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Geriatric Nutritional Risk Index and In-Hospital Mortality in Older Patients with Acute Myocardial Infarction Undergoing Percutaneous Coronary Intervention: A Retrospective Cohort Study

Anna Yuliana[1]*, Agussalim Bukhari[1], Andi Yasmin Syauki[1], Nurpudji Astuti Daud[1], Haerani Rasyid[1], Mardiana Madjid[1]

[1]Department of Clinical Nutrition, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia

ABSTRACT: Malnutrition may contribute to adverse outcomes after acute myocardial infarction (AMI), particularly in older patients, but evidence regarding the Geriatric Nutritional Risk Index (GNRI) and in-hospital mortality after percutaneous coronary intervention (PCI) remains limited. This retrospective cohort study evaluated the association between GNRI and in-hospital mortality among 119 patients aged ≥60 years with AMI who underwent PCI at a tertiary referral hospital in Indonesia between January 2023 and December 2025. GNRI was calculated from serum albumin and body weight relative to ideal body weight. The primary outcome was in-hospital all-cause mortality. Survivors and non-survivors were compared using appropriate nonparametric and categorical tests, and binary logistic regression was used to estimate the association between continuous GNRI and mortality. Sixteen patients (13.4%) died during hospitalization. Median GNRI was significantly lower among non-survivors than survivors [84.88 (76.70–94.93) vs 93.82 (86.37–96.79); p=0.029]. Major nutritional risk was present in 37.5% of non-survivors compared with 9.7% of survivors. Higher GNRI was associated with lower odds of in-hospital mortality in crude analysis (OR 0.904, 95% CI 0.844–0.968; p=0.004) and after adjustment for age and multivessel disease (aOR 0.911, 95% CI 0.849–0.977; p=0.009). Lower GNRI was associated with greater in-hospital mortality, supporting its potential utility as a readily available marker of nutritional vulnerability in older patients with AMI undergoing PCI.

1. INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of death worldwide [1]. Reperfusion strategies and percutaneous coronary intervention (PCI) have substantially advanced acute coronary care, yet outcomes remain worse in older adults, in whom multimorbidity, frailty, and age-related physiologic changes complicate recovery [2,3].

Nutritional status has emerged as an additional prognostic domain in AMI. Among patients aged ≥75 years discharged after successful primary PCI, low GNRI was independently associated with long-term all-cause mortality [4]. In AMI patients undergoing PCI, GNRI-defined malnutrition was associated with mortality within 1 month and beyond 1 month after PCI [1]. In the COREA-AMI registry, increasing nutritional risk by GNRI showed graded associations with major bleeding, all-cause death, and ischemic events during long-term follow-up [5].

The GNRI combines serum albumin with body weight relative to ideal weight and can be calculated from routinely available data [1]. Lower GNRI has been associated with long-term mortality in very old patients with ACS [6], one-year MACE after

PCI [7], and improved risk stratification when combined with systemic inflammatory indices or the GRACE score [8,9]. A 2025 meta-analysis of 24 studies including 35,002 older PCI patients found low GNRI associated with higher all-cause mortality (HR 2.50, 95% CI 2.08–3.00) [10].

Evidence specifically addressing in-hospital mortality among older AMI patients undergoing PCI remains less developed. Recent ICU-based data linked low GNRI to in-hospital mortality in critically ill AMI patients, but did not specifically address older PCI-treated patients [11]. We therefore evaluated the association between GNRI and in-hospital mortality among older patients with AMI undergoing PCI in Indonesia.

2. MATERIALS AND METHODS

2.1 Study Design and Participants

This single-center retrospective cohort study included older patients with acute myocardial infarction (AMI) who underwent percutaneous coronary intervention (PCI) at Dr. Wahidin Sudirohusodo General Hospital, a tertiary referral hospital in Makassar, Indonesia. Medical records corresponding to index hospitalizations between January 2023 and December 2025 were reviewed. The study evaluated the association between the Geriatric Nutritional Risk Index (GNRI) and in-hospital mortality.

Patients were eligible if they were aged ≥60 years, had a documented diagnosis of AMI, underwent PCI during the index hospitalization, had sufficient anthropometric and laboratory data to calculate GNRI, and had complete information on hospital disposition. All patients meeting these eligibility criteria during the study period were included using total sampling. Patients with severe acute infection or clinical conditions expected to substantially interfere with nutritional assessment or interpretation of mortality, including cirrhosis and stage 5 chronic kidney disease, were excluded.

