RESEARCH ARTICLE

Can malnutrition predict mortality in heart failure with low ejection fraction? Implications for multidisciplinary care

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Abstract

Objective: To evaluate malnutrition's predictive potential and significance with respect to overall mortality related to heart failure with low ejection fraction.

Method: The cross-sectional, prospective study was conducted from June to August 2022 at the Kahramanmaras Sutcu Imam University and the Osmaniye Duzici State Hospital, Turkiye, and comprised heart failure patients with low ejection fraction. Malnutrition status was evaluated using the Global Leadership Initiative on Malnutrition criteria. The patients were divided into malnutrition group A and healthy group B. Prognostic nutritional index and controlling nutritional status scores were calculated and compared. All the patients were followed up either through telephone or clinic visits for up to one year. The number of hospitalisations and length of hospital stay within a year were recorded and compared. Data was analysed using SPSS 22.

Results: Of the 216 patients with mean age 67.10±5.59 years, 110(50.9%) were males and 106(49.1%) were females. There were 57(26.4%) subjects in group A and 159(73.6%) in group B. Malnutrition, length of hospital stay and increased neutrophils were independent predictors of death (p<0.05).

Conclusion: Malnutrition was found to be common among heart failure outpatients, and could significantly help identify those at increased mortality risk.

Keywords: Malnutrition, Heart failure, Low ejection fraction, Mortality. (JPMA 76: 65; 2026)

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Introduction

Malnutrition is common among individuals suffering from heart failure (HF). Given the increasing ageing population, there is an expected rise in the number of HF patients, particularly among the elderly, at a higher risk of developing malnutrition due to reduced daily activity, sarcopenia and cognitive impairment, commonly referred to as frailty.[1] Several factors contribute to the onset of malnutrition, such as decreased appetite, malabsorption due to intestinal swelling, increased energy requirements, reduced food intake, amplified nutrient loss, heightened metabolism, and increased cytokines, like tumour necrosis factor-alpha (TNF-α), cortisol, epinephrine, renin and aldosterone, and hypercatabolism induced by the cytokines. Healthcare professionals have often relied on measures like body weight or body mass index (BMI) to evaluate this condition. Notably, significant weight loss indicates the progression of cardiac cachexia, a state of excessive catabolism, and its prognosis is highly unfavourable once it occurs. Other indicators, like serum albumin or pre-albumin levels and lymphocyte counts, have been found to mirror nutritional status, and are linked to a poorer prognosis. Since these markers can be influenced by the dilution effects of fluid retention associated with HF, relying on a single parameter is not ideal for accurately assessing a patient's nutritional status.[2] Recently, three screening tools have been employed to comprehensively and objectively evaluate the nutritional status of HF patients. To standardise the diagnosis of malnutrition, the Global Leadership Initiative on Malnutrition (GLIM) has introduced new universally applicable criteria for diagnosing malnutrition in adults.[3] The Controlling Nutritional Status (CONUT) scores, which take into account serum albumin levels, total cholesterol levels, and total lymphocyte counts, align with established nutritional assessments, like the Subjective Global Assessment (SGA).[4] The Prognostic Nutritional Index (PNI), which involves serum albumin levels and lymphocyte counts, was initially developed for cirrhotic patients, but has since gained widespread use as an objective measure of nutritional status.[5] While all three nutritional assessment tools have demonstrated their ability to predict adverse outcomes in acute and chronic HF patients, it remains unclear how the rates of diagnosing malnutrition and their

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predictive prognostic capabilities may vary across these scoring methods in larger HF populations. To our knowledge, no study has so far assessed the prognostic significance of the GLIM, CONUT, and PNI criteria on mortality in HF outpatients. The current study was planned to fill the gap in literature by evaluating the predictive potential and significance of malnutrition, as determined by the GLIM, CONUT and PNI criteria, regarding overall mortality related to HF with low ejection fraction (EF).

