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Relationship between prognostic nutritional index and contrast-induced nephropathy in patients presenting with acute coronary syndrome

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ABSTRACT

Objectives: This study aimed to evaluate malnutrition with the prognostic nutritional index (PNI) and investigate its association with contrast-induced nephropathy (CIN).

Patients and methods: The retrospective cohort study was conducted with 162 acute coronary syndrome (ACS) patients (113 males, 49 females; mean age: 58.6±12.6 years; range, 26 to 92 years) admitted between Novomber 2016 and September 2017. The patients were divided into two groups according to laboratory, angiographic, demographic, and echocardiographic parameters: those with CIN (n=16) and those without CIN (n=146). The data were obtained from the hospital system, and patients with complete parameters were included in the study. The PNI score was computed by multiplying the total lymphocyte count (mm^3) by 0.005 and adding it to 10 times the serum albumin (g/dL).

Results: Twenty-one (12.9%) of the patients had non-end-stage renal failure before the procedure, and the development of CIN was significantly higher in those with baseline renal dysfunction (50% *vs.* 8.9%, p<0.001). The PNI was significantly lower in the group that developed CIN (43.5±5.5 *vs.* 50.4±8.7, p=0.002). In-hospital mortality occurred in six (3.7%) of 162 patients who underwent PCI due to myocardial infarction. The mortality rate was significantly higher in the CIN group (18.8% *vs.* 2.1%, p=0.01). In the receiver operating characteristic analysis, a PNI <46 had 69% sensitivity and 75% specificity (area under the curve=0.76, 95% confidence interval 0.646-0.879, p=0.001) for determining CIN.

Conclusion: In conclusion, this study demonstrated that PNI, a marker of nutritional status, may be a reliable predictor of contrast nephropathy in patients presenting with ACS.

Keywords: Acute coronary syndrome, contrast-induced nephropathy, prognostic nutritional index.

Acute coronary syndrome (ACS) is a significant contributor to mortality, with a steady rise in occurrence despite advancements in therapy, such as percutaneous coronary intervention (PCI).[1] Therefore, it is necessary to identify high-risk patients based on their modifiable risk factors and use suitable therapies to enhance their prognosis. Malnutrition is an escalating worldwide health problem linked to adverse consequences of several illnesses.[2] The correlation between malnutrition and a poor outcome in cardiovascular disease has been demonstrated.[3,4] Nevertheless, there is a lack of studies investigating the correlation between malnutrition and contrast-induced nephropathy (CIN) in patients with ACS. Hence, this study aimed to examine the impact of the prognostic nutritional index (PNI) on the development of CIN in individuals who presented with ACS.

PATIENTS AND METHODS

The retrospective cohort study included 162 consecutive patients (113 males, 49 females; mean age: 58.6±12.6 years; range, 26 to 92 years) with ACS patients without end-stage renal disease and were admitted to the cardiology clinic of the Çiğli Training and Research Hospital between Novomber 2016 and September 2017. Patients with end-stage

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renal disease and those receiving hemodialysis were excluded from the study. Percutaneous coronary intervention was carried out in accordance with the treatment recommendations, based on the judgment of the attending physician. A written informed consent was obtained from each patient. The study protocol was approved by the İzmir Bakırçay University Ethics Committee (date: 08.03.2023 no: 2023/905). The study was conducted in accordance with the principles of the Declaration of Helsinki. Since the study did not include details on the exact kind of PCI therapy, the interventional cardiologists made the decision to perform predilatation, utilize intravascular ultrasound, and choose a particular type of drug-eluting stent. Patients were divided into two groups: those who developed CIN after the operations and those who did not develop CIN. Laboratory and echocardiographic data of the patients were obtained from the hospital information system.

The PNI score was calculated by multiplying the total lymphocyte count (mm^3) by 0.005 and adding it to 10 times the serum albumin (g/dL).[5]

Table 1
Baseline characteristics of patients according to CIN development

	CIN + (n=16)			CIN – (n=146)			
Variables	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			72.4±8.9			57.1±12.1	<0.001
Sex							
Male	6	37.5		107	73.3		0.007
Hypertension	15	93.8		95	65.1		0.02
Diabetes mellitus	10	62.5		36	24.7		0.003
Smoking	3	18.8		66	45.2		0.06
Hypercholesterolemia	16	100		138	94.5		1
Chronic renal failure	8	50		13	8.9		<0.001
CVD history	2	12.5		4	2.7		0.1
Prior CAD	8	50		54	37.2		0.32
Chronic heart failure	3	18.8		9	6.2		0.1
Multivessel disease	7	43.8		74	50.7		0.59
LVEF (%)			37.8±12.1			45.5±9.6	0.004
Received medication							
Statin	14	87.5		136	93.2		0.33
ACE-i/ARB	6	37.5		89	61		0.07
BB	13	81.3		122	83.6		0.73
MI type							
Anterior MI	9	56.3		55	37.7		
Inferior MI	5	31.3		57	39		0.32
Other MI	2	12.5		34	23.3		
Killip classification							
Killip Class I	5	31.3		115	78.8		
Killip Class II	6	37.5		23	15.8		<0.001
Killip Class III and IV	5	31.3		8	5.5		
In-hospital mortality	3	18.8		3	2.1		0.01