Missing data were handled using a complete-case approach. Records lacking body weight, height, serum albumin, or in-hospital vital status, which were required for calculation of GNRI or ascertainment of the primary outcome, were excluded before analysis. No statistical imputation of missing values was performed. The final analytic cohort comprised 119 patients.

2.2 Data Collection and Clinical Variables

Data were retrospectively extracted from medical records using a structured data collection form. Variables included age, sex, body weight, height, body mass index (BMI), comorbidity status, coronary artery involvement, serum albumin, differential blood counts, neutrophil-to-lymphocyte ratio (NLR), PCI-related information, and hospital disposition.

Coronary involvement was classified as single-vessel or multivessel disease according to the documented coronary angiography report. NLR was calculated from neutrophil and lymphocyte measurements recorded in the differential blood count. Serum albumin obtained during the first 24–48 hours of hospitalization was used for GNRI calculation. Laboratory measurements were obtained from the hospital laboratory, and extracted data were checked for completeness and consistency before analysis.

2.3 Geriatric Nutritional Risk Index and Outcome Assessment

GNRI was calculated using the established equation: GNRI = 14.89 × serum albumin (g/dL) + 41.7 × (actual body weight/ideal body weight) [1,12].

Ideal body weight was estimated using a sex-specific modified Broca index: (height [cm] − 100) × 0.90 for men and (height [cm] − 100) × 0.85 for women [13]. When actual body weight exceeded ideal body weight, the actual-to-ideal body weight ratio was set to 1 [12,13].

GNRI was analyzed primarily as a continuous variable to preserve information across the full range of scores. For descriptive analyses, nutritional risk was additionally classified as major risk (GNRI <82), moderate risk (82 to <92), low risk (92 to ≤98), or no nutritional risk (>98), according to the original GNRI risk classification [12]. These categories were used for descriptive and group-comparison purposes, whereas GNRI was retained as a continuous variable in the primary regression analysis to avoid loss of information and statistical power.

The primary outcome was in-hospital all-cause mortality, defined as death occurring during the index hospitalization. Patients discharged alive constituted the comparison group. For regression analyses, mortality was coded as 1 for in-hospital death and 0 for discharge alive.

2.4 Statistical Analysis

The distribution of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables were presented as median (Q1–Q3) and compared between survivors and non-survivors using the Mann–Whitney U test. Categorical variables were presented as frequencies and percentages. Fisher's exact test was used for binary categorical comparisons, whereas the Fisher–Freeman–Halton exact test was used to compare the four GNRI nutritional-risk categories between survivors and non-survivors because of sparse expected cell counts.

The association between continuous GNRI and in-hospital mortality was evaluated using binary logistic regression, with in-hospital death coded as 1 and survival to discharge as 0. The crude model estimated the odds ratio (OR) and 95% confidence interval (CI) for in-hospital mortality per one-point increase in GNRI. Because only 16 in-hospital deaths occurred, the multivariable model was deliberately kept parsimonious to reduce the risk of model overfitting. Age and multivessel disease were included as clinically relevant potential confounders representing baseline age-related risk and the anatomical burden of coronary artery disease, respectively. The number of covariates was intentionally restricted in relation to the limited number of outcome events. Serum albumin was not entered as a separate covariate because it is a direct component of GNRI. BMI was likewise not included because it is derived from the same underlying anthropometric measurements incorporated into the GNRI calculation, thereby avoiding redundant adjustment and potential overadjustment.

Results were reported as ORs or adjusted ORs (aORs) with 95% CIs. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. All statistical tests were two-sided, and $P<0.05$ was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics version 30.0 (IBM Corp., Armonk, NY, USA).

3. RESULTS AND DISCUSSION

3.1 Study Population and Baseline Characteristics

A total of 119 older patients with acute myocardial infarction undergoing percutaneous coronary intervention were included in the analysis. Of these, 103 patients (86.6%) survived to hospital discharge and 16 (13.4%) died during hospitalization. Non-survivors had significantly lower GNRI values than survivors [84.88 (76.70–94.93) vs 93.82 (86.37–96.79); p=0.029]. The distribution of GNRI nutritional-risk categories also differed between groups (p=0.049), with major nutritional risk observed in 37.5% of non-survivors compared with 9.7% of survivors. Non-survivors also had higher NLR [6.47 (6.05–11.95) vs 4.84 (3.44–7.81); p=0.026] and lower serum albumin [3.00 (2.73–3.58) vs 3.50 (3.20–3.80) g/dL; p=0.017]. Age, sex, BMI, multivessel disease, and comorbidity status were not significantly different between groups.

