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Pre-Contrast Hemogram-Derived Indices as Predictors for Contrast-Induced Acute Kidney Injury After Percutaneous Coronary Intervention

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ABSTRACT

Background: Percutaneous coronary intervention (PCI) may be complicated by contrast-induced acute kidney injury (CI-AKI). The current study aimed to evaluate the pre-contrast hemogram-derived indices as early predictors for CI-AKI after PCI. **Methods:** A case-control study involved 54 patients who underwent PCI, divided into Group I (27 patients with no CI-AKI), and Group II (27 patients with CI-AKI). After the history taking, examination and routine laboratory investigations, the pre-contrast hemogram-derived indices were calculated; neutrophil / lymphocyte ratio (NLR), platelet / lymphocyte ratio (PLR), neutrophil / platelet ratio (NPR), monocyte / lymphocyte ratio (MLR), eosinophil / lymphocyte ratio (ELR), and systemic immune-inflammation (SII) index (platelet count × (neutrophil count / lymphocyte count).

Results: There were significantly higher pre-contrast leucocytes, neutrophils, NLR ($p<0.001$) (HS), MLR, PLR, NPR, ELR, and SII in group II than in group I ($p<0.05$). The ROC analysis showed a significant predictive value for pre-contrast NLR (92.8% sensitivity & 51.3% specificity), NPR (42.8% & 86.4%), and SII (53.5% & 81.1%), MLR (85.7% & 43.2%), and ELR (50% & 78.3%). The odds ratios (OR) for pre-contrast hemogram-induced indices as predictors for CI-AKI, with confidence interval (CI) 95% were as follows; NLR OR (2.12), CI (0.71-6.31); PLR OR (2.11), CI (0.71-6.26); SII OR (2.96), CI (0.96-9.11); MLR OR (2.89), CI (0.95-8.72); ELR OR (3.4), CI (1.11-10.4); NPR OR (2.5), CI (0.82-7.53); RDW OR (2.12), CI (0.71-6.31). **Conclusion:** Hemogram-derived indices including pre-contrast SII, NLR, NPR, and MLR may be useful early predictors for CI-AKI after PCI.

Keywords: CI-AKI; NLR; NPR; SII; MLR; PLR; ELR.

INTRODUCTION

Acute kidney injury (AKI) is a sudden, and often reversible reduction in kidney function [(1).] AKI is defined as a rise in serum creatinine (Cr) levels by 0.3 mg/dl or more within 48 hours, or its increase ≥1.5 times the baseline levels within the prior 7 days, or urine volume <0.5 ml / kg /h for 6 hours [(2).] When percutaneous coronary intervention (PCI) for acute coronary syndrome is complicated with AKI, this predicts worse outcomes, and the athero-embolic renal disease has a higher likelihood of long-term renal replacement therapy [(3).] A post-PCI increase of 0.3 mg/dL or more in serum Cr within 48 hours predicts mortality and major adverse cardiovascular events most accurately [(4).] Contrast-associated AKI (CA-AKI) may result from ischemia of the renal medulla or tubules, vasoconstriction, or from cholesterol embolism [(5).] Serum Cr is a delayed marker for contrast-induced AKI (CI-AKI), so newer biomarkers are used for early diagnosis [(6).] Platelets and leucocytes play a role in thrombogenesis, and inflammation, thus hemogram-derived ratios such as the neutrophil / lymphocyte ratio (NLR), neutrophil / platelet ratio (NPR), platelets / lymphocyte ratio (PLR), in addition to systemic immune inflammation index (SII) could help as predictors for patients at risk for Intensive Care Unit (ICU) admission [(7).] The NLR is easily calculated from the differential white blood cell (WBC) count and is an early biomarker for AKI [(8).] The SII is calculated as neutrophil count × platelet count / lymphocyte count and reflects the inflammatory and immune status of patients [(9).] Preoperative high SII index was found to be an independent risk factor of CI-AKI following PCI [(10).] The study was conducted to evaluate the role of pre-contrast hemogram-derived indices as predictors for CI-AKI after PCI.