Patients and Methods

The cross-sectional, prospective study was conducted from June to August 2022 at the Kahramanmaras Sutcu Imam University (KSIU) and the Osmaniye Duzici State Hospital (ODSH), Turkiye. After approval from the KSIU ethics review committee, the sample size was calculated with α=0.05, power=0.80, and an effect size of 0.44. A priori power analysis was performed using G*Power (version 3.1.9.7, Düsseldorf, Germany). Under unequal allocation, the minimum required number for the smaller group was 57 patients. Our study included 216 patients (57 [26.4%] in Group A and 159 [73.6%] in Group B), thus exceeding this requirement. Notably, a previous nutritional validation study was successfully conducted with 53 patients, supporting the adequacy of our sample size.[6] The sample was raised using consecutive sampling technique. Those included were HF patients of either gender with left ventricular ejection fraction (LVEF) < 40%. Patients with moderate or severe valvular regurgitation or stenosis, those with metallic or bioprosthetic heart valves, patients with malignancy, patients with acute or chronic infections at the time of initial presentation, those with episodes of acute coronary syndrome within the preceding two months were excluded, and so were those who were lost to follow-up.

After taking written informed consent, demographic and clinical characteristics of all the patients were recorded. The participants underwent surface electrocardiography and two-dimensional (2D) echocardiography. Routine haematological and biochemical laboratory tests were also conducted. The malnutrition status of the patients was evaluated according to the GLIM criteria.[3] PNI and CONUT scores were also calculated.[4,5]

According to the GLIM criteria, malnutrition is assessed in two stages. The first stage is the initial screening in which individuals at risk of malnutrition are identified. The validated Nutritional Risk Screening 2002 (NRS-2002) tool was used for this screening. A total score >3, according to the NRS-2002, identified individuals at risk of malnutrition. In the second stage, malnutrition was identified, and its severity was evaluated. This stage included phenotypic and aetiological criteria. Phenotypic criteria included involuntary weight loss, reduced muscle mass, and low body mass index (BMI). The aetiological criteria examined the two categories of disease/inflammation burden and inadequate food consumption or digestion. According to the GLIM criteria, the diagnosis of malnutrition was defined as the presence of one phenotypic and aetiological criterion. Malnutrition severity was assessed as Grade 1 (moderate) and Grade 2 (severe). This was based solely on the phenotypic criteria. Grade 1 malnutrition was defined as weight loss of 5-10% in the preceding six months, or 10-20% in >6 months, or BMI <20 at age <70 years or BMI <22 at age ≥70 years, or mild-to-moderate decreased muscle mass by validated assessment methods. Grade 2 malnutrition was assessed as >10% weight loss in the preceding six months or >20% weight loss in >6 months, or BMI <18.5 at age <70 years or BMI <20 at age ≥70 years, or severe decreased in muscle mass by validated assessment methods.[7]

The PNI is a simple assessment method that reflects a person's nutritional and immunological status. PNI has increased cardiovascular disease, malignancy and infection states. The PNI score was calculated using the formula: 10×serum albumin (g/dl)+0.005×total lymphocyte count (mm). Severe malnutrition was classified as PNI <35, moderate malnutrition as PNI 35-38, and PNI >38 was identified as normal.[8]

The CONUT score comprised three parameters; total cholesterol, lymphocyte count, and albumin, reflecting nutritional status, the state of the immune system, and protein reserve, respectively. The total score was categorised as ≤2 low at ≥3 high.[9]

The presence of HF was determined by echocardiographic examination. Parasternal (short and long) and apical (two-chamber, four-chamber) images were obtained from the participants in the left lateral and decubitus positions, according to the American and European echocardiography guidelines.[10] LVEF was calculated using Simpson's method. LVEF <40% was considered HF with low LVEF.[11]

All the patients were followed up for up to one year, and values were monitored at 1, 6 and 12 months. In cases requiring home care and unable to attend hospital visits, follow-up information was collected via phone interviews with either the patients themselves or their relatives. Data regarding admissions to other hospitals and survival status was gathered through both the hospital's electronic records and telephone follow-ups. The number of hospitalisations and length of hospital stay (LOS) within a year were also recorded. The patients were divided into malnutrition group A and healthy group B. Death from any cause was considered the endpoint.