Table 1. Baseline Characteristics According to In-Hospital Survival Status

Characteristic	Overall (n=119)	Survivors (n=103)	Non-survivors (n=16)	*p*-value
Age, years	67.00 (64.00–70.00)	67.00 (64.00–70.00)	70.00 (64.00–73.00)	0.352[a]
Male sex, n (%)	87 (73.1)	74 (71.8)	13 (81.3)	0.553[b]
BMI, kg/m²	21.49 (19.53–23.44)	21.49 (19.53–23.53)	21.23 (18.24–22.58)	0.405[a]
GNRI	93.12 (84.90–96.79)	93.82 (86.37–96.79)	84.88 (76.70–94.93)	0.029[a]*
GNRI nutritional-risk category, n (%)				0.049[c]*
No nutritional risk (>98)	23 (19.3)	21 (20.4)	2 (12.5)	
Low nutritional risk (92–98)	48 (40.3)	43 (41.7)	5 (31.3)	
Moderate nutritional risk (82–<92)	32 (26.9)	29 (28.2)	3 (18.8)	
Major nutritional risk (<82)	16 (13.4)	10 (9.7)	6 (37.5)	
NLR	5.42 (3.48–7.91)	4.84 (3.44–7.81)	6.47 (6.05–11.95)	0.026[a]*

Serum albumin, g/dL	3.50 (3.10–3.70)	3.50 (3.20–3.80)	3.00 (2.73–3.58)	0.017[a]*
Multivessel disease, n (%)	85 (71.4)	77 (74.8)	8 (50.0)	0.070[b]
Comorbidity present, n (%)	109 (91.6)	95 (92.2)	14 (87.5)	0.623[b]

Note: Continuous variables are presented as median (Q1–Q3), and categorical variables as n (%). [a]Mann–Whitney U test; [b]Fisher's exact test; [c]Fisher–Freeman–Halton exact test. *Statistically significant at $p<0.05$. BMI, body mass index; GNRI, Geriatric Nutritional Risk Index; NLR, neutrophil-to-lymphocyte ratio.

3.2 Association of GNRI With In-Hospital Mortality

The distribution of GNRI according to in-hospital survival status is shown in Figure 1. Non-survivors had substantially lower median GNRI values and a wider distribution toward lower values compared with survivors.

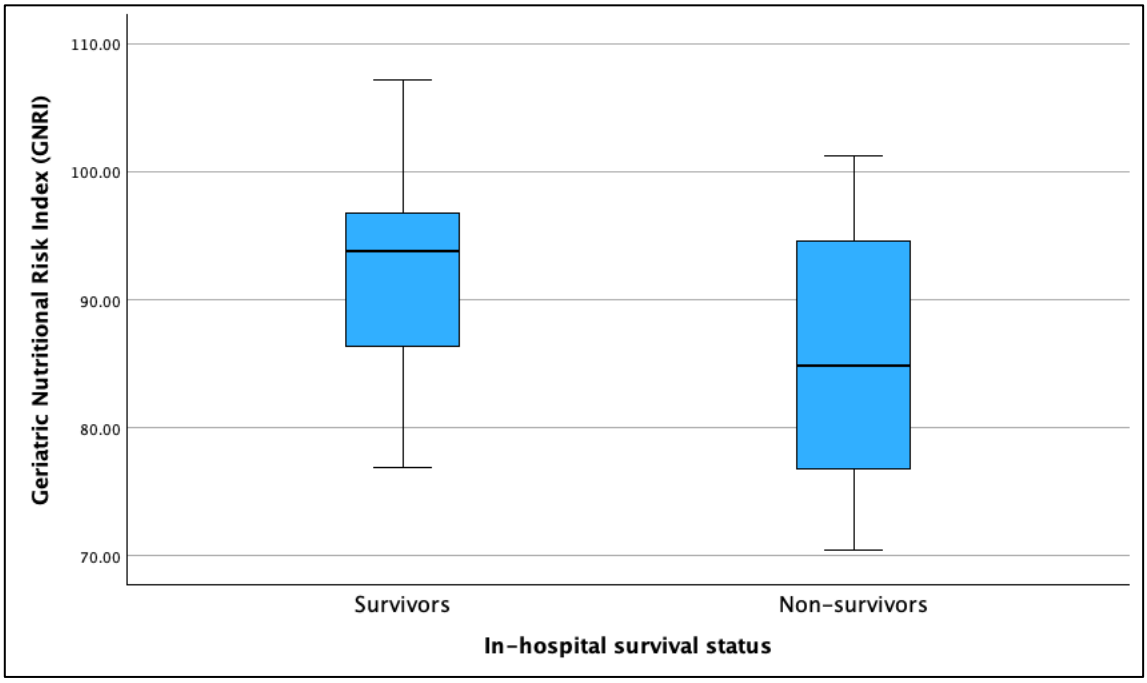

Figure 1. Distribution of the Geriatric Nutritional Risk Index According to In-Hospital Survival Status. Boxes represent the interquartile range, with the horizontal line indicating the median. GNRI was lower among non-survivors than survivors [84.88 (76.70–94.93) vs 93.82 (86.37–96.79); Mann–Whitney U test, $p=0.029$].

