ORIGINAL RESEARCH

Bagcilar Med Bull 2026;11(3):359-365
DOI: 10.4274/BMB.galenos.2026.46036

Prognostic Impact of Combined Anemia and Hypoalbuminemia Phenotypes in Patients Hospitalized with Acute Heart Failure

Akut Kalp Yetersizliği Hastalarında Kombine Anemi ve Hipoalbüminemi Fenotiplerinin Prognostik Etkisi

Can Baba Arın[1], Mehmet Zafer Aydın[2], Mehmet Kamil Teber[2], Nizamettin Selçuk Yelgeç[1]

[1]University of Health Sciences Turkey, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiology, İstanbul, Turkey
[2]University of Health Sciences Turkey, Kayseri City Training and Research Hospital, Department of Cardiology, Kayseri, Turkey

Abstract

Objective: Hematologic and nutritional abnormalities are common in patients hospitalized with acute heart failure and may be associated with adverse outcomes. This study aimed to evaluate the clinical characteristics and in-hospital mortality of patients classified according to combined anemia and hypoalbuminemia phenotypes.

Method: This retrospective study included 220 consecutive patients hospitalized with acute heart failure. Anemia was defined according to World Health Organization sex-specific hemoglobin thresholds, and hypoalbuminemia was defined as a serum albumin level <3.5 g/dL. Patients were classified into four phenotypes: Neither anemia nor hypoalbuminemia, anemia alone, hypoalbuminemia alone, and both anemia and hypoalbuminemia. Associations between phenotype groups and all-cause in-hospital mortality were evaluated using categorical comparisons and logistic regression. Given the limited number of mortality events, a parsimonious age-adjusted model and bootstrap sensitivity analysis were performed.

Results: Among 220 patients, 21 (9.5%) died during hospitalization. The four phenotype groups comprised 39 patients with neither abnormality, 34 with anemia alone, 45 with hypoalbuminemia alone, and 102 with both abnormalities. In-hospital mortality rates were 7.7%, 17.6%, 8.9%, and 7.8%, respectively, with no significant overall difference among groups (Fisher's exact p=0.402). In the age-adjusted logistic regression model, phenotype group was not significantly

Öz

Amaç: Akut kalp yetersizliği nedeniyle hastaneye yatırılan hastalarda hematolojik ve nütrisyonel bozukluklar sık görülmekte olup olumsuz klinik sonuçlarla ilişkili olabilir. Bu çalışmada, kombine anemi-hipoalbüminemi fenotiplerine göre sınıflandırılan hastaların klinik özelliklerinin ve hastane içi mortalitelerinin değerlendirilmesi amaçlandı.

Yöntem: Bu retrospektif çalışmaya akut kalp yetersizliği nedeniyle hastaneye yatırılan 220 ardışık hasta dahil edildi. Anemi, Dünya Sağlık Örgütü'nün cinsiyete özgü hemoglobin eşik değerlerine göre; hipoalbüminemi ise serum albümin düzeyinin <3,5 g/dL olması olarak tanımlandı. Hastalar anemi ve hipoalbüminemi bulunmayanlar, yalnızca anemisi olanlar, yalnızca hipoalbüminemisi olanlar ve hem anemi hem de hipoalbüminemisi olanlar olmak üzere dört fenotipe ayrıldı. Fenotip grupları ile tüm nedenlere bağlı hastane içi mortalite arasındaki ilişkiler kategorik karşılaştırmalar ve lojistik regresyon analizleriyle değerlendirildi. Mortalitenin sınırlı sayıda olması nedeniyle parsimoniyöz, yaşa göre düzeltilmiş bir model ve bootstrap duyarlılık analizi uygulandı.

