Medeniyet Med J. 2020;35:47-54
doi:10.5222/MMJ.2020.86094

Original Study

Impact of Geriatric Nutritional Index in Contrast-Induced Nephropathy Developed in Patients with Non-ST Segment Elevation Myocardial Infarction who Underwent Percutaneous Coronary Intervention

Geriyatrik Beslenme İndeksinin Perkütan Koroner Girişim Uygulanmış ST Segment Yükselmesiz Miyokard İnfarktüslü Hastalarda Gelişen Kontrast Kaynaklı Nefropati Üzerine Olan Etkisi

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Ethics Committee Approval: This study approved by the Adana Training and Research Hospital, Clinical Studies Ethic Committee, 22 May 2019, 2019/463.
Conflict of interest: The authors declare that they have no conflict of interest.
Funding: None.
Informed Consent: Not Applicable.

Cite as: Kucukosmanoglu M, Yildirim A, Yavuz F, Dogdus M, Kilic S. Impact of geriatric nutritional index in contrast induced nephropathy in non-ST segment elevated myocardial infarction patients performed percutaneous coronary intervention. Medeniyet Med J. 2020;35:47-54.

ABSTRACT

Objective: *Geriatric nutritional risk index (GNRI) is a useful tool to determine the nutritional status of patients. Any study has not evaluated the impact of GNRI in development of contrast- induced nephropathy (CIN) after percutaneous coronary intervention (PCI). We aimed to evaluate whether GNRI could predict CIN after PCI.*
Method: *A total of 1116 patients with non-ST elevation myocardial infarction (non-STEMI) that underwent PCI were enrolled to the present study. The GNRI was calculated using a previously reported formula: GNRI=14.89 × albumin (g/dL) + 41.7 × body weight (kg)/ideal body weight (kg). CIN was defined as an increase in serum creatinine level of ≥0.5 mg/dL or ≥25% above baseline within 72 hours after the PCI procedure. The patients were categorized into two groups as CIN (+) and CIN (-).*
Results: *The mean age of the CIN (+) group was significantly higher than the CIN (–) group (64.8±10.67 vs. 60.5±10.61 years; p<0.001). The mean values of height, weight, and body mass index were significanlty lower in CIN (+) group than CIN (-) group (p<0.001, for all). The mean of GNRI was significantly lower in the CIN (+) group than the CIN (-) group (101.4±8.7 vs. 112.1±12.9; p<0.001). Serum albumin level was significantly lower in the CIN (+) group (3.71±0.52 g/dL vs. 3.94±0.53 g/dL; p<0.001). Left ventricular ejection fraction (LVEF) was significantly lower in the CIN (+) group (50.7%±9.07 vs. 54.3%±7.20; p<0.001).*
Conclusion: *In this study, GNRI, serum albumin level, BMI, and LVEF were independent predictors of CIN. Moreover, GNRI was better than both serum albumin level and BMI in predicting development of CIN.*

Keywords: *Geriatric nutritional index, contrast induced nephropathy, percutaneous coronary intervention*