METHODS

A case-control study that was conducted from July 2022 to December 2022, at the Intensive Care Unit, Zagazig University Hospitals, Egypt, and included 54 patients who had PCI; divided into; Group 1: (27 patients with no CI-AKI), and Group 2: (27 patients with CI-AKI).

Inclusion Criteria:

Patients >18 years, who underwent diagnostic or therapeutic PCI, using iso-ionic iodinated contrast-media 200-250 ml. They were well-hydrated with intravenous saline 0.9% for 12 hours after the PCI. Patients with CI-AKI were diagnosed according to the KDIGO criteria as a rise in serum Cr 0.3 mg/dl or more within 48 hours, or its rise 1.5 times or more the baseline level within the prior 7 days, or urine volume <0.5 ml/kg/h for 6 hours [2].

Exclusion Criteria:

Patients <18 years, AKI before PCI, hypovolemia, congestive heart failure, kidney transplantation, hemodialysis, nephrotoxic agents, exposure to contrast agent 30 days before the PCI, use of nephrotoxic

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medications after PCI, major trauma, surgery, or active infection were excluded.

Procedures:

All participants were subjected to the following: History taking, examination, and laboratory investigations including blood urea nitrogen (BUN), serum creatinine (Cr), eGFR, liver function tests, lipid profile, serum uric acid, albumin, fasting blood sugar (FBS), serum electrolytes, arterial blood gases (ABG), cardiac enzymes, C-reactive protein (CRP). Markers to exclude infection were made including tests for hepatitis-C, hepatitis-B, and HIV, ferritin, urine analysis, and stool analysis. Complete blood count (CBC) was done before PCI with the calculation of hematological ratios; neutrophil/ lymphocyte ratio (NLR), platelet/ lymphocyte ratio (PLR), neutrophil/ platelet ratio (NPR), monocyte/ lymphocyte ratio (MLR), eosinophil/ lymphocyte ratio (ELR), and systemic immune-inflammation (SII) index (platelet count × (neutrophil count/ lymphocyte count). Kidney functions and hematological ratios were repeated 48 after PCI. Radiological investigations: chest x-ray, echocardiography, ECG, pelviabdominal ultrasound, or other investigations were made as needed.

Ethics approval:

The Institutional Review Board has reviewed and assessed the study (IRB#9682/3-8-2022) regarding the potential risks and benefits based on the World Medical Association's code of ethics for experimentation on humans; the Helsinki Declaration. Patients' written informed consent was also obtained.

Statistical Analysis:

Data were collected and analyzed using SPSS 22. Continuous data were presented by mean±SD and categorial data by the count and percentage. An unpaired t-test was used for the 2 groups comparison, a paired t-test for comparing pre- and post-contrast data in same groups, and a Chi-Square test for categorical data comparison. Parametric data or Normally distributed were represented as mean ± SD, and analyzed using independent t test. Non parametric data or skewed data were represented as median (Range), and analyzed using Mann Whitney test. Paired non parametric data were analyzed using Wilcoxon test. Pearson's correlation coefficient was used for the association between data. Receiver operating characteristic (ROC) curve analysis was made for the cutoff values, the area under the curve (AUC), sensitivity, and specificity. Then multiple regression analysis was made for the independent factors for predicting CI-AKI, and Odds ratios were calculated. P-value <0.05 was considered significant (S), p-value <0.001 highly significant (HS), and p-value >0.05 non-significant (NS).

RESULTS

As regards the demographic data, no statistically significant differences were found between the 2 studied groups (p>0.05) (NS) (Table 1). Regarding laboratory investigations, statistically significantly higher serum triglycerides (p<0.0001) (HS), fasting blood sugar, serum phosphorous, and CRP and statistically significantly lower serum sodium were found in group II than in group I (p<0.05) (S), with no statistically significant differences as regards other parameters (p>0.05) (NS) (Table 1).

