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Prognostic nutritional index and the risk of acute kidney injury in patients with acute coronary syndrome

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SUMMARY

OBJECTIVE: Recent studies have linked malnutrition with undesirable outcomes in cardiovascular diseases. However, the underlying mechanism is unknown. Contrast-induced acute kidney injury (CI-AKI) increased cardiovascular mortality after percutaneous coronary intervention (PCI). This study hypothesizes that prognostic nutritional index (PNI) plays a role in the development of CI-AKI in patients with acute coronary syndrome undergoing emergency PCI.

METHODS: This study enrolled 551 patients. PNI was determined as 10× serum albumin (g/dL)+0.005×total lymphocyte count (mm³). CI-AKI was characterized as the increase in serum creatinine ≥0.3 mg/dL level within 48 h after PCI. Patients were classified as either CI-AKI (+) or CI-AKI (–).

RESULTS: CI-AKI has occurred in 72 of 551 patients (13.1%). PNI was significantly lower in the CI-AKI (+) group than in the CI-AKI (-) group (44.4±6.6 versus 47.2±5.8, p<0.001, respectively). Multivariate logistic regression analysis showed that PNI [odds ratio, OR: 1.631, 95% confidence interval (CI): 1.168–2.308, p=0.02] and estimated glomerular filtration rate (OR: 3.26, 95%CI 1.733–6.143, p<0.001) were independent risk factors for CI-AKI.

CONCLUSIONS: PNI is an independent risk factor for CI-AKI. The development of CI-AKI may be the mechanism responsible for the relationship between poor nutritional status and adverse cardiac events.

KEYWORDS: Prognostic nutritional index. Acute kidney injury. Acute coronary syndrome. Percutaneous coronary intervention.

INTRODUCTION

Poor nutritional status is linked to increased morbidity, mortality, hospitalization time, and reduced quality of life in patients with malignancy and renal disease[1,2]. Recent studies also linked malnutritional status with poor clinical outcomes in cardiovascular diseases such as acute heart failure, stable coronary artery disease, myocardial infarction, pulmonary embolism, and prognostic importance[3-5]. However, the pathophysiology is not defined yet.

Contrast-induced acute kidney injury (CI-AKI) is linked to morbidity and mortality in acute coronary syndrome (ACS). Furthermore, CI-AKI is one of the complications that can occur after percutaneous coronary intervention (PCI)[6,7]. The CI-AKI pathophysiology is complex, and the underlying mechanism is unknown[8].

Prognostic nutritional index (PNI) that can be calculated using serum albumin level and total lymphocyte used to evaluate immunonutritional status[9]. We hypothesized that the mechanism underlying poor clinical outcomes

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associated with under nutritional status in patients with ACS can be CI-AKI. The objective of this study was to investigate the relationship between PNI and CI-AKI in patients with ACS who underwent emergency PCI.

METHODS

This study prospectively evaluated 600 patients diagnosed with ACS that underwent emergency PCI. However 49 patients were excluded due to missing serum albumin levels or total lymphocyte count (n=15), end-stage renal disease (n=12), malignancy (n=11), death during PCI (n=5), active infection (n=3), previous chronic inflammatory disease (n=2), and severe liver cirrhosis (n=1). The study was completed with a total of 551 patients. The local ethics committee (Approval #2017,12,07,3028) approved the study. All patients signed a consent form after PCI.

The diagnosis of ACS was based on the guidelines of the European Society of Cardiology and the American College of Cardiology including ST-segment and non-ST segment/unstable angina. CI-AKI was characterized according to the Kidney Disease Improving Global Outcome criteria with an increase in serum creatinine (SCr) ≥0.3 mg/dL or ≥50% from the baseline SCr levels within 48 h after PCI[10]. Patients were classified as CI-AKI (-) or CI-AKI (+). PNI was calculated as 10× serum albumin (g/dL) + 0.005× total lymphocyte count (mm^3)[11].