ÖZ

Amaç: *Geriyatrik beslenme risk indeksi (GBİ) hastaların beslenme durumunu belirlemek için kullanılan yararlı bir araçtır. GBİ' nin perkütan koroner girişimler (PKG) sonrasında gelişen kontrast kaynaklı nefropati (KKN) gelişimi ile ilişkisini değerlendiren bir çalışma yoktur. Biz bu çalışmamızda GBİ'nin PKG sonrası gelişen KKN'nin öngördürücüsü olup olmadığını değerlendirmeyi amaçladık.*
Yöntem: *Merkezimizde ST segment yüksekliği olmayan miyokard infarktüsü tanısı almış ve PKG uygulanmış 1116 hasta çalışmaya dahil edildi. GBİ hastane kayıtlarındaki veriler kullanılarak 14.89 × serum albumin (g/dL) + 41.7 × güncel vücut ağırlığı (kg)/ideal vücut ağırlığı (kg) formülü ile hesaplandı. KKN, PKG işleminden sonraki 72 saat içinde serum kreatinin seviyesinin ≥0,5 mg/dL veya başlangış düzeyinden ≥%25 oranında artması olarak tanımlandı. Hastalar KKN gelişenler (KKN (+)) ve KKN gelişmeyenler (KKN (-)) olarak iki gruba ayrıldılar.*
Bulgular: *KKN (+) grubunun yaş ortalaması KKN (-) grubundan anlamlı olarak yüksekti (64.8±10.67 ve 60.5±10.61; p<0.001). Ortalama boy, kilo ve vücut kitle indeksi (VKİ) değerleri KKN (+) grubunda KKN (-) grubuna göre anlamlı olarak daha düşüktü (hepsi için p<0.001). GBİ ortalaması KKN (+) grubunda KKN (-) grubundan anlamlı olarak daha düşüktü (101.4±8.7 vs. 112.1±12.9; p<0.001). KKN (+) grubunda serum albümin düzeyi anlamlı olarak daha düşüktü (3.71±0.52 g/dL ve 3.94±0.53 g/dL; p<0.001). Sol ventrikül ejeksiyon fraksiyonu (SVEF) KKN (+) grubunda anlamlı derece de düşüktü (%50.7±9.07 ve %54.3±7.20; p<0.001).*
Sonuç: *Bu çalışmada, GBİ, serum albumin seviyesi, VKİ ve SVEF KKN'in bağımsız belirleyicileri olarak tespit edildi. Dahası, GBİ KKN gelişimini öngördürmede hem serum albümin seviyesinden hem de VKİ'den daha iyi bulundu.*

Anahtar kelimeler: *Geriyatrik beslenme indeksi, kontrast kaynaklı nefropati, perkütan koroner girişim*

Received: 24 December 2019
Accepted: 14 February 2020
Online First: 28 February 2020

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INTRODUCTION

Contrast-induced nephropathy (CIN) is a commonly observed undesirable condition in patients undergoing percutaneous coronary intervention (PCI). CIN is related with poorer outcomes for patients, including need for renal dialysis and higher mortality[1-3]. Previous studies reported that the prevalence of CIN after CAG or PCI procedures range from 2%-15% and can reach up to 50% in patients under high risk such as diabetes mellitus (DM) and pre-procedural renal failure[4,5]. CIN was previously defined as an increase in baseline serum creatinine level (Cr) 25% or 0.5 mg/dL within 48-72 h after PCI[1-3]. Although, the physiological mechanisms of CIN are not certain, oxidative stress, inflammation, renal medullary hypoxia, and negative effects of contrast media are supposed to be the underlying physiological mechanisms[6-9]. The previous studies determined that age, DM, heart failure, anemia, chronic (pre-existing) kidney disease, hypoalbuminemia, metabolic syndrome, overuse of high osmolar contrast medium, and peripheral vascular disease are predictors of CIN[1-9]. Also, relation between body mass index (BMI) and development of CIN were evaluated in different studies and the results are confounding[1-4]. The clinical impact of malnutrition has been shown in chronic kidney disease and cardiovascular diseases[5-8]. Geriatric nutritional risk index (GNRI) is a simple tool to identify the nutritional status of subjects. The clinical impact of GNRI has been demonstrated in many diseases[9-12]. GNRI is calculated using a simple formula that contains albumin and BMI.

According to past medical knowledge and consensus, the impact of GNRI in development of CIN after PCI in non-ST-segment elevation myocardial infarction (NSTEMI) patients has not been evaluated yet. The aim of present study is evaluation of whether GNRI could predict CIN after PCI in NSTEMI patients.

