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Original Article

Investigation of the complete blood count parameters as an early diagnostic tool in contrast-induced nephropathy after contrast-enhanced computed tomography

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Abstract

Aim: We investigated complete blood count parameters as inflammatory biomarkers and compared these to serum creatinine values as early diagnostic criteria of ongoing contrast-induced nephropathy. Contrast-induced nephropathy is an important cause of acute kidney injury. Early diagnosis can reduce morbidity and mortality. There is no clear predictor parameter for the early diagnosis of contrast-induced nephropathy.

Material and Methods: Patients who underwent contrast-enhanced computed tomography examination were included in this retrospective study. Contrast-induced nephropathy was defined as 25%, a higher increment or a 0.5 mg/dL elevation above the baseline serum creatinine levels within 72 hours. Patients were divided into contrast-induced nephropathy and non-contrast-induced nephropathy groups. The complete blood count parameters obtained before and within the first 24 hours after contrast-enhanced computed tomography were compared between groups.

Results: The post-contrast-enhanced computed tomography neutrophil-to-lymphocyte ratio values were significantly higher in the contrast-induced nephropathy group compared to the non-contrast-induced nephropathy group (11.85±1.56 vs 7.29±0.49; p = 0.000). Comparison of the post-contrast-enhanced computed tomography values of the platelet-to-lymphocyte ratio, mean platelet volume-to-platelet count ratio, and lymphocyte to monocyte ratio revealed no statistically significant differences between the groups (p = 0.283, 0.128, and 0.792 respectively).

Conclusions: An increased neutrophil-to-lymphocyte ratio level after a contrast-enhanced computed tomography procedure is associated with the development of contrast-induced nephropathy. The use of the neutrophil-to-lymphocyte ratio in the emergency department as a predictive parameter can significantly improve the diagnostic process, favorably acting on the prognosis of patients developing contrast-induced nephropathy.

Keywords: Acute kidney injury; contrast agents; emergency department; inflammatory biomarkers; neutrophil-to-lymphocyte ratio

INTRODUCTION

Contrast-induced nephropathy (CIN) is an important cause of acute kidney injury (AKI) in outpatients and inpatients. Due to the common use of iodine-containing contrast agents for diagnostic purposes, CIN is the third leading cause of hospital-acquired renal insufficiency (1). The incidence of CIN has been reported to be 6% in patients undergoing contrast-enhanced computed tomography (CECT) (2); however, emergency department (ED)-based studies are limited in terms of CIN incidence.

Although the pathophysiology of CIN has not been completely understood; the toxic effects of the contrast agent on tubular epithelial cells, apoptosis, disturbances in intrarenal hemodynamics, and medullary hypoxia are examined as potential factors involved in the underlying mechanisms of CIN (3). Diagnostic criteria define CIN as a clinical condition with a 25%, a higher increment or a 0.5 mg/dL elevation above the baseline serum creatinine (SCr) levels within 72 hours (4). These diagnostic criteria allow for detecting AKI late based on baseline SCr levels a limitation. CIN may result in permanent kidney injury and the need for dialysis. Early diagnosis and treatment can reduce morbidity and mortality (5). The CECT procedure is frequently used to achieve an accurate and precise diagnosis by ED physicians. Outpatients after the CECT procedure may be overlooked in terms of incidence of AKI compared with hospitalized patients from the ED.

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However, CIN can be diagnosed late even if patients are hospitalized. Therefore, there is a need to identify more sensitive biomarkers that would provide early warning criteria for AKI. Several parameters including plasma neutrophil gelatinase (NGAL), plasma cystatin-C (CysC), and urinary NGAL have been investigated as potential biomarkers to allow for early diagnosis of CIN-related AKI (6). But most of these biomarkers are expensive and not routinely used in ED practice unlike the complete blood count (CBC).

AKI is known to be associated with intrarenal and systemic inflammatory conditions that include several processes related to complex responses involved in tissue repair after injury (7). An ongoing process starts at the site of injury with the migration of neutrophils and macrophages, followed by lymphocytic adaptive responses (8). The neutrophil-to-lymphocyte ratio (NLR) and other parameters derived from the CBC have been studied in the literature commonly as potential markers indicating inflammatory responses in malignancies, hypertension, and cardiovascular diseases (9-11). Several tests including CBC have been performed routinely on the admission of the patient to the ED as components of standard delivery of care; however, an emergency physician rarely evaluates the results of these tests for their diagnostic/prognostic value to construct a knowledge base with the potential to influence standards of the ED practices directly (12).