Binary logistic regression showed that higher GNRI was associated with lower odds of in-hospital mortality. In the crude analysis, each one-point increase in GNRI was associated with lower odds of in-hospital death (OR 0.904, 95% CI 0.844–0.968; $p=0.004$). After adjustment for age and multivessel disease, GNRI remained significantly associated with mortality (aOR 0.911, 95% CI 0.849–0.977; $p=0.009$). Age and multivessel disease were not statistically significant in the adjusted model. The adjusted model was significant overall ($p=0.006$), with no evidence of poor calibration based on the Hosmer–Lemeshow test ($p=0.393$).

Table 2. Association of GNRI With In-Hospital Mortality

Panel A. Crude Model		
Variable	**OR (95% CI)**	***p*-value**
GNRI, per 1-point increase	0.904 (0.844–0.968)	0.004*
Panel B. Adjusted Model		
GNRI, per 1-point increase	0.911 (0.849–0.977)	0.009*

Age, per year	1.057 (0.947–1.181)	0.324
Multivessel disease†	0.396 (0.127–1.230)	0.109

Note: Binary logistic regression was performed with in-hospital death coded as 1 and survival to discharge as 0. Panel A presents the crude association between GNRI and mortality. Panel B was adjusted for age and multivessel disease. †Reference category: single-vessel disease. OR, odds ratio; aOR, adjusted odds ratio; CI, confidence interval; GNRI, Geriatric Nutritional Risk Index. *Statistically significant at $p<0.05$.

3.3 Discussion

In this cohort of older patients with acute myocardial infarction undergoing PCI, lower GNRI was associated with in-hospital mortality. Non-survivors had lower GNRI values than survivors, and each one-point increase in GNRI was associated with 8.9% lower odds of in-hospital death after adjustment for age and multivessel disease (aOR 0.911, 95% CI 0.849–0.977). The direction of this association is consistent with previous AMI studies linking lower GNRI to mortality after PCI and during critical illness [1,11]. Particularly relevant to our setting, Hosseini et al. (2026) recently showed that worsening nutritional impairment was associated with in-hospital mortality among patients aged ≥65 years with STEMI undergoing primary PCI, although nutritional status was assessed using CONUT rather than GNRI [14].

The association observed here is also consistent with longer-term evidence from coronary populations. Among patients aged ≥75 years discharged after successful primary PCI for AMI, lower GNRI was associated with higher long-term mortality [4]. In 10,161 patients with AMI undergoing PCI, Lim et al. (2023) reported progressively higher risks of all-cause death, major bleeding, and ischemic events with increasing GNRI-defined nutritional risk [5]. An updated meta-analysis of 24 studies involving 35,002 older PCI patients similarly found that low GNRI was associated with greater all-cause mortality (HR 2.50, 95% CI 2.08–3.00) [10]. Broader evidence from 81,361 patients with coronary artery disease also supports an association between malnutrition and both mortality and major adverse cardiovascular events [15].

Evidence focused on early outcomes is increasingly consistent with this pattern. Tang et al. (2025) reported higher in-hospital and 30-day mortality among 5,506 critically ill patients with AMI who had lower GNRI [11]. In another AMI cohort, higher GNRI was associated with lower 30-day mortality [16], while Xie et al. (2024) demonstrated an inverse association between continuous GNRI and 28-day mortality among patients undergoing coronary revascularization [17]. These populations are not directly interchangeable with older PCI-treated AMI patients, but their findings support the relevance of nutritional vulnerability during the early phase of acute cardiovascular illness rather than only during long-term follow-up [16,17].

Our categorical findings warrant more cautious interpretation. Mortality was highest in the major-risk category, in which 6 of 16 patients (37.5%) died, but the intermediate categories did not show a clear monotonic gradient. Given only 16 deaths and relatively small category-specific counts, the continuous GNRI analysis provides a more statistically stable representation of the association in this cohort. This approach is supported by external studies demonstrating inverse associations between GNRI modeled continuously and short-term mortality, although their populations and GNRI distributions differed from ours [16,17].

