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Original article

Impact of Geriatric Nutritional Risk Index on cardiovascular outcomes in patients with stable coronary artery disease

Ayako Kunimura (MD)[a,b,*], Hideki Ishii (MD, PhD)[a], Tadayuki Uetani (MD, PhD)[b], Toshijirou Aoki (MD)[a], Kazuhiro Harada (MD)[a], Kenshi Hirayama (MD)[a], Yosuke Negishi (MD)[a], Yohei Shibata (MD)[a], Takuya Sumi (MD)[a], Kazuhiro Kawashima (MD)[a], Yosuke Tatami (MD)[a], Toshiki Kawamiya (MD)[a], Dai Yamamoto (MD)[a], Susumu Suzuki (MD)[a], Tetsuya Amano (MD, PhD, FJCC)[c], Toyoaki Murohara (MD, PhD, FJCC)[a]

[a] Department of Cardiology, Nagoya University Graduate School of Medicine, Nagoya, Japan
[b] Department of Cardiology, Chubu Rosai Hospital, Nagoya, Japan
[c] Department of Cardiology, Aichi-Medical University, Nagoya, Japan

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ABSTRACT

Background: The association between malnutrition and cardiovascular prognosis in patients with stable coronary artery disease remains unclear. The aim of this study was to evaluate the association between Geriatric Nutritional Risk Index (GNRI), a simple tool to assess nutritional risk, and long-term outcomes after elective percutaneous coronary intervention (PCI).

Methods: This study consisted of 802 patients (age, 70 ± 10 years, male, 69%) who underwent elective PCI. GNRI was calculated at baseline as follows: GNRI = [14.89 × serum albumin (g/dl) + [41.7 × (body weight/body weight at body mass index of 22)]]. Patients were then divided into three groups as previously reported: GNRI <92, 92 to ≤98, and >98. The endpoint of this study was the composite of cardiac death or non-fatal myocardial infarction.

Results: During a median follow-up period of 1568 days, 56 cardiac events occurred. Using Kaplan–Meier analysis, the 4-year event-free rates were found to be 79% for GNRI <92, 90% for GNRI 92 to ≤98, and 97% for GNRI >98 (log-rank test $p < 0.001$). GNRI <92 and GNRI 92 to ≤98 showed 6.76-fold [95% confidence interval (CI) 3.13–14.56, $p < 0.001$] and 3.03-fold (HR 3.03, 95%CI 1.36–6.78, $p = 0.007$) increase in the incidences of cardiac death or non-fatal myocardial infarction compared with GNRI >98 after adjusting for confounding factors.

Conclusion: GNRI significantly associated with cardiac events after elective PCI. Further studies should be performed to establish appropriate therapeutic strategies for this vulnerable patient group.

Introduction

Malnutrition has been identified as an independent predictor of an unfavorable prognosis in multiple patient groups, such as elderly patients [1,2], patients with end-stage renal disease [3,4], and those with chronic heart failure [5,6]. Lane et al. also demonstrated that malnutrition has been associated with the development of atherosclerosis and a higher incidence of cardiovascular mortality in elderly patients [7].

Previous clinical studies [8,9] have found that underweight patients were associated with a significantly higher incidence of cardiovascular events and mortality compared with normal-weight and obese patients after percutaneous coronary intervention (PCI). This relationship has often been said to be a reverse causation, as patients are often likely to be underweight because of malnutrition or cachexia. However, the association between malnutrition and the long-term cardiovascular outcomes following PCI remains unclear.

Geriatric Nutritional Risk Index (GNRI) is a simple tool to accurately assess a patient's risk of malnutrition-related complications using three objective parameters: body weight,

* Corresponding author at: Department of Cardiology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550, Japan. Fax: +81 52 744 2785.
E-mail address: akuni1127@med.nagoya-u.ac.jp (A. Kunimura).

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body height, and serum albumin [10,11]. Previous studies compared the validity of several nutritional tools and reported that GNRI was as useful as the others for the assessment of nutritional risk [12]. The aim of this study was to evaluate the predictive value of GNRI for poor cardiovascular outcomes in patients who underwent elective PCI.