Blood samples were collected 30 min after emergency admission but before PCI (baseline measurements) for the measurement of serum albumin, SCr, and complete blood count (CBC) within the first 30 min after an emergency admission, before PCI. Serum albumin and SCr were measured with the Olympus AU 600 autoanalyzer (Olympus Optical Co., Ltd., Schimatsu-Mishima, Japan). CBC was measured with an automatized CBC device (Abbott cell Dyn, Chicago, IL, USA). Demographic characteristics and risk factors were asked after PCI, and the patient was stabilized. Patients were not given treatment to prevent CI-AKI before the procedure. Intravenous hydration was given for at least 12 h after PCI. The duration of intravenous infusion was depended on the patient and the physician. All patients received a nonionic, iso-osmolar contrast agent. Coronary intensive care unit and follow-ups were performed by the cardiologist managing the patient. The treatments were arranged according to the current guidelines.

Statistical analysis

All statistical tests were carried out using SPSS version 22.0 (SPSS, Chicago, IL, USA). Continuous variables are shown as mean and standard deviation (SD) or median with interquartile ranges, and categorical variables are shown as percentages (%). Normal distribution was determined with the Kolmogorov-Smirnov test. If variables were normally distributed, the Student's t-test was used. The Mann–Whitney U test was used for continuous variables of non-normal distribution. The between-group comparisons were achieved by χ^2-test for categorical variables. To predict CI-AKI, a multiple stepwise logistic regression analysis with the backward elimination method was performed. The elimination criterion was defined as having a probability of above 0.10. The covariates in the regression model were as follows: age, gender, heart rate, systolic blood pressure, Mehran risk score, cardiogenic shock, urea, basal creatinine value, estimated glomerular filtration rate (eGFR), high-density lipoprotein, ejection fraction, PNI, and contrast amount. A $p<0.05$ was considered significant.

RESULTS

A total of 551 patients with ACS admitted to the coronary angiography laboratory for emergency PCI were enrolled. The average age was 62.5±10.7 years with 63% male (n=347). CI-AKI has occurred in 72 of 551 patients (13.1%). During the study, 17 patients died (3.1%). The demographic characteristics of the patients included in this study are presented in Table 1. Based on the development of CI-AKI development, 479 patients were classified as CI-AKI (-) and 72 were classified as CI-AKI (+). Age was significantly different between the two groups [age (years): CI-AKI (–), 62.1±10.7; CI-AKI (+), 65.1±0.1; p=0.02]. However, gender was not different [male, n (%): CI-AKI (-), 304 (63.5%); CI-AKI (+), 43 (59.7%); p=0.54]. Baseline SCr value, heart rate, Mehran risk score, amount of contrast used, hospital stay, and mortality rate were higher in the CI-AKI (+) group whereas the systolic blood pressure and eGFR rate were increased in the CI-AKI (-) group. Comorbidities such as hypertension, diabetes mellitus, previous myocardial infarction, previous stent implantation, and previous bypass operation were similar between the groups. The clinical features and laboratory results of the two groups are shown in Table 2.

The PNI was significantly lower in the CI-AKI (+) group [PNI: CI-AKI (+), 44.4±6.6; CI-AKI (-), 47.2 ± .8; p<0.001]. The multivariate logistic regression analysis showed that PNI [odds ratio, OR: 1.631, 95% confidence interval (CI): 1.168–2.308, p=0.02] and eGFR (OR: 3.26, 95%CI 1.733–6.143, p<0.001) were independent risk factors for CI-AKI (Table 3).

DISCUSSION

This study shows that PNI and eGFR levels are independent risk factors for the development of CI-AKI in patients with ACS undergoing PCI.

Recent studies have shown the clinical significance of nutritional status in cardiovascular diseases[3-5,12,13]. PNI is a marker

Table 1. Demographic characteristics of patients.