As regards the pre-contrast hematological values and indices, a highly statistically significantly higher WBCs, neutrophils, and NLR (p<0.0001) (HS), a statistically significantly higher MLR, PLR, NPR, ELR, and SII (p<0.05) (S) were found in group II than in group I, with no statistically significant differences as regards other parameters (p>0.05) (NS) (Table 2).

Regarding the 48-hours post-contrast kidney function tests, a statistically significantly higher serum creatinine, and BUN and a statistically significantly lower eGFR were found in group II than in group I (p<0.0001) (HS) (Table 3). In addition, patients in group II, had a statistically significantly higher serum creatinine and BUN (p<0.05) (S), and a highly statistically significantly lower eGFR (p<0.0001) (HS), 48-hours post-contrast than pre-contrast (Table 4).

The ROC analysis for the validity of the hematological indices for predicting CI-AKI showed that there was a statistically significant predictive value (p<0.05), for pre-contrast ELR (50% sensitivity & 78.3% specificity), MLR (85.7% sensitivity & 43.2% specificity), NPR (42.8% sensitivity & 86.4% specificity) and SII (53.5% sensitivity & 81.1% specificity), with no significant predictive value for PLR and RDW (p>0.05) (Table 5).

As regards the multiple regression analysis for hematological indices as predictors for CI-AKI, the SII had a highly statistically significant values (p<0.001), and NPR and ELR had a statistically significant value (p<0.05) for predicting CI-AKI. No statistically significant values were found for MLR, NLR, PLR, or RDW for predicting CI-AKI (Table 6).

Regarding the multiple regression analysis for other pre-contrast parameters for predicting CI-AKI, there were high statistically significant predictive value (p<0.001) for pre-contrast serum albumin, ALT, AST, triglycerides and CRP, and a statistically significant predictive value (p<0.05) for pre-contrast serum phosphorous, cholesterol, FBS, potassium, ferritin, CK-MB, creatinine, and eGFR. No statistically significant predictive value (p>0.05) was found for pre-contrast calcium, LDL, uric acid, sodium, Troponin T, BUN, and creatinine (Table 6).

The odds ratios (OR) for pre-contrast hemogram-induced indices as predictors for CI-AKI, with confidence interval (CI) 95% were as follows; NLR OR (2.12), CI (0.71-6.31); PLR OR (2.11), CI (0.71-6.26); SII OR (2.96), CI (0.96-9.11); MLR OR (2.89), CI (0.95-8.72);

ELR OR (3.4), CI (1.11-10.4); NPR OR (2.5), CI (0.82-7.53); RDW OR (2.12), CI (0.71-6.31) (Table 7).

Table (1): Comparison of demographic data and pre-contrast laboratory tests between the 2 studied groups.