Bulgular: Toplam 220 hastanın 21'i (%9,5) hastanede yatış sırasında yaşamını yitirdi. Anemi ve hipoalbüminemisi olmayan grupta 39, yalnızca anemisi olan grupta 34, yalnızca hipoalbüminemisi olan grupta 45 ve her iki bozukluğun birlikte bulunduğu grupta 102 hasta vardı. Hastane içi mortalite oranları sırasıyla %7,7, %17,6, %8,9 ve

Address for Correspondence: Can Baba Arın, University of Health Sciences Turkey, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiology, İstanbul, Turkey
E-mail: canbabaarin@gmail.com **ORCID:** orcid.org/0000-0003-1647-3120
Received: 16.05.2026 **Accepted:** 23.09.2026 **Publication Date:** 29.09.2026

Cite this article as: Arın CB, Aydın MZ, Teber MK, Yelgeç NS. Prognostic impact of combined anemia and hypoalbuminemia phenotypes in patients hospitalized with acute heart failure. Bagcilar Med Bull. 2026;11(3):359-365

Abstract

associated with in-hospital mortality (overall p=0.391), and none of the individual phenotype comparisons reached statistical significance. Bootstrap sensitivity analysis yielded consistent findings. Receiver operating characteristic analysis showed limited discrimination for mortality for both albumin [area under the curve (AUC) 0.594, 95% confidence interval (CI) 0.477-0.711; p=0.157] and hemoglobin (AUC 0.474, 95% CI 0.344-0.603; p=0.694).

Conclusion: Combined anemia-hypoalbuminemia phenotypes were associated with distinct clinical and laboratory profiles but were not significantly associated with in-hospital mortality in this cohort. Neither admission albumin nor hemoglobin alone demonstrated significant discrimination for in-hospital mortality. These findings should be interpreted cautiously given the limited number of mortality events.

Keywords: Acute heart failure, anemia, hypoalbuminemia, mortality, phenotype, prognosis

Öz

%7,8 olup gruplar arasında anlamlı fark saptanmadı (Fisher'ın kesin testi, p=0,402). Yaşa göre düzeltilmiş lojistik regresyon modelinde fenotip grubu hastane içi mortalite ile anlamlı olarak ilişkili değildi (genel p=0,391) ve fenotiplerin hiçbirinde referans gruba kıyasla anlamlı ilişki saptanmadı. Bootstrap duyarlılık analizi de benzer sonuçlar verdi. Alıcı işlem karakteristiği analizinde albümin [eğri altında kalan alan (AUC) 0,594; %95 güven aralığı (GA) 0,477-0,711; p=0,157] ve hemoglobinin (AUC 0,474; %95 GA 0,344-0,603; p=0,694) hastane içi mortaliteyi ayırt etme performansı sınırlı bulundu.

Sonuç: Kombine anemi-hipoalbüminemi fenotipleri farklı klinik ve laboratuvar özellikleriyle ilişkili olmakla birlikte, bu kohortta hastane içi mortalite ile anlamlı bir ilişki göstermedi. Başvuru sırasındaki albümin ve hemoglobin düzeylerinin hiçbiri tek başına hastane içi mortalite açısından anlamlı ayırt edici performans göstermedi. Bulgular, mortalite olaylarının sınırlı sayıda olması nedeniyle dikkatle yorumlanmalıdır.

Anahtar kelimeler: Akut kalp yetersizliği, anemi, hipoalbüminemi, mortalite, fenotip, prognoz

Introduction

Acute heart failure (AHF) is among the foremost precipitants of urgent hospital admission and is burdened by substantial early mortality and high rates of return hospitalization, even in the context of evolving treatment paradigms (1). The trajectory of illness among affected individuals is highly heterogeneous, mirroring the diverse pathophysiological substrates and varying degrees of cardiac decompensation that underlie this syndrome (2). As a result, the availability of practical and dependable markers for prognostic stratification is critical to enable individualized patient management and support timely therapeutic decision-making (3).

Anemia is a prevalent comorbid condition in the heart failure population, consistently linked to impaired exercise tolerance, increased readmission frequency, and worse long-term survival (4,5). The mechanisms underlying anemia in this context are heterogeneous, encompassing contributions from renal dysfunction, iron deficiency states, chronic low-level systemic inflammation, hemodynamic fluid redistribution, and disruption of neurohumoral regulatory systems (6). In parallel, a decline in serum albumin concentration is a common biochemical accompaniment of acute cardiac decompensation, potentially reflecting malnutrition, hepatic congestion from venous hypertension, systemic inflammatory burden, and the cumulative severity of the patient's condition (7,8). A body of prior evidence has demonstrated that hypoalbuminemia confers elevated risk for adverse cardiovascular outcomes and mortality in populations with heart failure (9).