Data was analysed using SPSS 22. Data normality was evaluated using the Shapiro-Wilk or Kolmogorov-Smirnov tests. Continuous variables were expressed as mean± standard deviation or median with interquartile range (IQR), while categorical variables were presented as frequencies and percentages. Comparisons between patients with and without malnutrition, as well as between survivors and non-survivors, were conducted using the independent sample t-test or Mann-Whitney U test for continuous variables, and the Pearson chi-square test or Fisher's exact test for categorical variables. To identify independent predictors of mortality, binary logistic regression analysis was performed, including five variables: neutrophil count (10^3/μL), lymphocyte count (10^3/μL), LOS (days), CONUT score, and the presence of GLIM-defined malnutrition. CONUT score and lymphocyte count were excluded from the regression model despite their univariate significance, due to potential multicollinearity, as CONUT encompasses lymphocyte count and overlaps with variables like GLIM. The Kaplan-Meier survival analysis was applied to assess the effect of GLIM-based malnutrition on patient survival. P<0.05 was considered statistically significant.

Results

Of the 216 patients with mean age 67.10±5.59 years, 110(50.9%) were males and 106(49.1%) were females. As per GLIM, there were 57(26.4%) subjects in group A and 159(73.6%) in group B. High CONUT malnutrition was present in 43(19.9%) patients, while, according to PNI, 31(14.4%) patients had severe malnutrition. Among known diseases, the incidence rate of chronic obstructive pulmonary disease (COPD) (p<0.001) was higher in GLIM group A. Angiotensin receptor/neprilysin inhibitor (ARNI) use and the incidence of coronary artery disease (CAD) (p<0.001) were higher in group B (Table 1).

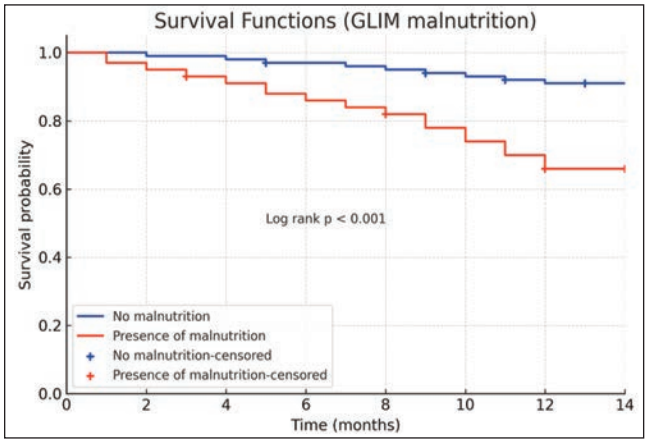

Figure: Kaplan Meier Survival curve of patients with GLIM malnutrition.
GLIM: Global Leadership Initiative on Malnutrition.

Table-1: Demographic and clinical features.