In this study, we aimed to evaluate the predictive value of CBC parameters for the potential development of CIN after hospitalization of ED patients subjected to CECT examination.

MATERIALS AND METHODS

Study Design and Participants

This retrospective non-interventional study was approved by the Gulhane Ethics Board of the University of Health Sciences on November 2019 (Committee IRB approval number: 2019/11-decision no: 19/206). All ED patients who underwent a CECT examination and were consequently admitted to an inpatient service in the period from 1 November 2016 to 31 March 2019 were screened retrospectively using the electronic patient management system (FONET®, Information Technology Incorporation, Turkey) of the Gulhane Training and Research Hospital to be enrolled in the study. All patients who underwent a CECT procedure and who did not meet the exclusion criteria were included in the study. Patients were selected retrospectively by the investigators. According the inclusion criteria established on presentation of the study for ethics committee approval, patients who were younger than 18 years of age; who did not undergo a CECT procedure; who were previously diagnosed with acute, acute on chronic renal failure (ACRF), or chronic renal failure at the time of admission; who had no CBC tests within 24 hours before and after the procedure, and who had no SCr levels tested within 72 hours after the procedure, were excluded from the study (Figure 1). Informed consent was waived by the non-interventional ethical committee due to the retrospective design of the study.

Age, sex, baseline CBC parameters obtained on admission to the ED, and the derived values obtained from CBC were obtained, including NLR, the mean platelet volume-to-platelet count ratio (MPV/PLT), and the lymphocyte to monocyte ratio (LMR). The CBC parameters and the NLR, platelet-to-lymphocyte ratio (PLR), MPV/PLT, LMR values obtained in the first 24 hours after CECT were recorded. Of the patients admitted to inpatient services, the SCr levels obtained from the blood samples collected within the 24-72 hours after CECT were recorded. Patients with SCr levels increasing by 25% or more and patients with increments of 0.5 mg/dL in SCr levels within 0-72 hours compared to the baseline levels obtained before CECT were identified as the CIN Group. Patients without CIN were defined as the non-CIN group (Figure 1). Only the serum creatinine levels were used, while the urine output values were not evaluated for the purposes of this study because the precise determination of urine output was expected to be challenging in ED patients.

Primary and Secondary Objectives

We defined the primary outcome of our study as the early detection of CIN within the first 24 hours after CECT. For this purpose, the CBC parameters; NLR, PLR, MPV/PLT, and LMR values obtained before and within the first 24 hours after CECT were compared between the CIN and non-CIN groups.

The secondary outcome of the study was defined as the ability to predict the likelihood of developing CIN based on the CBC parameters, NLR, PLR, MPV/PLT, and LMR, obtained before the patient underwent CECT. For this purpose, CIN and non-CIN groups were compared by analyzing their pre-CECT values.

Power analysis

The sample size was calculated with an alpha value of 0.05, 80% power, an enrollment ratio of 1, and NLR values of 2.14 in group 1 (control group or the non-CIN group) and 6 in group 2 (CIN group), indicating that 52 patients in either group for a total of 104 participants should be included in the study (13).

Laboratory Analysis

In the defined patient screening period of the study; the creatinine values were analyzed with a Beckman Coulter AU680 chemistry analyzer (Beckman Coulter, Miami, FL, USA) and Beckman Coulter AU480 chemistry analyzer (Beckman Coulter, Miami, FL, USA), and the CBC was analyzed with CBC Sysmex XN-1000 (Sysmex America, Inc., Lincolnshire, IL, USA) and Beckman Coulter, UniCel DxH800 (Beckman Coulter, Miami, FL, USA).

CECT Procedure

No treatment protocol was applied for patients undergoing CECT relative to the administration of any

specific pharmacological agents to reduce a potential risk of developing CIN. The anatomic site examined with CECT, the CECT technique, and the content and volume of contrast agents used for each patient were recorded (Table 1). All CECT procedures were performed using water soluble, non-ionic, and low osmolar (915 mosm/kg) iodinated contrast agents (Omnipaque ™, Kopaq ™, or Biemexol ™. Dosage range: min; 75 cc – max; 130 cc). The tests were performed using a 320-detector CT device and the images were reconstructed with a thickness of 0.5 mm.