Although both abnormalities have individually demonstrated prognostic relevance, relatively little attention has been directed toward their simultaneous occurrence as an integrated clinical phenotype. Considering that anemia and hypoalbuminemia may reflect partially overlapping yet distinct aspects of systemic disease burden, evaluating their combined phenotypic patterns may provide additional insight into clinical heterogeneity and their potential relationship with short-term outcomes (10).

With this rationale, the present investigation was undertaken to evaluate the association between phenotypic combinations of anemia and hypoalbuminemia and all-cause in-hospital mortality in a cohort of patients hospitalized with AHF.

Materials and Methods

Study Design and Population

This was a single-center retrospective observational study carried out at at Mogadishu Somalia-Turkey Recep Tayyip Erdoğan Training and Research Hospital. Consecutive adult patients admitted with a confirmed diagnosis of AHF were screened for eligibility. A total of 220 patients with available clinical and laboratory data at admission formed the final study cohort.

AHF was diagnosed in accordance with established international guideline criteria, incorporating clinical

features, relevant laboratory results, and echocardiographic findings consistent with acute decompensated heart failure (1). Demographic characteristics, comorbidities, laboratory parameters, and echocardiographic data were obtained from the institutional electronic medical records.

Definitions and Study Groups

Based on anemia and hypoalbuminemia status, patients were assigned to one of four phenotypic subgroups:

1. No anemia/no hypoalbuminemia

2. Anemia only

3. Hypoalbuminemia only

4. Combined anemia and hypoalbuminemia.

Classification was based on hemoglobin and serum albumin values obtained at hospital admission. Anemia was defined according to World Health Organization thresholds as a hemoglobin level below 13 g/dL in men and below 12 g/dL in women. Hypoalbuminemia was defined as a serum albumin concentration below 3.5 g/dL.

Data Collection

The following baseline variables were systematically recorded: Age, sex, diabetes mellitus, hypertension, chronic kidney disease, left ventricular ejection fraction, hemoglobin, serum albumin, C-reactive protein (CRP), pro-B-type natriuretic peptide, and neutrophil-to-lymphocyte ratio (NLR).

Study Outcome

The primary study endpoint was all-cause mortality occurring during the index hospitalization.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation, and categorical variables as numbers and percentages. Between-group comparisons of continuous variables were performed using one-way analysis of variance. Categorical variables were compared using the Pearson chi-square test or Fisher's exact test, as appropriate according to expected cell counts.

Associations with in-hospital mortality were evaluated using binary logistic regression analysis. Univariable analyses were first performed for the anemia-hypoalbuminemia phenotype categories and selected clinically relevant variables. Because only 21 in-hospital deaths occurred, the multivariable analysis was intentionally restricted to a parsimonious model including age and the four-category anemia-hypoalbuminemia phenotype variable to reduce the risk of model overfitting. The group with neither anemia nor hypoalbuminemia served as the reference category. Results are reported as odds ratios (ORs) with 95% confidence intervals (CIs). To assess the robustness of the regression findings in the setting of the limited number of events, a sensitivity analysis using 1,000 bootstrap samples with bias-corrected and accelerated 95% CIs was also performed.

Receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminatory ability of admission serum albumin and hemoglobin levels for in-hospital mortality. Death was defined as the positive outcome. Areas under the curve (AUCs) with 95% CIs were calculated. All tests were two-sided, and a p-value <0.05 was considered statistically significant.

Ethical Approval

Ethical approval was obtained from the Institutional Ethics Committee of Mogadishu Somalia-Turkey Recep Tayyip Erdoğan Training and Research Hospital (approval no: 1206, date: 02.05.2025). All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

Results

A total of 220 patients were included in the analysis. Based on admission hemoglobin and serum albumin levels, 39 patients (17.7%) had neither anemia nor hypoalbuminemia, 34 (15.5%) had anemia alone, 45 (20.5%) had hypoalbuminemia alone, and 102 (46.4%) had both anemia and hypoalbuminemia. Significant between-group differences were observed for hemoglobin and serum albumin levels ($p<0.001$ for both), as expected from the phenotype definitions. Baseline demographic, clinical, and laboratory characteristics according to phenotype group are presented in Table 1. Overall, 21 of 220 patients (9.5%) died during the index hospitalization. In-hospital mortality occurred in 3 of 39 patients (7.7%) with neither anemia nor hypoalbuminemia, 6 of 34 (17.6%) with anemia alone, 4 of 45 (8.9%) with hypoalbuminemia alone, and 8 of 102 (7.8%) with both abnormalities. Although mortality was numerically highest in the anemia-only group, the overall difference among phenotype groups was not statistically significant (Fisher's exact $p=0.402$). Therefore, no stepwise pattern of increasing mortality across the phenotype categories was observed.