Characteristics	551 of patients
Age (years)	62.5±10.7
Female, n (%)	204 (37)
Body mass index (kg/m^2)	27.6±3.5
Admission heart rate (beats/min)	75.7±15.5
Admission systolic blood pressure (mmHg)	123.8±22.2
Admission diastolic blood pressure (mmHg)	73.4±15.4
Hypertension, n (%)	227 (41.2)
Diabetes mellitus, n (%)	163 (29.6)
Known coronary artery disease, n (%)	110(20.0)
Previous myocardial infarction, n (%)	66 (12.0)
Previous treatments	
Acetylsalicylic acid, n (%)	127 (23.0)
β-Blocker, n (%)	67 (12.2)
Angiotensin-aldosterone system antagonists, n (%)	205 (37.2)
Clinical presentation	
ST-segment elevation myocardial infarction, n (%)	368 (66.8)
Non-ST-segment elevation myocardial infarction, n (%)	122 (22.1)
Unstable angina pectoris, n (%)	61 (2.9)
Hematocrit (%)	41.1±4.8
Urea (mg/dL) median (interquartile range)	36.2 (17.3
Basal creatinine (mg/dL) median (interquartile range)	0.81 (0.24)
Serum albumin (mg/dL)	3.78±0.46
Prognostic nutritional index	46.8±6.0
Contrast amount (mL) median (interquartile range)	160 (70)
Estimated glomerular filtration rate (mL/min/1.73m^2)	89.4±27.1
Left ventricular ejection fraction (%) median (interquartile range)	54 (18)
Transradial approach, n (%)	179 (32.5)
Mehran risk score median (interquartile range)	2.0 (5.0)
Contrast-induced acute kidney injury, n (%)	72 (13.1)
Cardiogenic shock, n (%)	16 (2.9)
Inhospital mortality, n (%)	17 (3.1)

Table 2. Comparison of features of contrast-induced acute kidney injury (–) and contrast-induced acute kidney injury (+) groups.

Variables	CI-AKI (-) (n=479) Mean±SD/ median (IQR)	CI-AKI (+) (n=72) Mean±SD/ median (IQR)	p
Age (years)	62.1±10.7	65.1±10.1	**0.02**
Female, n (%)	175 (36.5%)	29 (40.3%)	0.54
BMI (kg/m^2)	27.5±3.3	27.9±4.2	0.38
Hypertension, n (%)	195 (40.7%)	32 (44.4%)	0.54
Diabetes mellitus, n (%)	138 (28.8%)	25 (34.7%)	0.30
Smoking, n (%)	90 (18.8%)	8 (11.1)	**0.03**
Heart rate (beats/min)	75.2±15.3	79.2±16.8	0.06
Systolic BP (mmHg)	124.6±21.8	118.1±24.0	**0.03**
Diastolic BP (mmHg)	73.9±15.3	69.9±16.0	0.05
Previous stent, n (%)	68 (14.2%)	12 (16.7%)	0.57
Previous CABG, n (%)	24 (5.0%)	6 (8.3%)	0.24
Previous MI, n (%)	55 (11.5%)	11 (15.3%)	0.35
Previous treatments			
β-Blocker, n (%)	57 (11.9%)	10 (13.9%)	0.63
ACE-İ/ARB, n (%)	177 (37.0%)	28 (38.9%)	0.75
Acetylsalicylic acid, n (%)	110 (23.0%)	17 (23.6%)	0.90
Clinical presentation			
STEMI, n (%)	316 (66.0%)	52 (72.2%)	0.55
NSTEMI, n (%)	108 (22.5%)	14 (19.4%)	
UAP, n (%)	55 (11.5%)	6 (8.3%)	
Transradial approach, n (%)	158 (33.0%)	21 (29.2%)	0.51
Ejection fraction (%)	55 (15)	48 (17.5)	**0.004**
LVEDD (cm)	4.7±0.6	4.6±0.6	0.79
LVESD (cm)	3.0±0.8	3.1±0.8	0.11
Mehran risk score	2 (5)	5 (5)	**<0.001**
Glucose (mg/dL)	114 (98–149)	121(101–181)	0.26
Urea (mg/dL)	35.7 (28.7–45.5)	39.4 (30.1–54.4)	**0.04**

Continue...