Variable		**(Group I) (n=27)**		**(Group II) (n=27)**		**t-test/U**	**P**
Sex	**Male No (%)**	18	(66.5%)	17	(62.9%)	X^2 1.2	>0.05
	Female No (%)	9	(33.5%)	10	(37.1%)		
Age (Years) Mean±SD		58.6± 9.8		61.6± 12.4		T=1.1	>0.05
Co-morbidities		No	%	No	%	X^2 1.4	>0.05
Diabetes Mellitus		17	62%	21	77%		
Hypertension		20	74%	24	88.8%		
Hepatitis C		5	18.5	4	14.8		
Hepatitis B		2	7.4	3	11.11		
ALT (U/L) Median Range		26 (11.5-96.1)		27.9 (9.3-745.9)		U=331.5	0.56
AST (U/L) Median Range		45 (16-237.8)		43.5 (16-503)		U=309.5	0.34
S. Albumin (g/dl) Mean±SD		3.8±0.7		3.7±0.5		T=-0.6	>0.05
HDL (mg/dl) Mean±SD		55.1±3.7		47.01±3.1		T=-1.2	>0.05
LDL (mg/dl) Mean±SD		131.9±29.9		122.4±9.4		T=-0.8	>0.05
S. Cholesterol (mg/dl) Mean±SD		187.5±41.08		169.4±6.08		T=-0.6	>0.05
S. Triglycerides (mg/dl) Mean±SD		160.3±8.2		286.2±35.2		T=-4.6	**<0.001****
S. Uric acid (mg/dl) Mean±SD		5.88±1.93		6.13±1.38		T=0.4	>0.05
Fasting blood sugar (mg/dl) Mean±SD		153.9±4.6		173.1±38.6		T=1.8	**<0.05***
S. Potassium (mmol/l) Mean±SD		4.15±0.54		4.26±0.72		T=0.6	>0.05
S. Sodium (mmol/l) Mean±SD		137.05±3.7		133.7±5.1		T=-3.0	**<0.05***
S. Calcium (mg/dl) Mean±SD		8.71±1.27		8.66±0.8		T=-0.1	>0.05
S. Phosphorous (mg/dl) Mean±SD		3.35±0.21		4.14±1.28		T=2.5	**<0.05***
CK-MB (IU/l) Median Range		24.46 (0.87-300)		7.72 (0.68-187.3)		U=318	0.42
Troponin-T (ng/l) Median Range		12.4 (0.8-193.1)		1267 (7.89-10000)		U=291	0.2
CRP (mg/dl) Median Range		12.4 (0.8-193.1)		38.3 (1.6-556.3)		U=237	**0.02***
S. Ferritin (ug/l) Median Range		80 (48-250.1)		68 (42-529.6)		U=299.5	0.26
S. Creatinine (mg/dl) Median Range		0.92 (0.01-3.4)		1.16 (0.36-8.91)		U=297.5	0.65
eGFR (ml/min) Mean±SD		66.8 ± 6.8		60.97± 3.3		T=-0.7	>0.05
BUN (mg/dl) Mean±SD		25.73±7.12		32.49±2.02		T=1.2	>0.05

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** High statistically significant difference. * Statistically significant difference. X2 Chi-Squared test.
Parametric data were represented as mean ± SD, and analyzed using independent t test.
Non parametric data were represented as median (Range), and analyzed using Mann Whitney test.

Table (2) Comparison between the 2 studied groups as regards pre-contrast hematological values and indices.

Variable	(Group I) (n=27)	(Group II) (n=27)	t-test/U	P
WBCs (x10³/µL) Mean±SD	9.91 ± 1.11	12.86 ± 2.68	T=2.8	**<0.001****
Neutrophils (x10³/µL) Mean±SD	7.08 ± 1.01	10.2 ±2.17	T=3.4	**<0.001****
Lymphocytes (x10³/µL) Mean±SD	1.94± 0.2	1.67 ± 0.2	T=-1.1	>0.05
Monocytes (x10³/µL) Mean±SD	0.75 ± 0.12	0.81 ± 0.13	T=0.7	>0.05
Eosinophils (x10³/µL) Median Range	0.1 (0.1-0.4)	0.1 (0.1-0.95)	U=343	0.65
Platelets (x10⁹L) Mean±SD	243.8 ± 8.8	257.1 ± 8.5	T=0.7	>0.05
RDW (%) Mean±SD	13.9 ± 2.89	14.48± 2.18	T=0.8	>0.05
NLR Median Range	3.7 (0.75-14.5)	5.62 (1.96-32)	U=175	**<0.001****
MLR Median Range	0.37 (0.12-0.92)	0.54 (0.11-2)	U=238	**0.02***
PLR Median Range	127.3 (47.7-285)	166 (58.8-980)	U=240.5	**0.03***
NPR Median Range	0.03 (0.01-0.07)	0.039 (0.01-0.07)	U=238	**0.02***
ELR Median Range	0.06 (0-1.2)	0.1 (0.02-0.55)	U=232	**0.02***
SII Median Range	960 (118.3-2479.5)	1490.5 (312-9464)	U=209	**0.007***

** High statistically significant difference. * Statistically significant difference.
Parametric data were represented as mean ± SD, and analyzed using independent t test.
Non parametric data were represented as median (Range), and analyzed using Mann Whitney test.