MATERIAL and METHODS

All patients diagnosed with non-STEMI who had undergone PCI in our center between January 1, 2018 and December 31, 2018 were retrospectively enrolled in the present study. Patients that were followed-up with medical therapy, those with a history of heart failure, active malignancy, hematologic disorder, kidney transplantation or end-stage renal disease requiring dialysis, nephrotic syndrome, liver dysfunction, systemic immune system, or connective tissue disease or given contrast media within the last two weeks, were excluded from the study. Also, patients who had used the nephrotoxic drugs during periprocedural period, and had not measured their serum creatinine levels before and 72 hours after the procedure were excluded from the study. Patients with typical chest pain, objective signs of myocardial ischemia, and elevated biochemical markers of myocardial necrosis were diagnosed with NSTEMI[13]. All patients included in the study received same low-osmolar contrast material.

Hypertension was defined as systolic and diastolic blood pressures ≥140/90 mmHg or use of any antihypertensive drug. Diabettes mellitus (DM) was defined as fasting blood glucose ≥126 mg/dL or HbA1c ≥6.5 or use of any antidiabetic drug. Dyslipidemia was determined as total cholesterol level >200 mg/dL, and/or undergoing treatment with statins and/or lipid-lowering agents. Current smoker was defined as the patient smoking at least 1 cigarette/day for at least one year.

The baseline characteristics of patients were recorded from patients' files, and routine laboratory parameters were retrieved from the hospital laboratory digital system. Transthoracic echocardiography was performed by experienced echocardiographers according to relevant guidelines[14]. GNRI values were calculated from the hospital admission database. The GNRI was calculated using a previously reported formula: GNRI = 14.89 × albumin (g/dL) + 41.7 × body weight (kg)/ide-

al body weight (kg). The ideal body weight was calculated as follows: body height - 100 - [(body height -150)/4] for males, and body height - 100 - [(body height - 150)/2.5] for females. The patients were classificated into CIN (+) and CIN (-) groups. The local ethics committee approved the protocol of the study.

Statistical analyses

Continuous variables with normal distribution were summarized as mean (±standart deviation) and compared between groups with Student's t-test. Variables without normal distribution summarized as median and interquartile range and compared between groups with Mann-Whitney U-test. Categorical variables were summarized as numbers and percentages and compared using chi-square test. To demonstrate the sensitivity and specificity of GNRI, albumin, and BMI and their cut-off values for CIN development, the receiver operating characteristics (ROC) curve was used. The area under curve (AUC) comparison of GNRI, albumin, and BMI were performed using the Delong method[15]. To predict independent parameter for CIN development multivariate logistic regression analysis were performed.

Two programs were used for statistical analysis: Statistical Package for the Social Sciences (SPSS 20.0) (SPSS Inc., Chicago, Illinois, USA) and MedCalc 15 statistical software (Ostend, Belgium). Statistical significance was considered when p value was <0.05%.

RESULTS

During the study period, 1116 patients (33.4% female, mean age 60.61±10.73 years) were included in the study. Of those, 190 (17.0%) developed CIN. Baseline demographic and medical characteristics of groups are presented in Table 1. Compared to the CIN (-) group, the mean age of the CIN (+) group was statistically significantly higher (64.8±10.7 vs. 60.5±10.6; p<0.001). The mean values of height, body weight, and BMI were significanlty lower in CIN (+) group than

Table 1. Baseline demographic and clinical characteristics and echocardiographic parameters of patients.