Table 2. Continuation.

Variables	CI-AKI (-) (n=479) Mean±SD/ median (IQR)	CI-AKI (+) (n=72) Mean±SD/ median (IQR)	p
Basal creatinine (mg/dL)	0.80 (0.71–0.92)	0.90 (0.82–1.23)	**<0.001**
eGFR (mL/min/1.73m^2)	91.6±26.5	75.0±27.1	**<0.001**
Hemoglobin (g/dL)	13.7±1.8	13.4±2.0	0.15
Hematocrit (%)	41.2±4.7	40.1±5.4	0.10
WBC (×10^9/L)	8.3±2.4	8.0±2.1	0.43
Lymphocyte count (×10^9/L)	1.8±0.7	1.6±0.6	**0.03**
Platelet count (×10^9/L)	245.2±75.2	237.6±85.2	0.43
Total cholesterol (mg/dL)	195.3±47.1	186.1±55.2	0.18
LDL (mg/dL)	120.9±41.1	127.0±45.3	0.25
HDL (mg/dL)	36.9±9.7	34.4±11.3	0.08
Triglyceride (mg/dL)	158 (111–217)	147 (116–188)	0.23
Albumin (g/dL)	3.8±0.4	3.6±0.5	**0.007**
Prognostic nutritional index	47.2±5.8	44.4±6.6	**<0.001**
Culprit coronary lesion			
Left anterior descending, n (%)	195 (40.7%)	38 (52.8%)	0.09
Circumflex, n (%)	105 (21.9%)	9 (12.5%)	
Right, n (%)	171 (35.7%)	20 (27.8%)	
Left main, n (%)	1 (0.2%)	1 (1.4%)	
Bypass graft, n (%)	7 (1.5%)	4 (5.6%)	
Contrast amount (mL)	160(60)	180 (81)	**0.01**
Hospital stay (day)	3 (2)	4 (3)	**0.001**
Cardiogenic shock	11 (2.3%)	5 (6.9%)	**0.03**
Inhospital mortality, n (%)	6 (1.3)	11 (15.3)	**<0.001**

CI-AKI: contrast-induced acute kidney injury; IQR: interquartile range; BMI: body mass index; BP: blood pressure; CABG: coronary artery bypass grafting; MI: myocardial infarction; ACE-İ: angiotensin-converting-enzyme inhibitors; ARB: angiotensin receptor blocker; STEMI: ST-elevation myocardial infarction; NSTEMI: non-ST-elevation myocardial infarction; UAP: unstable angina pectoris; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; eGFR: estimated glomerular filtration rate; WBC: White blood cell; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol. Bold numbers indicate statistically significant results (p<0.05).

for the evaluation of nutritional status but is also used for the assessment of immune and inflammatory status[9,14]. The reduction of serum albumin levels, one of the components of PNI, is associated with the increased severity of inflammation[15]. Hypoalbuminemia is an independent predictor of the long-term mortality in patients with ACS treated with primary PCI[15,16]. Other studies have shown that lymphocytes (another component of PNI) are playing a role in the inflammatory response in various stages of atherosclerosis[17]. For example, low lymphocyte counts were associated with complications and mortality in acute myocardial infarction[18].

In light of this information, it is not surprising that PNI is an independent predictor of mortality in patients with acute myocardial infarction[12,14]. However, the pathophysiological mechanism has not been clearly established. Various mechanisms are emphasized. A decrease in serum albumin levels leads to the increased inflammatory process, increased platelet aggregation, and increased blood viscosity, causing deterioration in endothelial function. Low lymphocyte counts contribute to this process due to an inadequate immunological reaction[19-22]. In this study, both albumin and lymphocyte counts were found to be different between groups.

