B}	18.0 (3.0-91.0)	0.611
p		0.040	0.001	0.003	0.564	

Data are mean±SD or median (minimum value-maximum value. $^{a-c}$ No significant difference between the groups with the same letter, $^{A-B}$ No significant difference between the groups with the same letter during hospitalization, discharge, and follow-up assessments.

group, and ten patients (16.7%) in the cold-dry group during the follow-up period. Upon examining mortality status in detail, determined despite the statistically significant difference between the warm-dry HF group and the other groups in terms of in-hospital mortality distribution (p=0.032), there was no significant difference between the groups in terms of out-of-hospital mortality (p>0.050). Furthermore, no significant difference was found between the groups in terms of rehospitalization (p=0.604).

Albumin measured at the time of initial hospitalization and discharge was significantly lower in the cold-wet HF group than in the other phenotypical classification groups (p<0.001). Albumin was significantly lower in the cold-wet group than in the warm-dry HF group (p=0.038). The mean value of albumin measured at discharge was significantly lower than the mean value of albumin measured at initial hospitalization and discharge in all groups (p<0.001) (Table 3). In these data, we concluded that hypoalbuminemia was particularly common in the cold-wet group and that this might be related to mortality. We predicted that the statistical difference in mortality between the groups was due to this difference in albumin. When we evaluated albumin within the groups, we found that the albumin value was low, especially at the discharge stage, and the statistical difference in mortality was due to this difference in albumin. We thought that this situation might have been caused by the psychological stress experienced during hospitalization period.

The median value of CRP at the initial hospitalization was significantly higher in the cold-wet HF group than in the other phenotypical classification groups, and the median value of CRP at discharge was significantly higher in the cold-wet HF group than in the warm-dry HF group (p<0.001). There were no significant differences between the groups in relation to the CRP values examined during the follow-up (p=0.177). The warm-wet HF group had a statistically higher CRP at discharge when compared to the follow-up evaluation (p=0.017). In the cold-wet HF group, the median CRP measured during the follow-up was significantly lower than the median CRP measured at initial hospitalization and discharge (p<0.001 for both). The cold-dry HF group had a significantly lower CRP during the follow-up when compared to the values measured at initial hospitalization and at discharge (p=0.011) (Table 3). In the comparison of CRP values, we found that the significant difference between the groups was due to the low value in the hot-dry group. In the CRP evaluation of the groups during the follow-up period, we found that there was no significant difference only in the hot-dry group. CRP was low in every period, and especially in the hot-dry group, suggesting that the inflammatory process of heart failure was milder in this group than in the other groups.

There were no significant differences between the phenotypical classification groups in terms of the ESR values measured at different times (p>0.050, Table 3). This lack of statistical difference in the ESR values between the groups suggested that ERS is not predictive of HF outcome. In the intragroup evaluation of ESR, there were significance differences among the values at different periods in each group. Further studies are needed to explain these differences.

The univariate analyses of acute phase reactants measured at the time of initial hospitalization showed that the risk of mortality was decreased 0.794-fold by a one-unit increase in albumin, was increased 1.013-fold by a one-unit increase in CRP, and was increased 1.026-fold by a one-unit increase in ESR (p<0.001, p=0.003, and p=0.002, respectively; Table 4). At discharge, a one-unit increase in albumin resulted in a 0.85-fold decrease in the mortality risk (p=0.043), while the differences were not significant for CRP and ESR (Table 4). Multivariate analyses showed that, at the time of initial hospitalization, a one-unit increase in albumin decreased the mortality risk 0.803-fold, while a one-unit increase in the ESR value increased the mortality risk 1.021-fold (p<0.001 and p=0.049, respectively, Table 4). The results were not significant for CRP. At discharge, the mortality risk was not significantly affected by changes in the albumin, CRP, and ESR values (p>0.050 for all; Table 4).

Discussion

The role of inflammation in HF has been previously demonstrated. Inflammation may cause myocardial damage, and mediators associated with inflammation may lead to the progression or worsening of HF [7]. The relationship between HF and inflammation was first investigated in 1955, and a positive correlation was found between the severity of HF and CRP [16]. In 1990, Levine et al. found that the severity of chronic HF increased as the tumor necrosis factor-alpha value increased [16].

Numerous studies have demonstrated a relationship between the prognosis of HF and inflammatory and anti-inflammatory mediators. In a retrospective cohort study of 546 patients, the risk of in-hospital mortality in patients admitted due to acute, non-ischemic HF was found to be nine times higher in those with an albumin value of <34 g/l compared to those with an albumin value above this cut-off point [17]. This group also investigated the effect of albumin on long term mortality in 509 patients with AHF and found that hypoalbuminemia was associated with increased mortality [18]. In a retrospective study of 730 patients, the relationship between hemoglobin, albumin, lymphocyte and platelet (HALP) index and short- and long-term mortality in patients with heart failure was examined. Higher HALP scores were found to be associated with a reduced risk of one-month mortality and one-year mortality in patients with heart failure [19]. In another retrospective study of 2077 patients, the relationship between red blood cell distribution width-to-albumin ratio (RAR) and prognosis in patients with non-ischemic heart failure was investigated. A high red blood cell distribution RAR was found to be associated with a significantly increased risk of all-cause death or heart transplantation in patients with non-ischemic heart failure [20].