Statistical Analysis

Statistical analyses were performed using SPSS Statistics for Windows, version 18.0 (SPSS Inc., Chicago, IL. USA). The categorical variables were summarized with descriptive statistics and were presented as numbers and percentages. Numerical variables were summarized and listed as mean ± standard deviation. The Kolmogorov Smirnov test was used to test whether the variables were distributed normally. The Wilcoxon signed-rank test was used as a non-parametric test for analyzing variables not conforming to a normal distribution. The Pearson correlation test was used to analyze the significance of a correlation between two parameters. A statistical significance was considered at a p-level of < 0.05. Ability of the assay to discriminate between patients with and without post-CECT SCr increase was evaluated by receiver-operating characteristic (ROC) curve analysis. An optimum cut-off value was defined for NLR providing the highest sensitivity and specificity.

RESULTS

A total of 594 patients who underwent CECT examination were identified. A total of 301 patients were excluded who did not meet the inclusion criteria. Thus, a total of 293 patients were included in the study (Figure 1). The mean age of the patients was 62.00 ± 1.00 years; 55.6% (n = 163) were males, and 44.4% (n = 130) were females.

Of the patients enrolled, 18.77% (55/295) were in the CIN group and 81.23% (238/295) were in the non-CIN group. The distribution of the comorbid diseases and anatomic-region-specific CECT examinations of the patients are presented in Table 1.

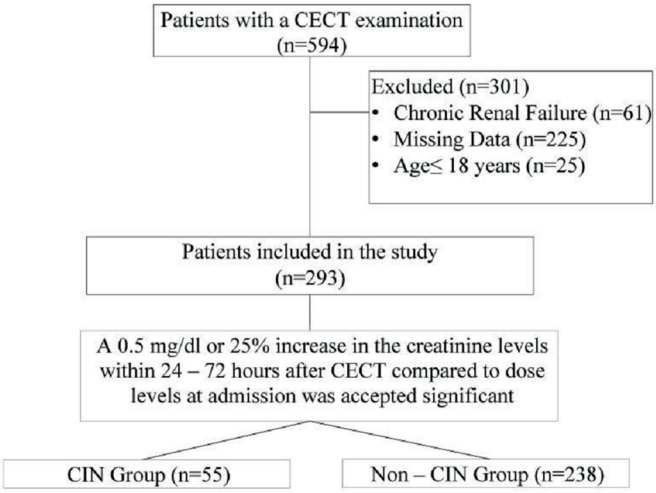

Figure 1. Study Flowchart

In the patients developing CIN, the NLR, PLR, MPV/PLT, and LMR values obtained with the 24-hour periods before and after the CECT procedure were compared; the post-CECT values were significantly higher (p = 0.001, 0.000, 0.038, and 0.000 respectively) (Table 2).

Table 1. Demographic and angiographic data of patients with contrast-enhanced computerized tomography

	Total n (%)	Non-CIN Group n (%)	CIN Group n (%)	p*
Number of Patients	293 (100)	238 (100)	55 (100)	
Age	62 ± 1	62 ± 1	65 ± 2	0.204*
Male Gender	163 (55.63)	129 (54.20)	34 (61.81)	0.306**
Hypertension	97 (33.10)	81 (34.03)	16 (29.09)	0.296**
Diabetes Mellitus	86 (29.35)	69 (29.99)	17 (30.90)	0.448**
CAD#	51 (17.40)	40 (16.80)	11 (20.00)	0.346**
Heart failure	34 (11.60)	27 (11.34)	7 (12.72)	0.463**
Atrial fibrillation	11 (3.75)	8 (3.36)	3 (5.45)	0.341**
COPD##, asthma	36 (12.28)	28 (11.76)	8 (14.54)	0.356**
Malignancy	24 (8.19)	19 (7.98)	5 (9.09)	0.486**
Alzheimer's dementia	17 (5.80)	15 (6.30)	2 (3.63)	0.349**
CT angiography of the abdominal aorta and lower extremities bilaterally	31 (10.58)	26 (10.92)	5 (9.09)	0.454**
CT angiography of the brain and arteries in the neck	109 (37.20)	88 (36.97)	21 (38.18)	0.492**
CT angiography of the pulmonary arteries	103 (35.15)	83 (34.87)	20 (36.36)	0.475**
CT angiography of the thoracic and abdominal aorta	50 (17.06)	42 (17.64)	8 (14.54)	0.372**