Parameters	All Patients N=1116	CIN (+) N=190	CIN (–) N=926	P
Age, year	60.61±10.73	64.8±10.67	60.5±10.6	<0.001*
Weight, kg	77.4±13.4	66.6±5.2	79.6±13.5	<0.001*
Height, m	1.67±0.08	1.65±0.08	1.67±0.07	<0.001*
BMI, kg/m²	27.3±4.6	26.6±4.1	28.43±4.59	<0.001*
Female, % (n)	33.8 (377)	34.7 (66)	33.6 (311)	0.760
Obesity, % (n)	30.2 (337)	19.5 (37)	32.4 (300)	<0.001*
Diabetes mellitus, % (n)	38.1 (425)	44.7 (85)	36.7 (340)	0.038*
Hypertension, % (n)	69.4 (775)	68.9 (131)	69.5 (644)	0.870
Hyperlipidemia, % (n)	39.0 (435)	36.3 (69)	39.5 (366)	0.409
Heart failure, % (n)	15.4 (171)	17.4 (33)	14.9 (138)	0.397
Chronic obstructive pulmonary disease, % (n)	12.2 (136)	7.9 (15)	13.1 (121)	0.047
Smoker, % (n)	40.1 (447)	33.2 (63)	41.5 (384)	0.033*
Stroke, % (n)	3.4 (38)	4.2 (8)	3.2 (30)	0.505
GNRI	110.3±12.9	101.4±8.7	112.1±12.9	<0.001*
Contrast volume (mL)	182.6±58.6	177±49.5	183±60	0.183
Echocardiography Parameters				
LVESV, mL	55.2±15.1	60.5±18.42	54.1±13.99	<0.001*
LVEDV, mL	118±19.5	121.5±20.68	118.0±19.29	0.027*
LVEF, %	53.7±7.67	50.7±9.07	54.3±7.20	<0.001*
sPAP, mmHg	22.7±5.65	23.6±4.89	22.6±5.79	0.898

*BMI: body mass index, CIN: contrast induced nephropathy, GNRI: geriatric nutritional index, LVEDV: left ventricular end-diastolic volume, LVEF: left vetricular ejection fraction, LVESV: left vetricular end-systolic volume, sPAP: systolic pulmonary artery pressure. *Statistically significant*

Table 2. Comparision of blood parameters of patients.

Parameters	All Patients N=1116	CIN (+) N=190	CIN (–) N=926	P
Total Protein, (g/dL)	6.64±0.83	6.59±0.62	6.65±0.87	0.356
Albumin, (g/dL)	3.9±0.5	3.71±0.52	3.94±0.53	<0.001*
Pre-Procedural Creatinine, (mg/dL)	0.87±0.34	0.84±0.33	0.88±0.34	0.160
Post-Procedural Creatinine, (mg/dL)	0.97±0.40	1.24±0.58	0.91±0.33	<0.001*
Uric acid, (mg/dL)	5.54±1.52	5.91±2.04	5.47±1.36	0.005*
Total bilirubin, (mg/dL)	0.55±0.31	0.51±0.27	0.56±0.32	0.046*
WBC, (10^9/L)	9100±2890	9572±3347	9016±2781	0.033*
Haemoglobin, (g/dL)	13.8±5.37	13.0±2.20	14.0±5.80	0.022*
Haematocrit, %	40.2±5.1	38.7±5.9	40.5±4.89	<0.001*
Platelet, (10^9/L)	244±73	235±83	246±71	0.117
Lymphocyte, (10^9/L)	2160±960	2159±1036	2162±951	0.971
Neutrophil, (10^9/L)	5930±2432	6359±2934	5841±2308	0.023*
Monocyte, (10^9/L)	1020±650	1053±484	1012±686	0.333
Total cholesterol (mg/dL)	186±43	179±38	188±44	0.004*
LDL-C (mg/dL)	121±34	119±32	122±35	0.226
HDL-C (mg/dL)	40±11	38±10	40±11	0.011*
Triglyceride (mg/dL)	163±99	147±77	166±103	0.005*
Glucose, (mg/dL)	148±75	169±85	144±72	<0.001*
HbA1c, %	6.6±1.8	6.77±1.62	6.61±1.79	0.262
Troponin I, (ng/L)	66 (18-456)	166 (30-1792)	58 (18-368)	<0.001*
BNP, (ng/L)	246 (98-1210)	260 (87-1600)	274 (94-1053)	0.341

BNP: brain natriuretic peptide, CIN: contrast induced nephropathy, HDL-C: high density lipoprotein cholesterol, LDL-C: low density lipoprotein cholesterol.
** Statistically significant*

Table 3. Logistic regression analysis for predictors of contrast-induced nephropathy.