CIN: Contrast induced nephropathy; SD: Standard deviation; CVD: Cerebrovascular diseases; CAD: Coronary artery disease; LVEF: Left ventricular ejection fraction; ACE-i: Angiotensin-converting enzyme inhibitors; ARB: Angiotensin II receptor blocker; BB: Beta blocker; MI: Myocardial infarction.

Contrast-induced nephropathy was defined as the impairment of kidney function, measured as either a 25% increase in serum creatinine from baseline or a 0.5 mg/dL (44 μmol/L) increase in absolute serum creatinine value, within 48 to 72 h after intravenous contrast administration.

Statistical analysis

Data were evaluated in IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). The distribution of variables was evaluated with the Kolmogorov-Smirnov test, the homogeneity of variance was evaluated with the Levene test, and all continuous variables presented normal distribution. Data determined by measurement were given as mean ± standard deviation (SD) for those with normal distribution. The unpaired t-test was used in the analysis of these data. Categorical data were shown as absolute and relative frequencies, and the chi-square test or Fisher exact test was used, as appropriate. Variables were analyzed at a 95% confidence interval, and a p-value <0.05 was considered statistically significant. Receiver operating characteristic analysis was used, and the area under the curve was calculated.

RESULTS

Contrast-induced nephropathy developed in 16 (10%) of the patients. Sixty-four (40%) of the patients had anterior myocardial infarction (MI), 62 (38%) had inferior MI, and 36 (22%) had MI involving other walls. The majority of patients (74%) who underwent primary PCI were in the Killip Class I. Twenty-one (12.9%) of the patients had non-end-stage renal failure before the procedure, and the development of CIN was significantly higher in those with baseline renal dysfunction (50% *vs.* 8.9%, p<0.001). Development of CIN was significantly higher in the female sex and older age (p=0.007 and p<0.001, respectively). Rate of diabetes mellitus and hypertension was higher in the CIN group, whereas the mean left ventricular ejection fraction value was lower in the CIN group (Table 1).

Table 2
Baseline laboratory parameters on admission of patients according to CIN development

	CIN + (n=16)	CIN – (n=146)	
Variables	Mean±SD	Mean±SD	*p*
Creatinine (mg/dL)	1.45±0.5	1.11±1.1	0.2
eGFR (mL/min/1.73 m^2)	50.6±26.2	83.6±28.4	<0.001
Fasting blood glucose (mg/dL)	150.5±57.9	123.3±50.5	0.04
Total cholesterol (mg/dL)	190.7±67.3	187.5±42.6	0.79
HDL-cholesterol (mg/dL)	40.1±7.7	39.1±11.1	0.74
LDL-cholesterol (mg/d)	115.6±56.7	114.9±39.9	0.95
Plasma triglycerides (mg/dL)	169.4±74.1	174.1±92.9	0.85
Sodium (mEq/L)	138.4±4.5	138.8±3.3	0.66
Potassium (mEq/L)	4.2±1.1	4.3±0.5	0.71
White blood cell count (×10^9/L)	13.11±6.1	12.1±4.13	0.35
Lymphocyte count (×10^9/L)	1.76±0.57	2.36±1.24	0.06
Neutrophil count (×10^9/L)	9.83±5.25	8.35±3.67	0.14
Hemoglobin (g/DL)	12.3±2.1	13.4±2.1	0.03
Hematocrit (%)	34.3±8.7	39.9±5.7	0.001
Platelet count (×10^9/L)	259.6±59.7	257.5±73.1	0.916
Albumin (g/dL)	3.46±0.4	3.85±0.57	0.009
PNI	43.5±5.5	50.4±8.7	0.002

CIN: Contrast induced nephropathy; SD: Standard deviation; eGFR: Estimated glomerular filtration rate; HDL: High density lipoprotein; LDL: Low density lipoprotein; PNI: Prognostic nutritional index.