Results of the logistic regression analyses for in-hospital mortality are presented in Table 2. In univariable analyses, none of the anemia-hypoalbuminemia phenotype categories was significantly associated with in-hospital mortality compared with patients with neither condition. Given the limited number of mortality events (n=21), the multivariable analysis was intentionally restricted to an age-adjusted parsimonious model to reduce the risk of overfitting.

In the age-adjusted model, the anemia-hypoalbuminemia phenotype variable was not significantly associated with in-hospital mortality overall (p=0.391). Compared with patients with neither abnormality, the adjusted OR was 2.431 (95% CI 0.552-10.706; p=0.240) for anemia alone, 1.211 (95% CI 0.252-5.820; p=0.811) for hypoalbuminemia alone, and 0.909 (95% CI 0.224-3.689; p=0.894) for combined anemia and hypoalbuminemia. Age was also not statistically significant in the adjusted model (OR 1.029, 95% CI 0.996-1.063; p=0.083). A sensitivity analysis based on 1,000 bootstrap samples yielded consistent findings, with no phenotype category reaching statistical significance; the wide bootstrap CIs further indicated substantial uncertainty associated with the limited number of mortality events.

ROC analysis demonstrated limited discriminatory performance of both admission serum albumin and hemoglobin for in-hospital mortality (Figure 1). The AUC was 0.594 (95% CI 0.477-0.711; p=0.157) for albumin and 0.474 (95% CI 0.344-0.603; p=0.694) for hemoglobin (n=220). Neither biomarker therefore demonstrated statistically significant discrimination for in-hospital mortality.

Table 1. Baseline characteristics according to combined anemia and hypoalbuminemia phenotypes

Variable	No anemia/no hypoalb. (n=39)	Anemia only (n=34)	Hypoalb. only (n=45)	Both (n=102)	p-value
Age, years	58.69±10.08	60.12±14.53	56.33±15.13	61.05±15.87	0.333
Male, n (%)	27 (69.2)	20 (58.8)	30 (66.7)	61 (59.8)	0.660
Diabetes mellitus, n (%)	29 (74.4)	22 (64.7)	27 (60.0)	51 (50.0)	0.054
Hypertension, n (%)	26 (66.7)	20 (58.8)	21 (46.7)	51 (50.0)	0.217
Chronic kidney disease, n (%)	6 (15.4)	7 (20.6)	3 (6.7)	13 (12.7)	0.323
Hemoglobin, g/dL	14.25±1.23	10.72±1.28	13.85±1.10	10.25±1.50	<0.001
Albumin, g/dL	4.20±0.38	4.09±0.47	2.60±0.53	2.60±0.52	<0.001
CRP, mg/L	44.79±38.71	68.09±70.12	87.16±96.66	75.66±69.31	0.054
ProBNP, pg/mL	5685±3231	6114±3468	6541±3568	5743±3457	0.572
Ejection fraction, %	38.85±22.26	37.21±18.55	43.44±21.69	44.26±19.48	0.232
NLR	3.11±2.11	7.93±18.18	5.90±6.21	7.77±13.81	0.199
Mortality, n (%)	3 (7.7)	6 (17.6)	4 (8.9)	8 (7.8)	0.402*

*: Data are presented as mean ± SD for continuous variables and n (%) for categorical variables. Group comparisons were performed using one-way analysis of variance or Pearson chi-square test, as appropriate. Fisher's exact test was used for the mortality comparison because of small expected cell counts. CRP: C-reactive protein, proBNP: Pro-B-type natriuretic peptide, NLR: Neutrophil-to-lymphocyte ratio, Hypoalb.: Hypoalbuminemia