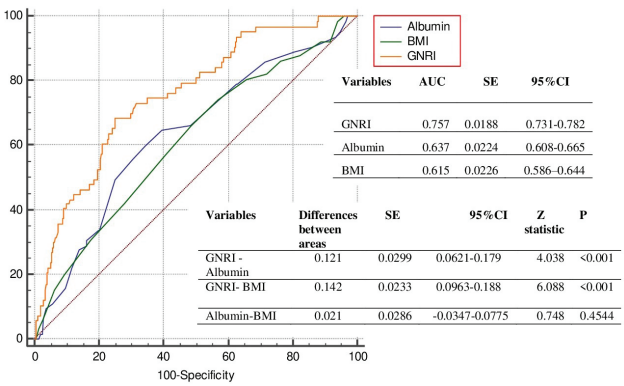
Analysis		Univariate		Multivariate
Variables	**P**	**OR [95% CI]**	**P**	**OR [95% CI]**
Age (per 1 year increase)	<0.001*	1.036 (1.020-1.052)	-	-
Diabetes mellitus (yes vs. no)	0.039*	1.395 (1.018-1.913)	-	-
Weight (per 1 kg decrease)	<0.001*	0.867 (0.845-0.890)	-	-
BMI (per 1 kg/m^2 decrease)	<0.001*	0.905 (0.870-0.943)	0.004*	0.942 (0.905-0.981)
Albumin (per 1 g/dL decrease)	<0.001*	0.468 (0.355-0.619)	0.019*	0.979 (0.962-0.996)
GNRI (per 1 point decrease)	<0.001*	0.871 (0.848-0.895)	<0.001*	0.762 (0.725-0.801
LVEF (per 1% decrease)	<0.001*	0.949 (0.932-0.966)	0.009*	0.971 (0.951-0.993)
Uric acid (per 1 mg/dL increase)	<0.001*	1.177 (1.080-1.310)	-	-
Hemoglobin (per 1 g/dL decrease)	<0.001*	0.847 (0.782-0.918)	-	-
HDL-C (per 1 mg/dL decrease)	0.011*	0.981 (0.966-0.996)	-	-
Triglyceride (per 1 mg/dL decrease)	0.020*	0.998 (0.996-1.000)	-	-
Troponin (per 1 ng/dL increase)	<0.001*	1.001 (1.000-1.001)	-	-

BMI: body mass index, HDL-C: high denstiy, GNRI: geriatric nutritional index, LVEF: left vetricular ejection fraction
**Statistically significant*

CIN (-) group (p<0.001, for all). Compared to the CIN (-) group, the rates of smoking and chronic obstructive pulmonary disease were significantly lower in the CIN (+) group (p<0.05, for both). Left ventricular ejection fraction (LVEF) was significantly higher in the CIN (-) group than CIN (+) group (54.3%±7.2% vs. 50.7%±9.1%; p<0.001). Laboratory parameters of groups are summarized in Table 2. Serum albumin level was significantly lower in the CIN (+) group (3.71±0.52 g/dL vs. 3.94±0.53 g/dL; p<0.001). Pre-procedural serum creatinine levels were similar between groups (CIN (+) 0.84±0.33 mg/dL vs. CIN (-) 0.88±0.34 mg/dL; p<0.160), as expectedly post-procedural

creatinine levels were statistically higher in the CIN (+) group (1.24±0.58 mg/dL vs. 0.91±0.33 mg/dL; p<0.001). Also, mean serum uric acid level, and white blood cell (WBC) count were significantly higher and hemoglobin level was lower in the CIN (+) group than the CIN (-) group (Table 2). The mean GNRI was significantly lower in the CIN (+) group than the CIN (-) group (101.4±8.7 vs. 112.1±12.9; p<0.001) (Table 1).