Table (3) Comparison between the 2 studied groups as regards the 48-hours post-contrast kidney function tests.

Variable	(Group I) (n=27)	(Group II) (n=27)	t-test	P
S. Creatinine (mg/dl) Mean±SD	1.16 ± 0.24	2.36 ± 0.19	4.08	**<0.001****
eGFR (ml/min) Mean±SD	70.53 ± 4.43	39.13± 3.84	-6.25	**<0.001****
BUN (mg/dl) Mean±SD	23.75±1.37	48.96±8.44	4.4	**<0.001****

** High statistically significant difference. eGFR (estimated glomerular filtration rate).
Parametric data were represented as mean ± SD, and analyzed using independent t test.

Table (4) Comparison between the pre-contrast and the 48-hours post-contrast kidney function tests in Group II.

Variable	Pre-contrast Group II	48-hours post-contrast Group II	t-test	P
S. Creatinine (mg/dl) Mean±SD	1.57 ± 0.62	2.36 ± 0.19	2.05	**<0.05***
eGFR (ml/min) Mean±SD	60.97± 3.3	39.13± 3.84	-3.9	**<0.001****
BUN (mg/dl) Mean±SD	32.49±6.02	48.96±8.44	2.2	**<0.05***

** High statistically significant difference. eGFR (estimated glomerular filtration rate).
Parametric data were represented as mean ± SD, and analyzed using independent t test.

Table (5) Receiver Operating Characteristic (ROC) analysis for pre-contrast hematological ratios as predictors of CI-AKI.

Variable	AUC	SD	CI 95%	Cutoff	Sens	Spec	p
ELR	0.64	0.07	0.51 to 0.75	**0.08**	50%	78.3%	**0.05*<**
MLR	0.64	0.07	0.51 to 0.75	**0.5**	85.7%	43.2%	**0.05*<**
NLR	0.72	0.06	0.59 to 0.82	**5.0**	92.8%	51.3%	**0.05*<**
NPR	0.66	0.06	0.53 to 0.77	**0.032**	42.8%	86.4%	**0.05*<**
PLR	0.6	0.07	0.74 to 0.72	**150.0**	21.4%	100%	NS
SII	0.7	0.06	0.85 to 0.81	**1000.0**	53.5%	81.1%	**0.05*<**
RDW	0.58	0.07	0.45 to 0.7	**14.0**	67.8%	51.3%	NS

* Statistically significant difference. AUC (area under the curve). SD (standard deviation). CI (confidence interval). Sens (sensitivity). Spec (specificity).

Table (6) Multiple regression analysis for pre-contrast hematological indices and laboratory investigations as predictors for CI-AKI.

Independent Variables (Pre-contrast)	Coefficient	SE	t	P
(Constant)	-1.86			
MLR	0.103	0.11	0.9	0.37
NLR	-0.028	0.015	-1.83	0.07
PLR	-0.0007	0.0005	-1.29	0.20
SII	0.002	0.0006	3.86	**<0.001****
NPR	9.95	3.6	2.75	**0.007***
ELR	-0.76	0.22	-3.33	**0.002***
RDW	0.03	0.018	1.7	0.09
S. Albumin (g/dl)	0.25	0.064	3.92	**<0.0001****
ALT (U/L)	0.002	0.0004	4.96	**<0.0001****
AST (U/L)	-0.001	0.0003	-5.44	**<0.0001****
S. Calcium (mg/dl)	0.0001	0.05	0.002	0.99
S. Phosphorous (mg/dl)	0.08	0.03	2.13	**0.03***
S. Cholesterol (mg/dl)	-0.002	0.0007	-2.67	**0.011***
S. LDL (mg/dl)	-0.0006	0.0009	-0.7	0.48
S. Triglycerides (mg/dl)	0.002	0.0003	5.97	**<0.0001****
Fasting blood sugar (mg/dl)	0.002	0.0008	2.46	**0.01***
S. Uric acid (mg/dl)	0.002	0.019	0.11	0.91
S. Potassium (mmol/l)	-0.13	0.05	-2.34	**0.02***
S. Sodium (mmol/l)	0.001	0.002	0.65	0.51
S. Ferritin (ug/l)	0.001	0.0005	2.46	**0.018***
CRP (mg/dl)	0.002	0.0004	4.52	**0.0001****
Troponin T (ng/l)	0.00001	0.00001	0.81	0.41
CK-MB (IU/l)	-0.008	0.0006	-2.32	**0.048***
BUN (mg/dl)	0.001	0.002	0.63	0.53
S. creatinine (mg/dl)	-0.01	0.06	-0.21	0.82
eGFR (ml/min)	0.009	0.003	3.03	**0.004***