	GLIM malnutrition (-) (Group B) (n=159) [n(%)]	GLIM malnutrition (+) (Group A) (n=57) [n(%)]	*p*-value
Mean Age (year)	66.83±5.95	67.84±4.42	0.246[a]
Male	77 (48.4)	33 (57.9)	0.220[b]
Mean BMI (kg/m^2)	22.45±3.75	22.56±4.03	0.859[a]
Mean GFR (mL/min/1.73 m^2)	56.50±16.54	57.18±17.43	0.792[a]
Hypertension, n (%)	40 (25.2)	17 (29.8)	0.493[b]
Diabetes mellitus, n (%)	29 (18.2)	10 (17.5)	0.907[b]
CAD	138 (86.8)	32 (56.1)	<0.001[b]
COPD	12 (7.5)	16 (28.1)	<0.001[b]
Presence of CIED	26 (16.4)	4 (7)	0.080[b]
The number of hospitalizations	1 (0–8)	1 (0–7)	0.432c
Total length of hospital stay (day)	5 (0–24)	5 (0–25)	0.328c
Mean LVEF, %	30.38±5.50	31.14±5.02	0.367[a]
NYHA classification			
I-II	91 (57.2)	33 (57.9)	0.931[b]
III-IV	68 (42.8)	24 (42.1)	
Usage Drug			
Antiplatelet	119 (74.8)	48 (84.2)	0.147[b]
Anticoagulant	35 (22)	8 (14)	0.196[b]
ACE inhibitors /ARB	78 (49.1)	24 (42.1)	0.367[b]
B-blockers	86 (54.1)	31 (54.4)	0.969[b]
Ivabradine	24 (15.1)	12 (21.1)	0.300[b]
Ca-channel blockers	10 (6.3)	3 (5.3)	0.780[b]
ARNI	27 (17)	1 (1.8)	0.003[b]
Statin	94 (59.1)	33 (57.9)	0.872[b]
Digoxin	34 (21.4)	10 (17.5)	0.537[b]

[a]: Independent sample t test, [b]: Chi-square test, [c]: Mann-Whitney U test, GLIM: Global Leadership Initiative on Malnutrition, ARNI: Angiotensin receptor / neprilysin inhibitor, ACE: Angiotensin-converting-enzyme, ARB: Angiotensin receptor blocker, BMI: Body mass Index, COPD: Chronic obstructive pulmonary disease. GFR: Glomerular filtration rate, NYHA: New York Heart Association.

Table-2: Comparison of 1-year outcomes with variables.

	Survivors n (%)	The dead n (%)	*p*-value
GLIM malnutrition (+)	37 (20.7)	20 (54.1)	<0.001[b]
Mean PNI	39.01 ± 5.53	37.86±5.87	0.256[a]
Mean CONUT	1.34 ± 1.48	2.29±1.56	0.001[a]
Coronary artery disease	142 (79.3)	28 (75.7)	0.621[b]
Total length of hospital stay, day	4 (0–24)	10 (0–25)	<0.001[b]
Number of hospitalizations	1 (0–8)	2 (0–7)	<0.001[b]
Mean LVEF, %	30.74 ± 5.45	29.83±4.98	0.353[a]
Mean Age (year)	66.86 ± 5.72	68.24±4.85	0.174[a]
Mean GFR (mL/min/1.73 m^2)	56.98 ± 17.07	55.22±15.17	0.564[a]
Diabetes Mellitus	28 (15.6)	11 (29.7)	0.043[a]
Hypertension	47 (26.3)	10 (27)	0.923[b]
Mean C-reactive protein (mg/L)	21.29 ± 8.05	21.67±7.62	0.791[a]
Brain natriuretic peptide (pg/mL K)	430 (70–3800)	450 (146–1500)	0.857[b]
Low-density lipoprotein, mg/dL	99.18±30.85	96.81±29.67	0.669[a]
Triglyceride, mg/dL	151.27±77.12	160.49±64.39	0.498[a]
Platelet (10^3/μL)	296.12±92.38	303.43±98.48	0.666[a]
Neutrophil (10^3/μL)	7.91±2.20	8.83±2.69	0.027[b]
Lymphocyte (10^3/μL)	2.88±0.81	2.58±0.72	0.040[a]

[a]: Independent sample test, [b]: Mann-Whitney U test, CONUT: Controlling nutritional status, GLIM: Global Leadership Initiative on Malnutrition, GFR: Glomerular filtration rate, LVEF: Left ventricular ejection fraction, PNI: Prognostic nutritional index.

Table-3: Binary regression analysis.