To predict CIN development, the cut-off value of albumin ≤3.8 g/dL has a 60.0% sensitivity and 65.66% specificity (AUC: 0.637; 95% confidence interval [CI] 0.608-0.665; p<0.001) and BMI ≤27 kg/m^2 has a 66.32% sensitivity and 51.51% specificity (AUC: 0.615; 95% CI 0.586-0.644; p<0.001), and GNRI ≤110.7 has a 85.8% sensitivity and 55.2% specificity (AUC: 0.757; 95% CI 0.731-0.782; p<0.001) in the ROC curve analyses (Figure 1). In the pairwise comparison of ROC analyses, GNRI was found to be a statistically significant better than albumin and BMI in predicting development of CIN (for both; p<0.001) (Figure 1).



Variables	AUC	SE	95%CI
GNRI	0.757	0.0188	0.731-0.782
Albumin	0.637	0.0224	0.608-0.665
BMI	0.615	0.0226	0.586-0.644

Variables	Differences between areas	SE	95%CI	Z statistic	P
GNRI - Albumin	0.121	0.0299	0.0621-0.179	4.038	<0.001
GNRI- BMI	0.142	0.0233	0.0963-0.188	6.088	<0.001
Albumin-BMI	0.021	0.0286	-0.0347-0.0775	0.748	0.4544

Figure 1. Comparison of Receiver operating characteristic (ROC) curves for CIN development.
AUC: area under the curve, BMI: body mass index, CI: confidence interval, GNRI: geriatric nutritional risk index, SE: standart error

To define the predictors of CIN, univariate and multivariate logistic regression analyses were performed and results are summarized in Table 4. The univariate analysis demonstrated that history of DM, age, BMI, weight, LVEF, GNRI, and levels of troponin, uric acid, HDL-C, triglyceride, hemoglobin and albumin were predictors of CIN development. The multivariate analysis that examined age, BMI, weight, DM, LVEF, GNRI, and troponin, uric acid, HDL-C, triglyceride, hemoglobin and albumin levels demostrated that albumin (per 1- g/dL decrease) (OR: 0.979; 95% CI 0.962-0.996; p=0.019), BMI (per 1 kg/m^2 decrease) (OR: 0.942; 95% CI: 0.905-0.981; p=0.004), GNRI (per 1 point decrease) (OR: 0.762; 95% CI 0.725-0.801; p<0.001), and LVEF (per 1% decrease) (OR: 0.971; 95% CI 0.951-0.993; p=0.009) were the independent predictors of CIN development.

DISCUSSION

In our study, we showed that low values of GNRI, serum albumin, BMI, and LVEF were independent predictors of CIN. Moreover, GNRI was better than both serum albumin and BMI in predicting development of CIN after PCI.

CIN is a frequent cause of acute kidney disease in hospitalized patients. Published studies have reported an increase rate of CIN in patients diagnosed with acute coronary syndrome and had undergone PCI. The prevalance of CIN in patients diagnosed with myocardial infarction and underwent PCI was found to be between 12 and 26%[16-18]. Since there is no sole marker for CIN, many markers have been reported to be related with CIN development[16-19]. Previous medical history of diabetes mellitus (DM) or chronic kidney diseases have been shown to be significant risk factors of CIN development after PCI[16-19]. Also, the contrast agent volume is an important risk factor for CIN development. Moreover, many other risk factors that are related to CIN development have been reported in different studies that performed PCI including advanced age, previous history of heart failure, hypoalbuminemia, anemia, C-reactive protein, low BMI, nephrotoxic drugs use, hemodynamic instability, white blood cell count and use of intra-aortic ballon pump[16-18,20-22]. The pathophysiology of CIN is not clear. The inflammation, oxidative stess, renal medullar hypoxia,