Patients and methods

Study population

This observational study consisted of 802 consecutive patients who underwent successful elective PCI for de novo lesions at Chubu Rosai Hospital, Nagoya, Japan between January 2008 and December 2012. We excluded patients with active inflammatory disease or malignancies (16 patients), or who were lost to follow-up (5 patients). All patients had angina, documented myocardial ischemia or both. The ethics committee at Chubu Rosai Hospital approved this study, and all patients provided written informed consent. This study complies with the Declaration of Helsinki.

Current smoker was defined as current habit or discontinuation ≤1 year before PCI. Diabetes mellitus was defined as the use of anti-hyperglycemic medication, previous diagnosis of diabetes mellitus, or glycated hemoglobin ≥6.5% (National Glycohemoglobin Standardization Program). Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or current anti-hypertensive medication. Dyslipidemia was defined as low-density lipoprotein cholesterol ≥140 mg/dl, high-density lipoprotein cholesterol <40 mg/dl, triglycerides ≥150 mg/dl, or current lipid-lowering medication.

Geriatric Nutritional Risk Index (GNRI)

The patient's serum albumin level, body weight, and body height were measured before the PCI to create a baseline value. GNRI was calculated by modifying the Nutritional Risk Index for elderly patients [11] in a manner previously reported by Yamada et al. (Eq. (1)) [12]:

$$\mathrm{GNRI} = [14.89 \times \text{serum albumin (g/dl)}] + [41.7 \times (\text{body weight}/\text{ideal body weight})]. \quad (1)$$

The body weight/ideal body weight ratio defaulted to 1 when the patient's actual body weight exceeded their ideal body weight. The ideal body weight was defined as the value calculated from the patient's height and a body mass index of 22 [13–15], instead of the value calculated using the Lorentz formula from the original GNRI equation [11]. Patients were then divided into three groups based on previously published thresholds: GNRI <92, 92 to ≤98, and >98 [11,16].

Coronary angiography and PCI

Baseline angiography was performed by independent investigators who were not involved in the procedures and were blinded to patient outcomes. A computerized quantitative analysis system (QCA-CMS System, version 6.0.39.0; MEDIS, Leiden, The Netherlands) was used with a guide catheter for calibration. The operators in charge were blinded to the patient's GNRI, and decided on the PCI device and technique based on the findings from the angiography and the conventional intravascular ultrasound.

Clinical follow-up

Clinical follow-up data were obtained through admission and outpatient medical records or by telephone interview. All patient follow-up data were collected by April 30th, 2015. The endpoint of this study was the composite of cardiac death or non-fatal myocardial infarction. Events at the time of the index procedure and during the index hospitalization were not included. For patients who had multiple cardiac events during the study period, the time until the first event was used in our calculations. Cardiac death was defined as death resulting from an acute myocardial infarction, fatal arrhythmia, and progression of heart failure. A death from undetermined cause was not counted as a cardiac death. Myocardial infarction was defined when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia with detection of a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile upper reference limit and with at least one of the following: (1) symptoms of ischemia; (2) new or presumed new significant ST-segmented wave changes or new left bundle branch block; (3) development of pathological Q waves in the electrocardiogram; (3) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality; (4) identification of an intracoronary thrombus by angiography or autopsy [17]. These events were assessed by investigators, who are blinded to the subjects.

Statistical analyses

Normally and non-normally distributed continuous values were expressed as the mean ± standard deviation and the median (interquartile range), respectively. Categorical variables were expressed as numbers (proportion). We compared normally distributed continuous variables using an analysis of variance (ANOVA), and non-normally distributed variables (GNRI, C-reactive protein, triglycerides, and brain natriuretic peptide) using the Kruskal–Wallis test. Categorical variables were compared using Fisher's exact test or the Chi-squared test. Multivariate regression analysis was used to determine the factors that correlated with GNRI. Event-free survival was analyzed using Kaplan–Meier estimation with the log-rank test. The Cox proportional hazards model was used to estimate the contribution of GNRI to the accuracy of the prediction of cardiac events during the follow-up period. We considered age, male sex, statins, brain natriuretic peptide, and conventional coronary risk factors (current smoker, estimated glomerular filtration rate, diabetes mellitus, hypertension, and dyslipidemia) as candidate variables for inclusion in our multivariate analysis. The performance of our model in the prediction of cardiac events with or without GNRI was evaluated by calculating *c*-statistics. Improvements in predictive accuracy were determined by calculating the net reclassification improvement (NRI) and the integrated discrimination improvement (IDI). A p-value < 0.05 was considered statistically significant. Calculations were performed by blinded investigators using SPSS statistics version 18.0 (IBM, Armonk, NY, USA) and R 2.13.1 with PredictABEL and pROC packages (R Development Core Team 2011, Vienna, Austria).