Predictor	B	*p*-value	OR	95% CI
GLIM malnutrition	1.385	0.007	3.994	1.470–10.850
Length of hospital stay (day)	0.122	<0.001	1.13	1.058–1.206
Neutrophils count (10^3/µL)	0.252	0.005	1.287	1.081–1.532

GL:IM: Global Leadership Initiative on Malnutrition, OR: Odds ratio, CII Confidence interval.

When the variables were compared between the death and survivor groups, the presence of GLIM-defined malnutrition (p<0.001) and higher CONUT scores (p=0.001) were more common in the death group (Table 2). A survival curve was generated according to the presence of GLIM malnutrition (Figure).

GLIM malnutrition, LOS and increased neutrophils were identified as independent predictors of death (Table 3).

Discussion

The current study investigated the evaluation of malnutrition's predictive potential and significance regarding overall mortality related to HF with low LVEF. Malnutrition was observed more frequently in those with chronic diseases. a high rate of malnutrition was found in patients with COPD. The systemic effects of COPD include systemic inflammation, nutritional abnormalities, skeletal dysfunction, and other potential systemic effects (cardiovascular, nervous and skeletal systems). CAD incidence was higher in the non-malnourished group. This was probably related to factors such as the patient's smoking habits and exposure to smoke, hypercholesterolaemia, uncontrolled diabetes, obesity, unhealthy diet, sedentary lifestyle, and excessive stress. These non-ischaemic heart failure patients had higher malnutrition. ARNI is a new-generation drug used in HF. Neprilysin, which is included in ARNI, is a neutral endopeptidase that mediates the degradation of endogenous vasoactive peptides, including natriuretic peptides, bradykinin, adrenomedullin, glucagon, and vasoactive intestinal peptides, thereby reducing their levels. Neprilysin is found in various tissues, but is most abundant in the kidneys. The purpose of neprilysin inhibition in the treatment of HF is to prevent the reduction in levels of these substances, which results in water and sodium retention, vasoconstriction and inappropriate remodelling due to neurohormonal overactivation.[12] Malnutrition was found to be less in those using this drug. This was perhaps due to ARNI causing an increase in atrial natriuretic peptide and urine levels, while still having a positive haemodynamic effect. The rate of mortality was high at the CONUT and GLIM malnutrition evaluations. A study conducted in Spain, which aimed at determining the effectiveness of using body mass index (BMI) as a means of describing the nutritional condition of patients with HF, concluded that BMI was not an accurate indicator of the actual nutritional status in HF patients.[13] Malnutrition in individuals with HF is linked to muscle, fat, and bone mass depletion. This condition can arise from reduced nutrient intake, heightened nutrient loss, an elevated metabolic rate, and disrupted cytokine function, which involves factors like TNF-α, cortisol, epinephrine, renin and aldosterone. The current study did not find a significant difference between malnutrition and healthy groups regarding BMI. The study evaluated the patients with HF using complicated scales and cytokine dysfunction. Medications employed in managing HF, such as ACE inhibitors, can potentially prevent malnutrition and the development of sarcopenia in HF patients.[14] The patients with HF were treated with ACE inhibitors, meaning this medication agent could thwart malnutrition because ARNI, was higher in the no-malnutrition group. In a prospective study using the CONUT test, it was observed that an increase in CONUT score was associated with increased mortality in patients classified according to the criteria established by the American College of Cardiology (ACC) and the European Society of Cardiology (ESC) in the Stage-B group.[15] The current study also observed that the CONUT scores were high among individuals in the death group. Serum albumin is employed to evaluate protein malnutrition independently of calorie malnutrition, where serum albumin levels decrease without affecting anthropometric measurements.[16] Weidenhammar et al. found that assessment using indicators like PNI, Geriatric Nutritional Risk Index (GNRI) and CONUT revealed a prevalent occurrence of malnutrition among HF patients. This malnutrition correlates with increased mortality rates, regardless of the HF type, standing independently from traditional influencing factors and even N-terminal pro-B-type natriuretic peptide (NTproBNP). Given their added predictive significance, incorporating nutritional assessments into standard evaluations could help pinpoint patients at high risk.[17] Higher CONUT scores correlate with higher rates of HF-related hospitalisations and mortality in elderly individuals with cardiovascular disease.[18] Additionally, findings suggest that PNI, GNRI and CONUT scores independently predict cardiovascular events, with the CONUT score demonstrating relatively superior predictive capabilities compared to PNI and GNRI. Patients who had cardiovascular events displayed worse nutritional status, higher CONUT scores, lower PNI scores, and decreased GNRI scores compared to those who did not experience cardiovascular events.[19] Similarly, the current study found that a higher CONUT score and higher percent of GLIM malnutrition (+) were significant regarding overall mortality related to HF with low EF. Yoshihisa et al. reported that in HF patients who were malnourished, increased mortality rates were observed alongside elevated levels of