The biological interpretation of GNRI also requires nuance. GNRI combines serum albumin with body weight relative to ideal weight, but neither component should be interpreted as representing a single biological pathway. In particular, serum albumin decreases during inflammatory illness and should not be regarded as a direct marker of protein-energy malnutrition [18]. Thus, lower GNRI during AMI may reflect a mixture of nutritional vulnerability and acute systemic illness. The higher NLR and lower albumin observed among non-survivors in our cohort are compatible with concurrent inflammatory and nutrition-risk signals rather than establishing a causal mechanism. Nutritional impairment and frailty may also coexist while retaining partly nonredundant prognostic information in older patients with MI [14,19].

The clinical appeal of GNRI lies in its simplicity and availability from routinely collected data. Its potential role is more appropriately considered complementary to established cardiovascular risk assessment than as a replacement for it. Adding GNRI to the GRACE score improved one-year risk discrimination in older patients with NSTEMI after PCI, while combining GNRI with systemic inflammatory indices has also improved risk stratification in older ACS populations [8,9]. A meta-analysis of 37,303 patients further showed that malnutrition was associated with higher mortality across ACS populations and nutritional assessment methods [20]. Importantly, nutritional risk is not synonymous with low BMI: adverse nutritional profiles can occur across BMI categories in ACS cohorts [21,22]. GNRI may therefore help identify patients requiring more detailed nutritional

and geriatric assessment, but these observational data do not demonstrate that GNRI-guided nutritional intervention reduces mortality.

Several limitations should be considered. First, the retrospective single-center design limits causal inference and may restrict the generalizability of the findings to other hospitals and patient populations. Second, the relatively modest sample size and particularly the small number of in-hospital deaths limited statistical precision and constrained the number of variables that could be included in the multivariable model. Consequently, residual confounding from clinical factors not incorporated into the adjusted model cannot be excluded. Third, GNRI was assessed during the acute phase of illness, when serum albumin may partly reflect inflammation and acute disease severity rather than nutritional depletion alone [18]. Frailty was also not incorporated into the present analysis despite evidence that frailty and nutritional impairment may provide complementary prognostic information in older patients with myocardial infarction [19]. Finally, ideal-body-weight estimation varies among published GNRI studies, which should be considered when comparing absolute GNRI values and thresholds across cohorts [1,17]. These findings should therefore be regarded as observational evidence requiring confirmation in larger prospective multicenter cohorts before GNRI can be recommended as a routine component of AMI risk-stratification protocols.

Overall, lower GNRI was associated with greater in-hospital mortality among older patients with AMI undergoing PCI, and the association remained after adjustment for age and multivessel disease. These findings support GNRI as an accessible marker of nutritional vulnerability that may complement conventional early risk assessment, while larger prospective multicenter studies are required to determine its incremental prognostic value and whether GNRI-informed clinical strategies can improve outcomes [9,10,20].

4. CONCLUSION

Lower Geriatric Nutritional Risk Index (GNRI) was associated with greater in-hospital mortality among older patients with acute myocardial infarction undergoing percutaneous coronary intervention. Non-survivors had lower GNRI values than survivors, and the association remained significant after adjustment for age and multivessel disease. These findings suggest that GNRI may provide a simple and readily available marker of nutritional vulnerability that complements early clinical risk assessment in this population. However, given the retrospective single-center design and the limited number of in-hospital deaths, the findings should be interpreted as evidence of association rather than as validation of GNRI as a stand-alone prognostic tool. Larger prospective multicenter studies are warranted to confirm these findings, evaluate the incremental prognostic value of GNRI beyond established cardiovascular risk factors, and determine whether GNRI-informed nutritional or geriatric assessment can improve clinical outcomes.

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AUTHOR CONTRIBUTIONS

Anna Yuliana: Conceptualization, Investigation, Data Curation, Formal Analysis, and Writing – Original Draft.

Agussalim Bukhari: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

Andi Yasmin Syauki: Methodology, Supervision, Validation, and Writing – Review & Editing.

Nurpudji Astuti Daud: Supervision, Validation, and Writing – Review & Editing.

Haerani Rasyid: Supervision, Validation, and Writing – Review & Editing.

Mardiana Madjid: Supervision, Validation, and Writing – Review & Editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the integrity and accuracy of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICS STATEMENT

The study was approved by the Research Ethics Committee of Hasanuddin University in collaboration with Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia (approval no. 540/UN4.6.4.5.31/PP36/2026; protocol no. UH26030415; April 21, 2026). Data access and extraction were performed only after ethical approval had been obtained. The study involved retrospective medical-record review without direct patient intervention, and patient identifiers were excluded from the analytic dataset. The study was conducted in accordance with the principles of the Declaration of Helsinki.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

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