Results

Baseline characteristics

Baseline characteristics are shown in Table 1. GNRI <92, 92 to ≤98, and >98 was measured in 136, 115, and 551 patients, respectively. Age, C-reactive protein, estimated glomerular filtration rate, brain natriuretic peptide, the prevalence of multiple vessel disease, the use of statins, and the use of β-blockers were significantly associated with GNRI. The prevalence of hypertension and dyslipidemia was significantly lower in GNRI <92, and GNRI <92 was associated with a lower systolic blood pressure and decreased low-density lipoprotein-cholesterol, high-density

Table 1
Baseline characteristics.

Variables	GNRI			
	<92 n = 136	92 to ≤98 n = 115	>98 n = 551	p-value
Age, years	75 ± 9	71 ± 9	69 ± 10	<0.001
Male, n (%)	87 (64.0)	77 (67.0)	389 (70.6)	0.3
Body mass index, kg/m^2	21.4 ± 3.8	22.9 ± 3.8	24.6 ± 3.3	<0.001
Serum albumin, g/dl	3.1 ± 0.4	3.7 ± 0.2	4.3 ± 0.3	<0.001
GNRI	86 (80–89)	95 (94–97)	106 (102–109)	<0.001
C-reactive protein, mg/l	0.20 (0.10–0.31)	0.13 (0.10–0.29)	0.10 (0.04–0.18)	<0.001
Current smoker, n (%)	38 (28.1)	32 (28.1)	168 (30.5)	0.8
eGFR, ml/min/1.73 m^2	52 ± 31	60 ± 23	65 ± 18	<0.001
Ejection fraction, %	60 ± 15	65 ± 13	69 ± 11	0.8
Diabetes mellitus, n (%)	76 (55.9)	56 (48.7)	285 (51.7)	0.5
Glycated hemoglobin, %	6.3 ± 1.4	6.3 ± 1.5	6.2 ± 1.1	0.5
Hypertension, n (%)	107 (78.7)	90 (78.3)	480 (87.1)	0.008
Systolic blood pressure, mmHg	133 ± 23	134 ± 23	140 ± 21	<0.001
Dyslipidemia, n (%)	84 (61.8)	88 (76.5)	418 (75.9)	0.003
LDL-cholesterol, mg/dl	95 ± 31	106 ± 32	117 ± 36	<0.001
HDL-cholesterol, mg/dl	41 ± 11	44 ± 14	49 ± 18	<0.001
Triglyceride, mg/dl	95 (66–128)	105 (69–147)	141 (101–188)	<0.001
BNP, pg/ml	262 (113–754)	123 (46–281)	54 (23–114)	<0.001
Multiple vessel disease, n (%)	81 (60.0)	60 (52.2)	233 (42.3)	<0.001
Previous PCI, n (%)	48 (35.3)	36 (31.3)	150 (27.2)	0.2
Previous CABG, n (%)	6 (4.4)	12 (10.4)	44 (8.0)	0.2
Medications				
Aspirin, n (%)	135 (99.3)	115 (100.0)	545 (99.1)	0.6
Thienopyridine derivatives, n (%)	126 (92.6)	109 (94.8)	527 (95.8)	0.3
Statins, n (%)	107 (78.7)	97 (84.3)	484 (88.0)	0.02
Calcium channel blocker, n (%)	55 (40.4)	46 (40.0)	253 (46.0)	0.3
β-Blockers, n (%)	69 (51.1)	53 (46.1)	211 (38.4)	0.02
ACE inhibitor or ARB, n (%)	98 (72.1)	76 (66.1)	337 (61.3)	0.06

Normally distributed continuous values are expressed as mean ± standard deviation. Non-normally distributed continuous values are expressed as median (interquartile range). Categorical values are expressed as number (percentage).
ACE, angiotensin-converting enzyme; ARB, angiotensin-II receptor blocker; BNP, brain natriuretic peptide; CABG, coronary artery bypass grafting; eGFR, estimated glomerular filtration rate; GNRI, geriatric nutritional risk index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PCI, percutaneous coronary intervention.