In the current study, hypoalbuminemia was associated with increased mortality, thus supporting earlier studies. However, considering that hypoalbuminemia will occur in patients with heart failure due to comorbidities and cachexia in addition to the inflammatory process, we believe that it is more appropriate to evaluate albumin as a part of the heart failure score rather than as a sole marker of inflammation.

In a study of 242 patients, the effect of ESR of patients with congestive HF were investigated [21]. The results showed that the one-year mortality risk was significantly lower in patients with high ESR values than those with normal or low ESR values. Also, after therapy, the improvement in functional capacity was found to be significantly higher in patients with high ESR values. In another study of 159 patients examined the relationship between ESR and mortality in patients with chronic HF, the mortality rate was significantly higher in patients with an ESR value of ≥15 mm/h than in those with a lower ESR. Each 1-mm increase in ESR was found to be associated with a 2.9% higher mortality risk [22]. In addition to these studies, we showed an association between an increased ESR value and increased mortality, supporting our hypothesis that ESR values would increase in parallel with the increased inflammation process during HF.

A study conducted with 2,618 patients examined the relationship between the CRP values measured at admission and discharge and one-year mortality and rehospitalization rates in patients hospitalized with acute decompensated HF [23]. The authors determined that a high CRP value (>10 mg/l) at admission or discharge was

Table 4. The effect of acute phase reactants on mortality as analyzed by binary logistic regression

Variable	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	p	OR (95% CI)	p
Albumin hospitalization(g/l)	0.794 (0.719–0.876)	<0.001	0.803 (0.723–0.891)	<0.001
CRP hospitalization(mg/l)	1.013 (1.005–1.022)	0.003	1.005 (0.996–1.014)	0.244
ESR hospitalization	1.026 (1.009–1.042)	0.002	1.021 (1.000–1.043)	0.049
Albumin discharge(g/l)	0.85 (0.726–0.995)	0.043	0.849 (0.702–1.027)	0.091
CRP discharge(mg/l)	1.011 (0.994–1.028)	0.200	1.005 (0.983–1.027)	0.673
ESR discharge(mm/h)	1.003 (0.973–1.035)	0.835	0.997 (0.961–1.033)	0.852

OR, odds ratio; CI, confidence interval; CRP, C-reactive protein.

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associated with one-year mortality in patients with ADHF but not with rehospitalization. Another study of 305 patients with chronic HF investigated whether various laboratory biomarkers examined during followed up with were beneficial for the prognosis evaluation [24]. The results showed that CRP was not a good predictor of the long-term outcomes of HF. In the current study, we observed a significant association between increased CRP and increased mortality due to the inflammation process effective in HF.

Inflammatory parameters are included in scoring systems for the evaluation of various clinical conditions, such as albumin in the HALP score for predicting mortality in patients with ileus [25], CRP in the Glasgow prognostic score for assessing prognosis in colorectal cancer [26], and both ESR and CRP in the A. C. H. E. score for evaluating the risk of skeletal-related events in renal cell carcinoma [27]. Thus, we anticipate that including these parameters as components of new scoring systems designed to predict mortality and prognosis in HF may yield more accurate predictions.

In the current study, the albumin value was significantly lower and the CRP value was significantly higher in the cold-wet HF group compared to the other groups, both at the initial hospitalization and discharge. Whether this was a coincidence or the clinical result of the combination of hypoperfusion and congestion remains unclear. Consequently, we attributed the significant association of hypoalbuminemia and elevated CRP with mortality predominantly to the cold-wet group, but further studies are needed to draw definitive conclusions on this issue.

In previous studies, the relationship of HF with CRP, ESR, and albumin has been examined separately. In contrast, in our study, we conducted a more comprehensive analysis by examining the relationship of these three acute phase reactants with mortality and prognosis in patients hospitalized with ADHF. In addition, our study is pioneering in assessing the effect of these three acute phase reactants on prognosis and mortality according to the new phenotypic classification of HF.

Limitations

The limitation of this study is that the patients were not followed up for a long time after hospital discharge.

Conclusion

In AHF patients, low albumin values at initial hospitalization and discharge, high CRP and ESR values at initial hospitalization predict increased mortality. This outcome was related to the phenotypic classes of heart failure and was especially evident in the cold-wet class. However, larger studies are needed to confirm these findings.

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