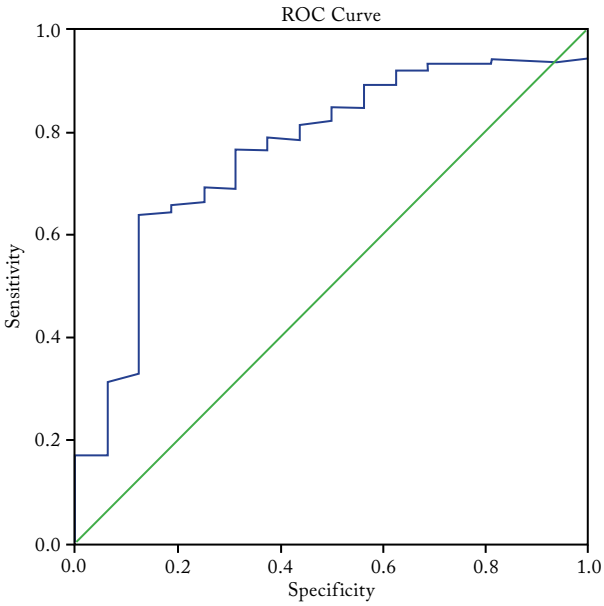

Figure 1. Receiver operating characteristic curve of PNI for predicting the development of CIN.

ROC: Receiver operating characteristic; PNI: Prognostic nutritional index; CIN: Contrast-induced nephropathy.

The PNI was significantly lower in the group that developed CIN (43.5±5.5 *vs.* 50.4±8.7, p=0.002). Furthermore, the rate of high fasting blood sugar and low hemoglobin and low hematocrit levels were significantly higher in the CIN group (p<0.05; Table 2).

In-hospital mortality occurred in six (3.7%) of 162 patients who underwent PCI due to MI. The mortality rate was significantly higher in the CIN group (18.8% *vs.* 2.1%, p=0.01).

In the receiver operating characteristic analysis (Figure 1), a PNI <46 had 69% sensitivity and 75% specificity (area under the curve=0.76, 95% confidence interval 0.646-0.879, p=0.001) for determining CIN.

DISCUSSION

The study assessed nutritional status using the PNI method and found that those with low PNI scores had significantly lower nutritional status and a higher incidence of CIN. Coronary angiography and PCI depend on the use of iodinated intravascular contrast to observe blood vessels and chambers. Although there have been improvements in imaging and interventional procedures, the use of iodinated contrast still has the potential to cause contrast-induced acute kidney damage in some high-risk individuals.[6] This disorder has a complicated pathogenesis, with a prevalence reaching up to 30%, and is linked to increased rates of both short-term and long-term illness and death.[7,8]

Considering the clinical situation and features of the patient, intravenous hydration should be taken into account as a component of the treatment for ACS patients with a low estimated glomerular filtration rate who are scheduled for invasive management. This is done to reduce the likelihood of CIN.[8-12] In our study, patients with ACS were analyzed. In these patients, it is recommended to use less contrast, administer high-dose statin in the early period, and provide intravenous hydration. Contrast-induced nephropathy is usually transient in these patients, but when dialysis is required, it can significantly impair long-term outcomes. Therefore, predicting which patients may develop CIN is important for post-PCI treatment decisions.

Prior research has shown a correlation between malnutrition and an increased susceptibility to contrast-associated acute kidney injury during PCI.[3] Hypoalbuminemia raises cardiovascular risk primarily via the reduction of albumin's antioxidant, oncotic pressure-maintaining, and antithrombotic abilities.[13] Decreased absolute lymphocyte numbers imply impaired immunological responses caused by malnutrition.[14] Malnutrition scores have the potential to aid in the categorization of risk and evaluation of prognosis.

In 1984, Onodera et al.[13] evaluated the nutritional and immunological condition of cancer patients who were having gastrointestinal surgery. In recent years, PNI has established recognition as a unique prognostic marker for several illnesses, including diabetic nephropathy, heart failure, and coronavirus disease 2019.[15-24] To our knowledge, this is the first study in the literature investigating the relationship between nutritional status and the development of contrast nephropathy in patients with ACS.

This study had several limitations, the primary one being its retrospective and single-center design. Additionally, the PNI's ability to predict nutritional status was similarly restricted. There may be variations in nutritional indices among various ethnic communities. As the PNI was generated using the original reference values, we did not have information about the changes in the nutritional values of

the patients. Prospective studies are necessary to determine the effect of dietary status on CIN in individuals with ACS.

In conclusion, this study demonstrated that PNI, a marker of nutritional status, may be a reliable predictor of contrast nephropathy in patients presenting with ACS. A low PNI score indicates an inadequate nutritional status, which is thought to accelerate inflammatory processes and lead to acute kidney injury.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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