lipoprotein-cholesterol and triglyceride levels. Lesion and procedure characteristics are shown in Table 2. The prevalence of right coronary artery disease and the number of stents needed were significantly higher in GNRI 92 to ≤98. The prevalence of left anterior descending artery disease was significantly lower in GNRI 92 to ≤98. On multivariate regression analysis (Table 3), GNRI was independently correlated with age ($\beta = -0.22$, $p < 0.001$), C-reactive protein ($\beta = -0.23$, $p < 0.001$), estimated glomerular filtration rate ($\beta = 0.17$, $p < 0.001$), hypertension ($\beta = 0.15$, $p < 0.001$), and dyslipidemia ($\beta = 0.10$, $p = 0.003$).

Clinical outcomes

During follow-up (median: 1568 days), 56 events were documented. Twenty-eight events (20.6%), 12 events (10.4%), and 16 events (2.9%) were diagnosed in patients with a GNRI <92, 92 to ≤98, and >98, respectively ($p < 0.001$, Table 4). The incidence of cardiac death was significantly increased in GNRI <92. On the other hand, there was no significant difference in the incidence of non-fatal myocardial infarction among the groups (Table 4). In Kaplan–Meier analysis, the 4-year event-free cumulative rates were 79%,

Table 2
Lesion and procedure characteristics.

Variables	GNRI			
	<92 n = 136	92 to ≤98 n = 115	>98 n = 551	p-value
Lesion location				
Right coronary artery, n (%)	50 (36.8)	45 (39.1)	151 (27.4)	0.01
Left anterior descending artery, n (%)	60 (44.1)	42 (36.5)	284 (51.5)	0.008
Left circumflex artery, n (%)	34 (25.0)	28 (24.3)	118 (21.4)	0.6
Left main trunk, n (%)	4 (2.9)	2 (1.7)	24 (4.4)	0.4
Saphenous vein graft, n (%)	0 (0.0)	1 (0.9)	5 (0.9)	0.5
AHA/ACC type B2/C, n (%)	50 (36.8)	48 (41.7)	216 (39.2)	0.7
QCA analysis				
Reference diameter, mm	2.2 ± 0.5	2.2 ± 0.6	2.2 ± 0.6	0.7
Diameter stenosis, %	71.6 ± 13.9	71.2 ± 13.4	71.8 ± 12.7	0.9
Bare-metal stent, n (%)	28 (20.6)	20 (17.4)	87 (15.8)	0.4
Drug-eluting stent, n (%)	105 (77.2)	96 (83.5)	444 (80.6)	0.5
Balloon angioplasty, n (%)	9 (6.6)	4 (3.5)	24 (4.4)	0.4
Number of stents	1.4 ± 0.8	1.5 ± 0.8	1.3 ± 0.7	0.03

Normally distributed continuous values are expressed as mean ± standard deviation. Categorical values are expressed as number (percentage).
ACC, American College of Cardiology; AHA, American Heart Association; GNRI, Geriatric Nutritional Risk Index; QCA, quantitative coronary analysis.

Table 3
Relationship between GNRI and baseline variables determined with multivariate regression analysis.

	β	p-value
Age	−0.22	<0.001
Male	0.022	0.5
Log C-reactive protein	−0.23	<0.001
Current smoker	−0.038	0.3
eGFR	0.17	<0.001
Diabetes mellitus	−0.044	0.2
Hypertension	0.15	<0.001
Dyslipidemia	0.10	0.003

eGFR, estimated glomerular filtration rate; GNRI, Geriatric Nutritional Risk Index.

Table 4
The number (percentage) of events during the follow-up period.

Clinical events	GNRI			
	<92 $n=136$	92 to ≤98 $n=115$	>98 $n=551$	p-value
Total events, n (%)	28 (20.6)	12 (10.4)	16 (2.9)	<0.001
Cardiac death, n (%)	24 (17.6)	6 (5.2)	6 (1.1)	<0.001
Non-fatal myocardial infarction, n (%)	4 (2.9)	6 (5.2)	10 (1.8)	0.1

Values in parentheses indicate the number (percentage) of events and p-values were obtained by Chi-square test.
GNRI, Geriatric Nutritional Risk Index.