*Mann-Whitney U Test , **Chi-Square test, # CAD; coronary artery disease, ## COPD; chronic obstructive pulmonary disease

Table 2. Complete blood count results before and after the contrast-enhanced computerized tomography in the contrast-induced nephropathy group

	Before CECT (mean ± standard deviation)	After CECT (mean ± standard deviation)	p*
White Blood Cell (x10^3)	12.32 ± 0.73	13.37 ± 0.95	0.241
Red Blood Cell (x10^3)	4.54 ± 0.12	4.28 ± 0.12	0.002
Hemoglobin (g/L)	12.95 ± 0.32	12.16 ± 0.32	0.000
Hematocrit (%)	38.95 ± 0.91	36.66 ± 0.96	0.001
Platelet x10^3	241.31 ± 13.02	219.13 ± 13.01	0.007
Mean corpuscular volume (fL)	86.74 ± 1.18	86.30 ± 1.12	0.957
Mean corpuscular hemoglobin (pg)	28.82 ± 0.45	28.67 ± 0.44	0.236
Mean corpuscular hemoglobin concentration (g/L)	33.18 ± 0.19	33.17 ± 0.18	0.915
Red blood cell distribution width-SD (fL)	46.62 ± 1.07	46.70 ± 1.01	0.068
Red blood cell distribution width-CV (%)	15.16 ± 0.29	15.20 ± 0.28	0.119
Platelet larger cell ratio (%)	30.45 ± 1.73	31.56 ± 2.0	0.523
Mean platelet volume (fL)	9.48 ± 0.21	9.59 ± 0.21	0.230
Plateletcrit (%)	0.27 ± 0.02	0.26 ± 0.02	0.504
Platelet distribution width (%)	12.97 ± 0.59	13.58 ± 0.75	0.236
Neutrophil (x10^3)	9.20 ± 0.73	11.11 ± 0.88	0.008
Neutrophil (%)	72.16 ± 2.01	81.83 ± 1.35	0.000
Lypmphocyte (x10^3)	2.22 ± 0.22	1.29 ± 0.12	0.000
Lypmphocyte (%)	19.20 ± 1.76	11.29 ± 1.01	0.000
Monocyte (x10^3)	0.72 ± 0.05	0.75 ± 0.06	0.869
Monocyte (%)	6.17 ± 0.34	6.03 ± 0.40	0.873
Eosinophil (x10^3)	0.11 ± 0.02	0.07 ± 0.03	0.003
Eosinophil (%)	1.16 ± 0.27	0.54 ± 0.20	0.000
Basophil (x10^3)	0.07 ± 0.01	0.03 ± 0.01	0.001
Basophil (%)	0.58 ± 0.11	0.29 ± 0.03	0.000
Immature granulocytes (x10. ^3/u)	0.20 ± 0.28	0.13 ± 0.04	0.850
Immature granulocytes (%)	1.66 ± 0.68	0.97 ± 0.37	0.754
NLR	7.01 ± 0.96	11.85 ± 1.56	0.001
LMR	0.42 ± 0.04	0.25 ± 0.02	0.000
MPV/PLT R	0.06 ± 0.02	0.07 ± 0.01	0.038
PLR	154.52 ± 15.25	226.68 ± 23.73	0.000

*Wilcoxon signed-rank test

In addition, the comparison of the NLR, PLR, MPV/PLT, and LMR values obtained with the 24-hour periods before and after the CECT in the non-CIN group were significantly higher in the post-CECT group (p = 0.024, 0.001, 0.000, and 0.000 respectively) (Table 3).

No statistically significant differences were found in the pre-CECT values of NLR, PLR, MPV/PLT, and LMR between the CIN and non-CIN groups (p = 0.262, 0.360, 0.248, and 0.134 respectively) (Table 4).