**High statistically significant difference. *Statistically significant difference. SE (standard error).

Table (7): Odds ratios for pre-contrast hemogram-derived indices as predictors for CI-AKI.

Pre-contrast Variables	(OR)	Upper 95% (CI)	lower 95% (CI)
NLR	2.125	6.31533565	0.715025337
PLR	2.115702	6.265471528	0.714423003
SII	2.96875	9.115967606	0.966817451
MLR	2.89	8.722589472	0.957525288
ELR	3.4	10.40191261	1.111334082
NPR	2.5	7.532630692	0.829723407
RDW	2.125	6.31533565	0.715025337

OR (odds ratio). CI (confidence interval).

DISCUSSION

Contrast-induced acute kidney injury (CI-AKI) is a known complication after radiological and cardiological procedures. Preventive measures to avoid CI-AKI include intravenous fluids and the use of low/iso-osmolar contrast [(11).] The early prediction of CI-AKI is important, and hence the requirement for easy and low-cost inflammatory markers could be helpful. Hemogram-derived ratios and indices such as NLR, PLR, MLR, ELR, NPR, RDW, and SII could be useful inflammatory markers and predictors for mortality and severity [(12).]

This case-control study included 54 patients who had PCI, divided into; Group 1: (27 patients with no CI-AKI), and Group 2: (27 patients with CI-AKI). Patients with CI-AKI had higher pre-contrast triglycerides, phosphorous, FBS, and CRP and lower serum sodium than those with no CIA-AKI. **Lie *et al.* [(13)]** mentioned that risk factors for developing CI-AKI included heart failure, low blood pressure, low serum albumin, and high contrast dose.

We found that patients who developed CI-AKI had pre-contrast higher WBCs, neutrophils, NLR, SII, MLR, PLR, ELR, and NPR than those who did not develop CI-AKI. This agrees with **Cakir *et al.* [(14)]** who found that higher NLR, MLR, and PLR were predictors for ICU mortality of patients with sepsis, and **Abu Alfeilat *et al.* [(15)]** who stated that high NLR in Emergency Department early predicted AKI.

Inflammation is an important factor in the pathogenesis of AKI, and the pre-operative PLR was associated with a U-shaped survival pattern among patients with AKI, with increased mortality in AKI with both low and high PLR [(16).] Also, **Bu *et al.* [(17)]** found that high NLR was useful for risk stratification for septic AKI, while **Jiang *et al.* [(18)]** mentioned that MLR was a reliable marker for AKI in ICU patients.

Koo *et al.* [(19)] found that the neutrophil / lymphocyte x platelets (N / LP) ratio was associated with the development of post-operative AKI after cardiovascular surgery. In addition, SII was found to be an independent predictor for CI-AKI after coronary artery angiography [(10).] Contrast media causes vasoconstriction and hypoxia to the renal medulla by increasing the endothelin and decreasing nitric oxide and prostaglandins, in addition to its toxicity on renal tubular cells [(20).]

Using the ROC analysis, hematological indices that showed a higher sensitivity for predicting CI-AKI were NLR (92.8%), and MLR (85.7%), while those which had higher specificity were NPR (86.4%), and SII (81.1%). Leucocytes, including the neutrophils, lymphocytes, and monocytes have a role in the systemic inflammation, while platelets have a role in the hemostasis [(7).]