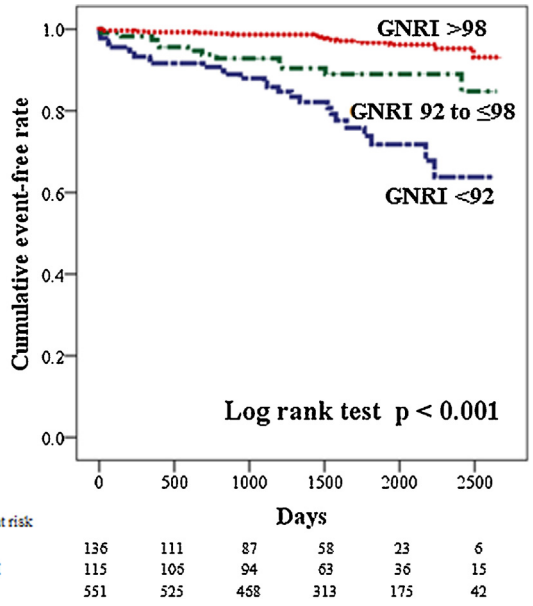

Fig. 1. Kaplan–Meier event-free survival curves based on GNRI. Event-free survival was significantly associated with GNRI, with the worst event-free survival curve for GNRI <92 (log-rank test $p < 0.001$). GNRI, geriatric nutritional risk index.

90%, and 97% for GNRI <92, 92 to ≤98, and >98, respectively (log-rank test: $p < 0.001$, Fig. 1). GNRI <92 and GNRI 92 to ≤98 showed 6.76-fold (95% confidence interval (CI) 3.13–14.56, $p < 0.001$) and 3.03-fold [hazard ratio (HR) 3.03, 95%CI 1.36–6.78, $p = 0.007$] increase in the incidences of cardiac death or non-fatal myocardial infarction compared with GNRI >98 after adjusting for confounding factors (Table 5). Adding GNRI to the established risk factors increased our algorithm's predictive accuracy (Table 6).

In a second analysis, we excluded 31 patients on maintenance hemodialysis and re-divided patients into three groups as previously mentioned. A Cox univariate analysis revealed that the HRs for cardiac death or non-fatal myocardial infarction were 8.89 (95% CI 4.63–17.07, $p < 0.001$) for GNRI <92 and 3.54 (95% CI 1.62–7.72, $p = 0.002$) for GNRI 92 to ≤98 compared with GNRI >98. After adjusting for age, male sex, statins, brain natriuretic peptide, and conventional coronary risk factors (current smoker, estimated glomerular filtration rate, diabetes mellitus, hypertension, and dyslipidemia), a Cox multivariate analysis found that the HRs for cardiac death or non-fatal myocardial infarction

Table 5
Predictive value of GNRI for cardiac death or non-fatal myocardial infarction after elective percutaneous coronary intervention by Cox analysis.

	Hazard ratio (95% confidence interval)			
	Model 1	Model 2	Model 3	Model 4
GNRI				
>98	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
92 to ≤98	3.56 (1.68–7.54)	3.51 (1.65–7.50)	3.19 (1.45–6.98)	3.03 (1.36–6.78)
<92	9.12 (4.92–16.88)	7.63 (4.00–14.56)	7.54 (3.76–15.13)	6.76 (3.13–14.56)

Model 1: Crude model.
Model 2: Adjusted for age and sex.
Model 3: Adjusted for variables included in Model 2 and conventional coronary risk factors (current smoker, diabetes mellitus, hypertension, dyslipidemia, and eGFR).
Model 4: Adjusted for variables included in Model 3, statins, and BNP.
BNP, brain natriuretic peptide; eGFR, estimated glomerular filtration rate; GNRI, geriatric nutritional risk index.

Table 6
Discrimination of each predictive model of cardiac death or non-fatal myocardial infarction after elective percutaneous coronary intervention.