Table 2. Logistic regression analysis for in-hospital mortality

Predictor	Univariable OR (95% CI)	p-value	Age-adjusted OR (95% CI)	p-value
Phenotype				
No anemia/no hypoalbuminemia	Reference	—	Reference	—
Anemia only	2.571 (0.590-11.198)	0.208	2.431 (0.552-10.706)	0.240
Hypoalbuminemia only	1.171 (0.245-5.585)	0.843	1.211 (0.252-5.820)	0.811
Both anemia and hypoalbuminemia	1.021 (0.257-4.065)	0.976	0.909 (0.224-3.689)	0.894
Age, per year	1.028 (0.996-1.060)	0.086	1.029 (0.996-1.063)	0.083
Ejection fraction, per 1%	0.997 (0.975-1.019)	0.796	—	—
Chronic kidney disease	2.279 (0.765-6.784)	0.139	—	—

OR: Odds ratio, CI: Confidence interval. The reference category was no anemia/no hypoalbuminemia. Because only 21 in-hospital deaths occurred, the multivariable model was intentionally restricted to age and phenotype group to reduce the risk of overfitting. Ejection fraction and chronic kidney disease were therefore evaluated only in univariable analyses. A sensitivity analysis using 1.000 bootstrap samples with bias-corrected and accelerated 95% confidence intervals did not materially alter the interpretation of the regression findings

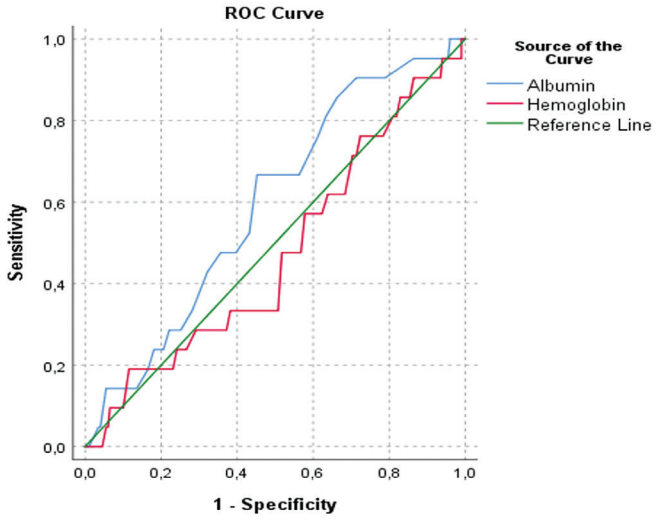

Figure 1. Receiver operating characteristic (ROC) curves for serum albumin and hemoglobin in discriminating in-hospital mortality. The areas under the curve were 0.594 (95% CI 0.477-0.711; p=0.157) for albumin and 0.474 (95% CI 0.344-0.603; p=0.694) for hemoglobin (n=220)

CI: Confidence interval

Discussion

This study evaluated the association between combined anemia-hypoalbuminemia phenotypes and in-hospital mortality among patients hospitalized with AHF. The principal finding was that, after reconstruction of the phenotype groups and reanalysis of the complete 220-patient cohort, in-hospital mortality did not differ significantly across the four phenotype categories. Although mortality was numerically highest among patients with anemia alone, none of the phenotype categories was significantly associated with mortality in either univariable or age-adjusted logistic regression analyses. In addition, neither admission serum albumin nor hemoglobin demonstrated statistically significant discriminatory ability for in-hospital mortality. These findings suggest that, in the present cohort, anemia-hypoalbuminemia phenotyping primarily characterized clinical and laboratory heterogeneity rather than providing independent short-term prognostic discrimination.

Anemia is widely recognized as a frequent and clinically meaningful accompaniment of heart failure and has been associated with reduced exercise capacity, increased rates of rehospitalization, and poorer long-term survival (4,5). Its pathophysiology in heart failure is multifactorial and may involve iron deficiency, persistent systemic inflammation, impaired renal function, neurohormonal dysregulation, and hemodilution related to volume overload (6,11,12). In the present study, patients with anemia alone had the numerically highest in-hospital mortality rate; however, this difference was not statistically significant, and the association remained non-significant after adjustment for age. The wide CIs around the effect estimates indicate substantial uncertainty and preclude firm conclusions regarding the independent short-term prognostic contribution of anemia in this cohort.