Table 3. Complete blood count parameters obtained before and after contrast-enhanced computerized tomography in the group with no contrast-induced nephropathy

	Before CECT (mean ± standard deviation)	After CECT (mean ± standard deviation)	p*
White Blood Cell (x10^3)	11.31 ± 0.52	10.54 ± 0.40	0.000
Red Blood Cell (x10^3)	4.65 ± 0.05	4.45 ± 0.05	0.000
Hemoglobin (g/L)	13.30 ± 0.15	12.72 ± 0.14	0.000
Hematocrit (%)	39.86 ± 0.40	38.10 ± 0.38	0.000
Platelet x10^3	259.39 ± 6.86	248.03 ± 6.42	0.000
Mean corpuscular volume (fL)	86.35 ± 0.54	85.76 ± 0.66	0.000
Mean corpuscular hemoglobin (pg)	28.73 ± 0.22	30.01 ± 1.16	0.880
Mean corpuscular hemoglobin concentration (g/L)	33.22 ± 0.11	33.61 ± 0.18	0.006
Red blood cell distribution width-SD (fL)	45.95 ± 0.55	46.04 ± 0.55	0.108
Red blood cell distribution width-CV (%)	15.04 ± 0.20	15.05 ± 0.17	0.980
Platelet larger cell ratio (%)	28.94 ± 0.78	29.92 ± 0.90	0.063
Mean platelet volume (fL)	9.32 ± 0.09	9.88 ± 0.40	0.000
Plateletcrit (%)	0.27 ± 0.01	0.26 ± 0.01	0.002
Platelet distribution width (%)	12.20 ± 0.24	12.71 ± 0.28	0.018
Neutrophil (x10^3)	7.80 ± 0.28	7.76 ± 0.30	0.134
Neutrophil (%)	69.48 ± 0.93	71.91 ± 0.96	0.245
Lypmphocyte (x10^3)	2.10 ± 0.09	1.62 ± 0.06	0.000
Lypmphocyte (%)	21.32 ± 0.80	18.40 ± 0.73	0.087
Monocyte (x10^3)	0.72 ± 0.02	0.76 ± 0.04	0.925
Monocyte (%)	7.08 ± 0.21	7.55 ± 0.26	0.014
Eosinophil (x10^3)	0.22 ± 0.08	0.15 ± 0.04	0.002
Eosinophil (%)	1.68 ± 0.25	1.95 ± 0.60	0.122
Basophil (x10^3)	0.05 ± 0.00	0.03 ± 0.00	0.000
Basophil (%)	0.56 ± 0.03	0.60 ± 0.16	0.000
Immature granulocytes (x10. ^3/u)	0.09 ± 0.01	0.09 ± 0.02	0.111
Immature granulocytes (%)	0.75 ± 0.10	0.73 ± 0.12	0.215
NLR	5.80 ± 0.40	7.29 ± 0.49	0.024
LMR	0.35 ± 0.02	0.26 ± 0.01	0.000
MPV/PLT R	0.04 ± 0.00	0.05 ± 0.00	0.000
PLR	176.25 ± 9.90	209.96 ± 11.62	0.001

*Wilcoxon signed-rank test

Table 4. Comparison of the complete blood count values between the two study groups before contrast-enhanced computerized tomography

	CIN (mean ± standard deviation)	non-CIN (mean ± standard deviation)	p*
NLR	7.01 ± 0.96	5.80 ± 0.40	0.262
LMR	0.42 ± 0.04	0.35 ± 0.02	0.134
MPV/PLT R	0.06 ± 0.02	0.04 ± 0.00	0.248
PLR	154.52 ± 15.25	176.25 ± 9.90	0.360

*Mann-Whitney U Test

The comparison of the post-CECT of PLR, MPV/PLT, and LMR values revealed no statistically significant differences between the CIN and non-CIN groups (p = 0.283, 0.128, and 0.792 respectively). The post-CECT NLR values were significantly higher in the CIN group compared to the non-CIN group (11.85 ± 1.56 vs 7.29 ± 0.49; p = 0.000) (Table 5).

The optimum cut-off value of the NLR providing the highest sensitivity and specificity levels was determined to be 8, yielding 52.73% sensitivity and 69.75% specificity (Table 6, Figure 2).