negative effects of contrast agents, impaired balance of vasodilation and vasoconstriction in renal medulla have been considered to be possible risk factors for the developmet of CIN. The association between hypoalbuminemia and cardiovascular morbidity/mortality has been shown in different studies. Moreover, studies have demonstrated that low serum albumin level is related to mortality in patients diagnosed with acute coronary syndrome[17,23-25]. However, the relationship between serum albumin and CIN is not clear, and several possible mechanisms may be involved. Antioxidative effects of albumin are important for the development of CIN. Because albumin is an important free oxygen radical capture from plasma and oxidative stress is a possible factor to the development of CIN, the low level of albumin is an significant predictor of CIN development[26,27]. Another possible relationship between albumin and CIN may be explained by inceased inflammation status in CIN patients[28,29]. Due to an inverse relation between inflamation and albumin, low albumin levels in CIN patients might be explained by increased inflamation status. The relation between BMI and CIN development has been evaluated in different studies and the results are confounding[1-4]. Some studies have shown that low BMI, while others have established that a high BMI is a risk factor. Recently, Kuno et al.[2] showed that the relationship between BMI and CIN is a reverse J-curve relationship and the incidence of CIN is high both in patients with BMIs <20 kg/m^2 and >30 kg/m^2 than those with BMIs between 20-30 kg/m^2. In the present study, we have shown that mean BMI of the CIN (+) group is lower than the CIN (-) group and BMI is an independent predictor of CIN development. GNRI is a simple index and well-established nutritional screening tool for elderly patients that has been evaluated in various cardiovascular diseases[9-12,30]. Malnutrition is common in patients suffered from a cardiovascular disease especially in heart failure patients[10,30]. The valid formula for the GNRI include serum albumin levels and weights of patients. Also, the formula included ideal weight of patients which is calculated from body height. In this present study, we showed that the GNRI is an independent predictor of CIN development. Moreover, it is significantly better than both serum albumin levels and BMI in predicting CIN development. The possible assocation between CIN development and malnutrition is not certain. The most possible symptom is inflammation. As emphasized above, inflammation is related to CIN development and malnutrition is closely associated with systemic inflammation. Consequently, patients with malnutrition are likely to experience CIN. Moreover, similar to BMI, GNRI is affected by both body weight and height. However, in the formula of GNRI the ideal body weight is also calculated which might explain the difference between BMI and GNRI. Many previous studies reported that CIN development is frequently seen in advanced age patients[31-33]. The possible mechanisms underlying CIN development in the elderly age is not clear but probably is associated with change in renal function with aging. In the present study, we found in the multivariate analysis, that age is not an independent predictor of CIN development. This might explain the reason why the mean age of study population was 60.61±10.73 years. The contrast volume is a modifiable risk factor for development of CIN and previous studies established the association between contrast volume and the risk of CIN development[33-35]. In their study, Nikolsky et al.[36] showed that increase of every 100 ml in the amount of contrast material resulted in a 30% increase in the odds ratio of CIN development. In the present study, the mean volume of contrast material was 182.6±58.6 mL which was comparable between groups. Anemia might be associated with CIN development. A different study published by Nikolsky et al. showed that decreased level of pre-procedural hematocrit is related to CIN development[37].

In the present study, the mean hematocrit and hemoglobin levels were found to be lower in the CIN (+) group. Although hemoglobin was a predictor of CIN development in the univariate

analysis, it lost its significance in the multivariate analysis. Heart failure has been reported to be a risk factor for CIN development in patients pwho underwent PCI[38]. Similarly, in this study the mean level of LVEF was signifincantly lower in the CIN group. Moreover, LVEF was an independent predictor of CIN development.

CONCLUSIONS

In this study, we have determined that the prevelance of CIN development is significantly higher in patients diagnosed with non-STEMI and underwent PCI. In addition, GNRI is an independent predictor of CIN development and is better than BMI and serum albumin level in patients diagnosed with non-STEMI who underwent PCI.

Study limitations

The main limitation of our study is its retrospective single- centered design. All patients diagnosed as post-PCI nephropathy which might be related to an extraneous causes as pre-renal disorders or cholestrol emboli. However, due to the nature of the study design it is not possible to diagnose these infrequently seen etiologies. Another limitation is that although we included all consecutive patients, some patients might be discharged after PCI without controlling their creatinine levels. But due to the sufficient number of patients in two groups, we believe that our results accurately reflect the patient population as a sample group. Another important limitation of study was that we included only patients diagnosed with non-STEMI.

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