Using the multiple regression analysis, independent predictors for CI-AKI included pre-contrast SII, NPR, ELR, serum albumin, ALT, AST, triglycerides, CRP, serum phosphorous, cholesterol, FBS, potassium, ferritin, CK-MB, creatinine, and eGFR. **Mo *et al.* [(21)]** found that baseline CK-MB, serum uric acid, and NT-pro-BNP were significant predictors for CI-AKI, and in 2020, the KDIGO recommendations stated that CI-AKI is defined if within 48 hours of contrast-media administration, serum Cr rise by 0.3 mg/dL or more or 1.5 times or more the baseline level.

Factors contributing to CI-AKI after PCI in patients with acute myocardial infarction (MI) include the volume of contrast media, increased sympathetic activity, inflammatory response, hypovolemia, hyperglycemia, older age, and anemia [(22).] **Shen *et al.* [(23)]** found that SII and small density-LDL can predict CI-AKI in patients with acute MI undergoing emergency PCI, and their combination is useful for early assessment of CI-AKI.

Butt *et al.* [(24)] found that NLR, but not PLR was a predictor for CI-AKI after PCI, while **Kurtul *et al.* [(25)]** found that using a multivariate analysis, NLR, eGFR, and CRP were independent predictors for CI-AKI.

The cutoff values for NLR, SII, NPR, MLR & ELR were (5.0, 1000.0, 0.032, 0.5 & 0.08), and AUC (0.72, 0.7, 0.66, 0.64 & 0.64). **Zheng *et al.* [(16)]** found that the cut-off values for NLR, MLR, and PLR were 9.2, 0.8, and 187.3, and AUC values were 0.825, 0.835, and 0.720 for predicting mortality in ICU patients with sepsis.

A few hours after myocardial ischemia, neutrophils infiltrate the infarct area, exacerbating tissue injury, then monocyte-derived macrophages remove tissue debris. Although interventional reperfusion after ischemia

improves blood and oxygen supply, it triggers a pro-inflammatory response and further damage to the myocardium mediated by neutrophils [26].

The odds ratios (OR) for pre-contrast hemogram-induced indices as predictors for CI-AKI, with confidence interval (CI) 95% were as follows; NLR OR (2.12), CI (0.71-6.31); PLR OR (2.11), CI (0.71-6.26); SII OR (2.96), CI (0.96-9.11); MLR OR (2.89), CI (0.95-8.72); ELR OR (3.4), CI (1.11-10.4); NPR OR (2.5), CI (0.82-7.53); RDW OR (2.12), CI (0.71-6.31). This agrees with **Chen *et al.* [27]** who found that NLR had an OR 1.38, CI 1.03-1.84) for AKI progression in critically ill patients.

Hemogram-derived indices especially pre-contrast NLR, SII, NPR, and MLR are independent early predictors for CI-AKI after PCI. These are easy, widely available, and low-cost useful markers that can be calculated from the CBC and can help predict patients at risk for developing CI-AKI after PCI.

This agrees with **Tanik *et al.* [28]** who found that higher NLR is a significant, and independent predictor for post-PCI-AKI in patients with ST-elevation MI, and **Jiang *et al.* [10]** found that SII had the strongest association with CI-AKI after coronary angiography or PCI.

Points of strength: This study has investigated detailed hemogram-derived indices including SII, NLR, NPR, MLR, PLR, ELR, and RDW in patients with PCI, with CI-AKI. It compared patients with and without CI-AKI and showed detailed findings as regards the validity of each of the hemogram-derived indices in the early prediction of CI-AKI.

Limitations of the study: The small sample size may be considered a point of limitation in this study.

CONCLUSIONS

Hemogram-derived indices including pre-contrast SII, NLR, NPR, and MLR may be useful early predictors for CI-AKI after PCI.

Conflicts of interest: None.
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