BNP, troponin I, C-reactive protein (CRP), TNF-α, adiponectin, right-sided volume and pressure overload, and reduced exercise capacity. However, they found that in predicting mortality among HF patients, PNI and GNRI outperformed the CONUT score.[20] CRP has been recommended for detecting cardiovascular risk: CRP levels of <1mg/L, 1-3mg/L, and >3mg/L indicate low, moderate, and high-risk groups, respectively.[21] The current study observed a significant increase in CRP, but did not find a difference between the death and survivor groups. This may have been due to the use of neprilysin which has an anti-inflammatory effect on patients.[22] The current study evaluated serum albumin as part of CONUT and PNI scales, but the utilisation of serum albumin comes with certain constraints, including its susceptibility to variation due to non-nutritional factors like, hydration status (excessive hydration can overestimate, while dehydration can underestimate serum albumin levels). Additionally, albumin is a negative acute-phase reactant, so conditions involving inflammation, infection and malignancies can also decrease serum levels.

Functional status and physical performance, in addition to nutritional condition, are key prognostic indicators in individuals with HF and reduced EF. In this patient group, malnutrition and sarcopenia are frequently observed and are closely linked with diminished exercise capacity and increased mortality risk. Evidence suggests that physiotherapy — especially programmes involving aerobic and resistance exercises — can significantly enhance muscular strength, physical function and overall quality of life in such patients. When physiotherapy is implemented alongside appropriate nutritional interventions, it may counteract the harmful consequences of both malnutrition and sarcopenia, thereby improving patient outcomes. Consequently, an integrated care model that combines nutritional and physiotherapeutic approaches is crucial for optimal management of malnourished HF patients with low EF.[23]

A 2025 retrospective cohort study found that malnutrition was a significant predictor of mortality among 357 patients admitted to the intensive care unit (ICU) for acute HF. Using the Nurses' Nutritional Assessment (NNA) and Nutritional Risk Index (NRI), the study identified malnutrition as an independent risk factor for death in this patient population.[24] Tang et al. in 2025 developed a nomogram model to estimate the likelihood of malnutrition in elderly HF patients based on the GLIM criteria. The model incorporates variables such as BMI, handgrip strength, fat-free mass (FFM), mid-upper arm circumference (MUAC), and serum albumin levels to predict malnutrition with high accuracy.[25] While the earlier study focussed on identifying patients at risk for malnutrition, the current research aimed evaluating malnutrition as a potential predictor of mortality in HF with reduced EF.

Li et al. in 2021 conducted a meta-analysis demonstrating that malnutrition assessed by CONUT score was a strong predictor of all-cause mortality in HF patients. The study found that malnourished patients had a mortality risk 1.92 times higher than those with normal nutritional status.[26]

The current study's limitations include the inability to control for sociodemographic parameters of the participants, the absence of anthropometric measurements, the need for further evaluation of other nutritional parameters, body density measurement using body plethysmography, assessment of vitamins and trace elements, evaluation of other acute-phase reactants, such as CRP and albumin, assessment of concomitant organ dysfunctions, and the requirement for additional investigations. Further research is required to validate the current findings.