CI-AKI with a sudden loss of renal function after contrast agent exposure occurs in 13.1% of the patient in our study, which is consistent with the literature (2–20%)[13]. The development of CI-AKI after PCI increases morbidity and mortality[23,24]. Interestingly, our study shows that both the hospital stay and the mortality rate were higher in the CI-AKI (+) group. The pathophysiology of CI-AKI has not been established yet and is very complex[25]. Inflammation, oxidative stress, free radical injury, and endothelial dysfunction are the main mechanisms involved in the pathophysiology of CI-AKI. The reduction of albumin, one of the major antioxidant agents in plasma, leads to an increase in oxidative stress. In addition, low serum albumin levels reflecting the inflammation burden in the body cause an increase in blood viscosity and impair endothelial function[8]. Notably, the mechanisms involved in the association of malnutrition with adverse cardiac events and many other mechanisms involved in the development of CI-AKI are similar. Thus, the relationship between poor nutritional status and CI-AKI is not surprising. In our study, the PNI value of the CI-AKI (+) group was significantly lower than that of the CI-AKI (-) group, and PNI was an independent predictor of CI-AKI.

Limitation

This study has limitations. First, this is a single-centered, observational study with relatively low enrollment. Second, the PNI values of the patients were only calculated once a baseline was not at follow-up and discharge. Third, the hormonal changes

Table 3. Independent predictors for contrast-induced acute kidney injury in patients with acute coronary syndrome undergoing emergency percutaneous coronary intervention.

Variables	Univariate analysis			Multivariate analysis		
	OR	(95%CI)	p	OR	(95%CI)	p
Age	2.08	(1.134–3.810)	0.01	1.65	(0.796–3.427)	0.17
Male	0.85	(0.514–1.416)	0.54			
Systolic blood pressure	0.59	(0.319–1.093)	0.09	0.57	(0.301–1.105)	0.09
Heart rate	1.69	(0.747–3.819)	0.20	1.01	(0.995–1.027)	0.17
Ejection fraction	2.25	(1.364–3.710)	0.002	2.01	(1.135–3.558)	**0.01**
Urea	2.01	(1.183–3.435)	0.01	092	(0.467–1.813)	0.81
Basal creatinine	2.50	(1.388–4.515)	0.002	0.91	(0.400–2.089)	0.83
eGFR	3.72	(2.171–6.396)	<0.001	3.26	(1.733–6.143)	**<0.001**
HDL-C	1.72	(0.982–3.038)	0.07			
Prognostic nutritional index	1.74	(1.281–2.708)	0.02	0.28	(1.631–2.308)	**0.02**
Mehran risk score	1.87	(1.139–3.122)	0.01	0.70	(0.348–1.417)	0.32
Contrast amount	3.38	(1.821–6.274)	<0.001	2.64	(1.582–4.916)	**0.01**
Cardiogenic shock	3.17	(1.070–9.422)	0.03	0.17	(0.680–9.144)	0.16

OR: odds ratio; CI: confidence interval; eGFR: estimated glomerular filtration rate; HDL-C: high-density lipoprotein cholesterol. Bold numbers indicate statistically significant results (p<0.05).

may affect PNI values and were not account for in this study. Fourth, specific inflammation and oxidative stress markers were not measured.

CONCLUSION

As a result, low PNI values are an important risk factor in the development of CI-AKI. The development of CI-AKI, in the association between poor nutritional status and increased adverse cardiac events, may be one of the underlying pathophysiological mechanisms. PNI may be guiding in determining patients with a high risk of the development of CI-AKI in patients with ACS who are undergoing emergency PCI, deciding to start preventive treatment earlier, and deciding which patients to monitor their renal function for longer.

AUTHORS' CONTRIBUTIONS

ALS: Project administration. **Aİ:** Writing-original draft. **AA:** Investigation. **ST:** Data curation. **NBA:** Data curation. **YA:** Methodology. **HA:** Data curation

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