Hypoalbuminemia is also frequently encountered in patients with heart failure and may reflect several interrelated processes, including systemic inflammation, hepatic congestion secondary to elevated venous pressure, nutritional impairment, and greater overall disease burden (7,8). Previous studies have reported associations between lower serum albumin levels and adverse outcomes in both acute and chronic heart failure populations (9,13). However, the present findings did not demonstrate a significant association between the isolated hypoalbuminemia phenotype and in-hospital mortality. Importantly, this should not be interpreted as contradicting the established biological and prognostic relevance of hypoalbuminemia in broader heart failure populations. Rather, it indicates that such an association could not be demonstrated in this particular cohort, in which the number of in-hospital deaths was limited.

The combined presence of anemia and hypoalbuminemia was likewise not associated with an increased risk of in-hospital mortality compared with the reference phenotype. Although anemia and hypoalbuminemia may arise through partially shared mechanisms, including inflammation, congestion, renal dysfunction, nutritional impairment, and neurohormonal activation (14), the present data do not support an additive prognostic effect of their coexistence. At the same time, the absence of a statistically significant association should not be interpreted as evidence that anemia adds no risk in patients with hypoalbuminemia. The relatively small number of mortality events and the resulting wide CIs limit the ability to distinguish between true absence of an association and insufficient statistical precision.

Inflammatory markers also varied numerically across the phenotype groups. CRP levels tended to be higher in groups characterized by hypoalbuminemia, whereas NLR values showed considerable variability. However, the between-group differences did not reach conventional statistical significance in the reanalysis. Therefore, although systemic inflammation remains a biologically plausible link among

AHF, anemia, and hypoalbuminemia, the present findings do not establish a graded inflammatory pattern across the four phenotypes.

ROC analysis provided additional perspective on the prognostic performance of the individual biomarkers. Admission serum albumin yielded an AUC of 0.594, whereas hemoglobin yielded an AUC of 0.474; neither value was statistically significant. These findings indicate limited discriminatory performance of either biomarker alone for in-hospital mortality in this cohort. Importantly, the current analysis also does not demonstrate that the combined phenotype classification provides incremental prognostic value beyond albumin, hemoglobin, or established clinical assessment. Accordingly, the phenotype approach should be regarded as an exploratory method of characterizing clinical heterogeneity rather than as a validated risk-stratification tool.

Clinical Implications

Hemoglobin and serum albumin are routinely available laboratory measurements obtained at hospital admission and can readily identify clinically recognizable anemia-hypoalbuminemia phenotypes without additional testing or cost. Nevertheless, the present findings do not support using these phenotype categories as a stand-alone tool for short-term mortality prediction in patients with AHF. Their potential clinical relevance may instead lie in identifying different patterns of hematologic and nutritional abnormalities that warrant broader clinical assessment. Larger prospective studies with a greater number of outcome events are required to determine whether these phenotypes provide prognostic information beyond established clinical risk factors.

Study Limitations

Several limitations of this study should be acknowledged. First, its retrospective, single-center design limits the generalizability of the findings to broader patient populations and different healthcare settings. Second, although the overall cohort included 220 patients, only 21 in-hospital deaths occurred. This limited number of outcome events reduced statistical power, particularly for comparisons across the four phenotype groups, and resulted in wide CIs around several effect estimates. To reduce the risk of overfitting, the multivariable analysis was therefore intentionally restricted to a parsimonious age-adjusted model. Although a bootstrap sensitivity analysis using 1.000 samples yielded findings consistent with the primary regression analysis, the substantial uncertainty surrounding the estimates should be considered when interpreting the results. Accordingly, the absence of statistically significant associations should not be interpreted as evidence of equivalence or absence of clinically relevant effects.

Furthermore, potentially important confounding variables, including iron status parameters, formal nutritional assessment scores, and a broader range of inflammatory biomarkers, were not systematically available. Residual confounding therefore cannot be excluded. Finally, outcome assessment was restricted to all-cause mortality during the index hospitalization, and no post-discharge or long-term outcomes were evaluated. Larger, prospective, multicenter studies with a greater number of outcome events and longer follow-up are needed to determine whether anemia-hypoalbuminemia phenotypes have prognostic value beyond established clinical risk factors.