A specific physical therapy protocol tailored to HF patients should be established to prevent sarcopenia, ensure haemodynamic integrity, increase exercise capacity, and reduce proinflammatory cytokines. Malnutrition and sarcopenia frequently affect individuals with HF having low EF, leading to impaired physical function and increased mortality. The integration of structured physiotherapy and nutritional support can enhance physical capacity and improve clinical outcomes. Managing these patients effectively requires a multidisciplinary treatment approach.

Conclusion

Malnutrition was found to be common among HF outpatients, and could significantly help identify those at increased mortality risk.

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References

1. Bonilla-Palomas JL, Gámez-López AL, Anguita-Sánchez MP, Castillo-Domínguez JC, García-Fuertes D, Crespin-Crespin M, et al. Impact of malnutrition on long-term mortality in hospitalized patients with heart failure. Rev Esp Cardiol 2011;64:752-8. doi: 10.1016/j.recesp.2011.03.009.
2. Lourenço P, Silva S, Friões F, Alvelos M, Amorim M, Couto M, et al. Low prealbumin is strongly associated with adverse outcome in heart failure. Heart 2014;100:1780-5. doi: 10.1136/heartjnl-2014-305747.
3. Cederholm T, Jensen GL, Correia MITD, Gonzalez MC, Fukushima R, Higashiguchi T, et al. GLIM criteria for the diagnosis of malnutrition - A consensus report from the global clinical nutrition community. Clin Nutr 2019;38:1-9. doi: 10.1016/j.clnu.2018.08.002.