	C-index	p-value	NRI	p-value	IDI	p-value
Established risk factors	0.68	Reference		Reference		Reference
+ body mass index	0.68	0.8	0.024	0.6	0.0022	0.2
+ serum albumin	0.76	0.003	0.40	<0.001	0.041	<0.001
+ GNRI	0.78	<0.001	0.46	<0.001	0.062	<0.001
+ body mass index vs. + GNRI	0.11[a]	<0.001	0.48	<0.001	0.060	<0.001
+ serum albumin vs. + GNRI	0.022[a]	0.1	0.15	0.049	0.022	0.02

Established risk factors included age, sex, and conventional coronary risk factors (current smoker, diabetes mellitus, hypertension, dyslipidemia, and estimated glomerular filtration rate).
GNRI, Geriatric Nutritional Risk Index; IDI, integrated discrimination improvement; NRI, net reclassification improvement.
[a] Differences between the two models.

were 7.78 (95% CI 3.53–17.18, $p < 0.001$) for GNRI <92 and 3.30 (95% CI 1.41–7.72, $p = 0.006$) for GNRI 92 to ≤98 compared with GNRI >98.

Discussion

The present study showed that GNRI was significantly associated with poor cardiac outcomes in patients with established coronary artery disease. To the best of our knowledge, this is the first study to evaluate the relationship between GNRI and long-term cardiac outcomes in this population. The results of this study suggest that evaluation of nutritional risk is important for risk stratification after elective PCI.

In previous studies, GNRI was demonstrated to be a nutrition-related risk index that makes it possible to classify patients according to a risk of morbidity and mortality in relation to pathologies in elderly patients that are often associated with malnutrition [11,16]. Thus, according to previous studies, patients with GNRI <92, 92 to ≤98, and >98 may represent those with "major-moderate nutrition-related risk", "low nutrition-related risk", and "no nutrition-related risk", respectively. Further studies are needed to assess if interventions designed to improve patients' nutritional status will improve their long-term cardiovascular outcomes.

In this study, GNRI <92 was associated with a lower prevalence of hypertension and dyslipidemia. Glycemic control was equivocal in GNRI <92 compared with GNRI 92 to ≤98 and GNRI >98. GNRI <92 did have better systolic blood pressure control as well as decreased low-density lipoprotein cholesterol and triglyceride levels. Conventional therapies for secondary prevention have not been found to be effective for patients with GNRI <92. Further studies should be performed to establish appropriate therapeutic strategies beyond the management of conventional coronary risk factors for this vulnerable patient group [18].

Some studies on patients who are on maintenance dialysis have found that chronic inflammatory status may causally tie underweight to increased mortality through malnutrition [19]. Previous studies have theorized that inflammation may promote a generally catabolic state, stimulating protein degradation and the suppression of protein synthesis. In addition, inflammation can also induce anorexia. Both these effects may cause protein-energy malnutrition and thus a lower body mass index [20–23]. This mechanism has also previously been reported in patients with cardiovascular disease [24]. In this study, increased C-reactive protein was independently correlated with lower GNRI. Increased C-reactive protein reflecting chronic inflammation might be an underlying cause of malnutrition in this study as previously reported, although this was not directly assessed.

Previous epidemiologic studies have found that underweight patients were associated with a significantly greater incidence of cardiovascular events and mortality compared with normal-weight or obese patients after PCI [8,9,25–27]. This relationship is often called a reverse causation, as these patients are often likely to be underweight because of malnutrition or cachexia. In our study, adding GNRI to traditional models for predicting cardiac events improved their predictive ability better than adding body mass index or serum albumin alone. Furthermore, adding body mass index to our prediction model did not improve our ability to predict negative cardiovascular outcomes in this study. Our results suggest that malnutrition, which may be a cause of underweight, is a stronger predictor of prognosis than underweight which may also be a result of intentional weight loss and may not always be a result of malnutrition or cachexia.

Study limitations

There are several limitations to this study. First, this was a single-center study with a relatively small study population. The observational nature of this study did not allow us to make definitive conclusions. Our findings require further confirmation to determine if there may be potential therapeutic implications. Second, we measured GNRI only once at baseline. Third, there is a possibility that some patients with lower GNRI may have an undiagnosed systemic illness, such as an occult malignancy. However, we assessed only cardiac events so the effects of non-cardiac diseases on our outcomes may be limited. Finally, dietary and exercise habits were not assessed in this study.

Conclusions

GNRI is independently associated with the incidence of cardiac events in patients after elective PCI. Our results might provide additional information for identifying high-risk patients who need careful attention after PCI. Further studies should be performed to establish appropriate therapeutic strategies for this vulnerable patient group.

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Conflict of interest

The authors declare that there is no conflict of interest.

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