Conclusion

In patients hospitalized with AHF, combined anemia-hypoalbuminemia phenotypes were not significantly associated with in-hospital mortality. Neither isolated hypoalbuminemia nor the combined presence of anemia and hypoalbuminemia independently predicted in-hospital death, and admission serum albumin and hemoglobin demonstrated limited discriminatory performance when evaluated individually. Although these readily available laboratory parameters may help characterize hematologic and nutritional heterogeneity in AHF, the present findings do not support their use, either individually or as combined phenotypes, as stand-alone tools for short-term mortality risk stratification. Larger prospective studies with a greater number of outcome events are needed to clarify their potential prognostic value.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Institutional Ethics Committee of Mogadishu Somalia-Turkey Recep Tayyip Erdoğan Training and Research Hospital (approval no: 1206, date: 02.05.2025). All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

Informed Consent: It was not deemed necessary, as the study was retrospective.

Acknowledgments

OpenAI's ChatGPT was used only for language editing and manuscript organization support. All scientific content,

interpretation, and final approval were performed entirely by the authors.

Footnotes

Authorship Contributions

Surgical and Medical Practices: C.B.A., N.S.Y., Concept: C.B.A., M.Z.A., Design: C.B.A., M.Z.A., Data Collection or Processing: C.B.A., M.K.T., Analysis or Interpretation: M.Z.A., M.K.T., N.S.Y., Literature Search: M.K.T., Writing: N.S.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al.; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42(36):3599-3726. Erratum in: Eur Heart J. 2021;42(48):4901.
2. Gheorghiade M, Pang PS. Acute heart failure syndromes. J Am Coll Cardiol. 2009;53(7):557-573.
3. Metra M, Teerlink JR. Heart failure. Lancet. 2017;390(10106):1981-1995.
4. Felker GM, Adams KF Jr, Gattis WA, O'Connor CM. Anemia as a risk factor and therapeutic target in heart failure. J Am Coll Cardiol. 2004;44(5):959-966.
5. Horwich TB, Fonarow GC, Hamilton MA, MacLellan WR, Borenstein J. Anemia is associated with worse symptoms, greater impairment in functional capacity and a significant increase in mortality in patients with advanced heart failure. J Am Coll Cardiol. 2002;39(11):1780-1786.
6. Tang WH, Van Lente F, Shrestha K, Troughton RW, Francis GS, Tong W, et al. Impact of myocardial function on cystatin C measurements in chronic systolic heart failure. J Card Fail. 2008;14(5):394-399.
7. Arques S, Ambrosi P. Human serum albumin in the clinical syndrome of heart failure. J Card Fail. 2011;17(6):451-458.
8. Bonilla-Palomas JL, Gámez-López AL, Moreno-Conde M, López-Ibáñez MC, Anguita-Sánchez MP, Gallego de la Sacristana A, et al. Hypoalbuminemia in acute heart failure patients: causes and its impact on hospital and long-term mortality. J Card Fail. 2014;20(5):350-358.
9. Ancion A, Allepaerts S, Oury C, Gori AS, Piérard LA, Lancellotti P. Serum albumin level and hospital mortality in acute non-ischemic heart failure. ESC Heart Fail. 2017;4(2):138-145.
10. Felker GM, Gattis WA, Leimberger JD, Adams KF, Cuffe MS, Gheorghiade M, et al. Usefulness of anemia as a predictor of death and rehospitalization in patients with decompensated heart failure. Am J Cardiol. 2003;92(5):625-628.
11. Westenbrink BD, Voors AA, de Boer RA, Schuringa JJ, Klinkenberg T, van Veldhuisen DJ. Bone marrow dysfunction in chronic heart failure patients. Eur J Heart Fail. 2010;12(7):676-684.
12. Uthamalingam S, Kandala J, Daley M, Patvardhan E, Capodilupo R, Moore SA, et al. Serum albumin and mortality in acutely decompensated heart failure. Am Heart J. 2010;160(6):1149-1155.
13. von Haehling S, Jankowska EA, van Veldhuisen DJ, Ponikowski P, Anker SD. Iron deficiency and cardiovascular disease. Nat Rev Cardiol. 2015;12(11):659-669.
14. Núñez J, Núñez E, Bodí V, Sanchis J, Miñana G, Mainar L, et al. Usefulness of the neutrophil to lymphocyte ratio in predicting long-term mortality in ST segment elevation myocardial infarction. Am J Cardiol. 2008;101(6):747-752.