Table 5. Comparison of the complete blood count values between the two study groups after contrast-enhanced computerized tomography

	CIN (mean ± standard deviation)	non-CIN (mean ± standard deviation)	p*
NLR	11.85 ± 1.56	7.29 ± 0.49	0.000
LMR	0.25 ± 0.02	0.26 ± 0.01	0.792
MPV/PLT R	0.07 ± 0.01	0.05 ± 0.00	0.128
PLR	226.68 ± 23.73	209.96 ± 11.62	0.283

*Mann-Whitney U Test

Table 6. Comparison of the two study groups after contrast-enhanced computerized tomography for neutrophil-to-lymphocyte ratio at a cut-off point of 8

	CIN n (%)	non-CIN (%)	p*
NLR ≤ 8	26 (47.3)	166 (69.7)	
NLR > 8	29 (52.7)	72 (30.3)	0.002
Total	55 (100)	238 (100)	

*Chi-square test

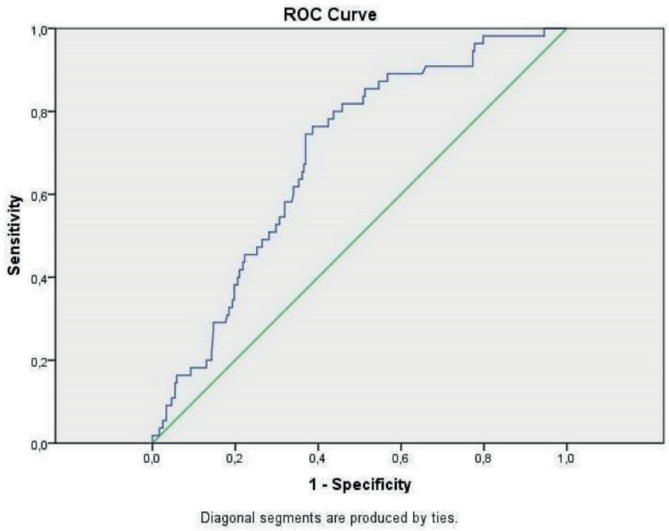

Figure 2. Receiver-operating characteristic (ROC) curve analysis. An increase in the 24 hour post-CECT NLR value >8 significantly predicts an absolute increase in SCr levels of ≥25% or ≥0.5 mg/dL after contrast exposure, with a sensitivity of 52.73% and a specificity of 69.75% (AUC; 0.692, 95% CI; 0.68 to 1.74, p = 0.002).

DISCUSSION

CIN is listed among factors causing hospital-acquired acute renal failure, significantly increasing the mortality, morbidity, and costs (14). CIN is diagnosed when an increase in creatinine values observed in blood samples collected from the patients within 24–72 hours of the follow-up period (4). Significantly higher NLR values were found in the first 24-hours after CECT in the CIN group compared to the non-CIN group, which suggests that the NLR may allow physicians to predict a potentially developing CIN in the first 24 hours rather than reaching a final diagnosis in the first 72 hours. In our study, no statistically significant differences were found in NLR values between the CIN and non-CIN groups before the contrast agent administration, strengthening the appropriateness of the groups used to compare the NLR values and to determine their predictive value in estimating a diagnosis of CIN in the first 24 hours after CECT.

Creatinine values increased in the non-CIN group in our study by less than 25%. In this respect, we considered that statistically significantly high NLR values found after contrast administration in the non-CIN group were related to the exposure of the patients to the contrast media.

In estimating the potential to develop CIN, to the best of our knowledge, there have been no other studies in the literature evaluating the predictive values of CBC parameters and the ratios derived from CBC results obtained within the first 24 hours after the CECT, which are frequently used as a diagnostic tool in the ED. In this respect, we consider our study results unique.

Yin et al. (4) found a 13.8% rate of developing CIN after a CECT procedure (4). Similar to their findings, we found a 18.77% rate of CIN in the post-CECT period. Given the

patient populations were similar in age and comorbid diseases, we consider that our study showed comparable CIN development rates with those of Yin et al. (4).

Abu Alfeilat et al. (15) and Kurtul et al. (16) reported NLR as a known biomarker in making the diagnosis of AKI. The significantly high NLR values in the first 24 hours after contrast agent administration in our study were consistent with those of the aforementioned studies. We appreciate that this similarity is related to the fact that CIN is also a cause of AKI and inflammatory processes play a role in the pathophysiology of both conditions.