4. Ignacio de Ulíbarri J, González-Madroño A, de Villar NG, González P, González B, Mancha A, et al. CONUT: a tool for controlling nutritional status. First validation in a hospital population. Nutr Hosp 2005;20:38-45.
5. Alvares-da-Silva MR, Reverbel da Silveira T. Comparison between handgrip strength, subjective global assessment, and prognostic nutritional index in assessing malnutrition and predicting clinical outcome in cirrhotic outpatients. Nutrition 2005;21:113-7. doi: 10.1016/j.nut.2004.02.002.
6. UCLA: Statistical Consulting Group. G*Power: Statistical power analyses, Version 3.1.9.7. [Online] 2021 [Cited 2022 November 09]. Available from URL: https://stats.oarc.ucla.edu/other/gpower/
7. Cederholm T, Jensen GL, Correia MITD, Gonzalez MC, Fukushima R, Higashiguchi T, et al. GLIM criteria for the diagnosis of malnutrition - A consensus report from the global clinical nutrition community. J Cachexia Sarcopenia Muscle 2019;10:207-17. doi: 10.1002/jcsm.12383.
8. Sze S, Zhang J, Pellicori P, Morgan D, Hoye A, Clark AL. Prognostic value of simple frailty and malnutrition screening tools in patients with acute heart failure due to left ventricular systolic dysfunction. Clin Res Cardiol 2017;106:533-41. doi: 10.1007/s00392-017-1082-5.
9. Kinugawa S, Fukushima A. Malnutrition in Heart Failure: Important But Undervalued Issue. JACC Heart Fail 2018;6:487-8. doi: 10.1016/j.jchf.2018.03.014.
10. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1-39.e14. doi: 10.1016/j.echo.2014.10.003.
11. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989;2:358-67. doi: 10.1016/s0894-7317(89)80014-8.
12. McMurray JJ, Packer M, Desai AS, Gong J, Lefkowitz MP, Rizkala AR, et al. Dual angiotensin receptor and neprilysin inhibition as an alternative to angiotensin-converting enzyme inhibition in patients with chronic systolic heart failure: rationale for and design of the Prospective comparison of ARNI with ACEI to Determine Impact on Global Mortality and morbidity in Heart Failure trial (PARADIGM-HF). Eur J Heart Fail 2013;15:1062-73. doi: 10.1093/eurjhf/hft052.
13. Rao BNVR. Clinical examinations in cardiology, 1st ed. Bengaluru, India: Elsevier India; 2009.
14. Anker S. Imbalance of catabolic and anabolic pathways in chronic heart failure. Implication for treatment of cardiac cahexia. Scand J Nutr 2002;46:3–10. DOI: 10.1080/110264802753704064.
15. Nochioka K, Sakata Y, Takahashi J, Miyata S, Miura M, Takada T, et al. CHART-2 Investigators. Prognostic impact of nutritional status in asymptomatic patients with cardiac diseases: a report from the CHART-2 Study. Circ J 2013;77:2318-26. doi: 10.1253/circj.cj-13-0127.
16. Sardesai VM. Introduction to Clinical Nutrition, 2nd ed. Basel, Switzerland: Marcel Dekker; 2003.
17. Weidenhammer A, Prausmueller S, Spinka G, Goliasch G, Arfsten H, Pavo N, et al. Malnutrition in patients with chronic heart failure. Eur Heart J 2022;43:ehac544-1056. doi:.10.1093/eurheartj/ehac544.1056.
18. Nochioka K, Sakata Y, Takahashi J, Miyata S, Miura M, Takada T, et al. Prognostic impact of nutritional status in asymptomatic patients with cardiac diseases: a report from the CHART-2 Study. Circ J 2013;77:2318-26. doi: 10.1253/circj.cj-13-0127.
19. Narumi T, Arimoto T, Funayama A, Kadowaki S, Otaki Y, Nishiyama S, et al. Prognostic importance of objective nutritional indexes in patients with chronic heart failure. J Cardiol 2013;62:307-13. doi: 10.1016/j.jjcc.2013.05.007.
20. Yoshihisa A, Kanno Y, Watanabe S, Yokokawa T, Abe S, Miyata M, et al. Impact of nutritional indices on mortality in patients with heart failure. Open Heart 2018;5:e000730. doi: 10.1136/openhrt-2017-000730.
21. Şişman AR, Küme T, Akan P, Tuncel P. C-reactive protein: Clinical significance, developments in measurement methods, pre-analytical and analysed variability. Turk J Clin Biochem. 2007;5:33-41.
22. Dindaş F, Güngör H, Ekici M, Akokay P, Erhan F, Doğduş M, et al. Angiotensin receptor-neprilysin inhibition by sacubitril/valsartan attenuates doxorubicin-induced cardiotoxicity in a pretreatment mice model by interfering with oxidative stress, inflammation, and Caspase 3 apoptotic pathway. Anatol J Cardiol 2021;25:821-8. doi: 10.5152/AnatolJCardiol.2021.356.
23. Abizanda P, López MD, García VP, Estrella Jde D, da Silva González Á, Vilardell NB, et al. Effects of an Oral Nutritional Supplementation Plus Physical Exercise Intervention on the Physical Function, Nutritional Status, and Quality of Life in Frail Institutionalized Older Adults: The ACTIVNES Study. J Am Med Dir Assoc 2015;16:439.e9-439.e16. doi: 10.1016/j.jamda.2015.02.005.
24. Lee JE, Hwang SY. Poor Nutritional Status Predicts Mortality in Patients With Acute Heart Failure Admitted to the Cardiac Intensive Care Unit: A Retrospective Cohort Study. Asian Nurs Res (Korean Soc Nurs Sci) 2025;19:53-9. doi: 10.1016/j.anr.2024.12.002.
25. Tang X, Zhang C, Xiao F, Yang F, Zhu X, Gao Y. A predictive model based on the GLIM diagnosis for malnutrition in older adult heart failure patients. Front Nutr 2025;12:e1551483. doi: 10.3389/fnut.2025.1551483.
26. Li H, Zhou P, Zhao Y, Ni H, Luo X, Li J. Prediction of all-cause mortality with malnutrition assessed by controlling nutritional status score in patients with heart failure: a systematic review and meta-analysis. Public Health Nutr 2022;25:1799-806. doi: 10.1017/S1368980021002470.

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