Demircelik et al. (17) investigated the relationship between CIN and PLR in patients, who underwent percutaneous coronary intervention (PCI). The authors reported that high pre-PCI PLR values (the optimal cut-off point for PLR was defined to be 148.3) found in their study could be used as an independent usable biomarker for predicting the development of CIN after PCI. On closer evaluation, in the Demircelik et al. (17) study no differences were reported between the platelet counts of the CIN and non-CIN groups (248 ± 70.8×103/mm3 and 250.3 ± 66.3×103/mm3, respectively; p = 0.82) but they could not clarify whether the high PLR value resulted from a high platelet count or a low lymphocyte count. However, after examining the number of platelets reported in their study, one can argue that the statistically significant difference in the PLR ratio between the two groups resulted from the low lymphocyte count in the CIN group. Contrary to the study results reported by Demircelik et al. (17), no differences were observed in the admission PLR values found before CECT between the two groups in our study, suggesting that PLR cannot be used as a predictive marker for CIN. In our study, all non-CIN and CIN group patients had normal SCr values before the CECT procedure. In contrast, the SCr mean value of the CIN group cases in Demircelik et al.'s (17) study was above the normal range (1.4 ± 0.37 mg/dL) before the procedure. Thus, we considered that the PLR value in our study was different from their study in terms of not being able to predict the development of CIN.

The intragroup comparisons before and after CECT in our study revealed significantly higher PLR values after CECT. However, the lack of a statistically significant difference in the PLR values after CECT between the two groups in our study indicated that an increase in PLR acts as an inflammatory biomarker, but unlike NLR, it cannot be used as a biomarker to make an early diagnosis of CIN.

MPV and LMR are new biomarkers used to demonstrate inflammatory processes (18,19). To the best of our knowledge, no studies are currently available in the literature, investigating the predictive values of MPV/PLT and LMR for identifying CIN accompanied by inflammatory processes. Our study results indicated that these parameters could not be used as predictive biomarkers for CIN independently or adjunctively to make a diagnosis.

Intragroup comparisons in our study revealed significantly lower platelet and lymphocytes counts after the procedure compared to the pre-CECT levels, suggesting that these findings might result from exposure to the contrast agent. The observed decrease in the lymphocyte count was much higher compared to that observed in the platelet count after the contrast agent administration in our study, indicating why post-CECT PLR values were found to be higher compared than the pre-CECT levels in the intragroup comparisons. However, this suggests that PLR cannot be used for the early detection of CIN patients. Since this was a retrospective study, conducted at a single center, and only the data obtained in the first 72 hours after the CECT was evaluated, patients developing CIN after this 72-hour follow-up period could have been missed. However, we consider that the number of potentially missed patients developing CIN in the post-CECT period is acceptable because the diagnostic criteria in the literature has been defined for the first 72 hours.

Based on the data we obtained from our study, we propose that patients discharged from the ED after any kind of CECT procedure in our hospital should routinely return to the hospital to check CBC parameters as well as SCr values on the first and third days after discharge. We also warn that the CBC parameters taken in the first 24 hours following the CECT procedure should not be ignored when they are transferred to another clinic from the ED. The spread of these practices may reduce the risk of CIN after CECT procedures.

Limitations

This was a retrospective and single center study, which was subject to the limitations in generalizability with this research design. Further, we could neither evaluate the SCr levels after 72 hours of admission nor the patient prognosis post-discharge.

CONCLUSION

To the best of our knowledge, this is the first study to evaluate the predictive value of NLR for early detection of CIN after a CECT procedure. We have demonstrated that an increased NLR level after a CECT procedure is associated with the development of CIN. We believe that the use of NLR in the ED as a predictive parameter can significantly improve the diagnostic process, favorably acting on the prognosis of patients developing CIN. The authors recommend that clinicians evaluate the CIN risk of the patients before the CECT procedure with laboratory findings as well as CBC parameters. We also recommended future studies be conducted in larger prospective cohorts to assess the value of CBC parameters in the prediction of CIN.

Conflict of interest: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical approval: This retrospective non-interventional study was approved by the Gulhane Ethics Board of the University of Health Sciences on November 2019 (Committee IRB approval number: 2019/11-decision no: 19/206).

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