Original Research Article

Neutrophil-to-lymphocyte Ratio and Its Relation with Markers of Inflammation and Myocardial Necrosis in Patients with Acute Myocardial Infarction and Chronic coronary syndrome

Abstract

Background: Inflammation has an essential role in atherosclerosis that is the leadingreason for acute coronary syndrome (ACS) whichincludes acute myocardial infarction (AMI) and unstable angina (UA).The objective of this work was to study the existence of difference in Neutrophil to Lymphocyte ratio and its relation to inflammatory markers between cases with AMI and cases with chronic coronary syndrome.

Methods: This workincluded sixty consecutive cases with AMI and ACS who presented to cardiovascular medicine department at Tanta university hospital. The cases were classified into two equal groups; group I**:** cases with AMI and group II:cases with chronic coronary syndrome.All participants were subjected toECG,Echocardiography, color Doppler, coronary angiography and laboratory investigations as differential CBC, cardiac enzymes, troponine, CK, CK-MB, CRP, urea, creatinine and random blood sugar.Patients who didn't meet the criteria for invasive treatment, continued on medication and further noninvasive investigations done, starting from stress ECG, stress echocardiography, or CCTA.

Results: There wereinsignificantly different between the two groups regarding age, gender and residency (p= 1.00).There was insignificant difference between the two groups regarding hypertension (p= 0.592), DM (p= 0.795) and dyslipidemia (p= 0.504). 7 (23.3%) cases were smokers in group I and 8(26.7%) patients were smokers in group II with insignificantly different between the two groups (p= 1.00).

Conclusions:NLR is a powerfulmarker of myocardial damage in acute myocardial cases.

Keywords: Neutrophil-to-lymphocyte Ratio, Myocardial Necrosis, AMI, Chronic Coronary Syndrome

Introduction:

Cardiovascular disorders are the leading reason for death in humans worldwide, and Coronary syndrome (myocardial infarction, MI) is from the common causes of deadly heart attack and heart failure. Based on the length of ischemia and metabolic requirement of the tissue, myocardial damage is caused by impaired vascular perfusion and reperfusion during MI.As a result, systemic and local inflammation might be generated, that is crucial for myocardial remodeling and scar formation [(1)].

There are two principal stages of inflammation in MI: stage of inflammation and stage of proliferation.Neutrophils are the initial leukocytes to populate an injured region. Their activation generates enormous quantities of inflammatory mediators that govern the reaction to tissue damage, manifesting as hypoxia, proteolytic enzymes, and other mediators [(2)]

At the location of an infarction, neutrophils emit free radicals that damage cardiomyocytes.The discharge of proteo-enzymes aids in the elimination of the infarct and boosts the recruitment of immune cells (in specific M1 macrophage)[(3)].

As a result, neutrophils not only recruit macrophages to the infarct place, however, facilitate the removal of waste. In contrary, lymphocytes have acrucial role in myocardial remodeling after inflammation.Such as, CD4+ T regulatory cells represent a specific anti-inflammatory immune regulatory lymphocyte subgroupthat is produced in the thymus and highly included for T cells with autoantigen specificity [(4)].

T cells are necessary for the enrollment of proangiogenic macrophages and the development of collateral arteries. B cells are found in monocyte enrollment[(5)].

The stimulation of fibroblasts and collagen deposition for scar creation and neovascularization (the proliferative stage) begin three to four days following MI. Inflammatory and anti-inflammatory mediator (IL-10, TGF-B and pro-resolving mediators)

releasefrom neutrophil or lymphocyte cells encourages neutrophil apoptosis and phagocytic eaten by macrophages [6].

Inflammation has a crucial role in the genesis and development of atherosclerotic plaque injuries. C-reactive protein (CRP), that is directly involved in plaque inflammation, stimulates the production of vascular cell adhesion molecule-1 (VCAM-1) in endothelial cells.But, the concentrations and values of high-sensitivity (hs) Troponin-CRP, white blood cell (WBC) count, and VCAM-1 in chronic coronary syndrome [7, 8].

Electrocardiogram (ECG) data are crucial for diagnosing myocardial ischemia. However, it is believed that just one-third of individuals with acute MI had ECG typical ST elevation, suggesting myocardial ischemia [9].

Due to the fact that a considerable proportion of patients with chest pain lack identifiable ischemia changes on the ECG, the diagnosis in these situations relies heavily on the increase in serum indicators as laboratory alterations in myocardial damage [10].

Patients and Methods:

This workincludedsixty consecutive cases who entered to Tanta university hospital and cardiac Islamic center, Al Azhar University, from the ER department collecting of STEMI and NSTEMI patients diagnosed by ischemic type of chest pain with and without ST-segment elevation or Q-waves together with elevated cardiac enzymes and positive troponinein the period from September 2021 to May 2022.

STEMI and NTEMI have extremely distinct biochemical markers.Biomarkers are prognostic and provide helpful diagnostic confirmation.On the contrary, in NSTEMI biochemical indicators have anessential role in diagnosis, differentiation from unstable angina (UA) and guiding management, in the outpatient clinic collecting of CCS (chronic coronary syndrome) complaining of typical chest pain during effort or stress or whomalready on anti-ischemic medications but still complaint of similar pain, patient post PCI procedure at least 6months.

Exclusion criteria arethe conditions affect the basal neutrophil to lymphocyte ratio (NLR); inflammatory diseases (gastritis, nephritis), any cause of ST elevation than MI, vviral infection, some forms of malignancies as lymphocytic leukemia and cases with acute cardiac conditions as dysrhythmia and HF.

Methods:

All cases were subjected to the following: Full history including name, age, history of medications and risk factors. NYHA class.

Full clinical examination: Both general and local examination including weight, height, body mass index (BMI), heart rate and both systolic and diastolic blood pressure.

ECG:

- Standard 12-lead resting ECGs were acquired by a typical ECG instrument (HP, Page-writer, USA) at a paper speed of 25 mm/s.
- During the process, participants were instructed to hold their breath for brief times to prevent chest wall movement from interfering with the signal.
- Two cardiologists performed ECG and heart rate (HR) measurements using a magnifying Glass (T or Q 150 mm Digital Caliper LCD) and were blinded to the clinical data.
- T-wave amplitude in lead III was determined as the magnitude of the biggest deflection above and below the baseline during a window spanning 80 milliseconds following the end of the QRS to the ending of the T wave.
- T-waves with an amplitude greater than 0.1 mV are considered inverted.

Any negative deflection at the start of the QRS wave was seen as the Q wave in lead III, whereas any negative deflection after the R wave was interpreted as the S wave in lead I.

Labs:

On admission, a sample from the antecubital vein was obtained from each caseprior to the administration of any medicine.

- Differential CBC using (Sysmex xs-800i) device: hemoglobin concentration (Hb %), RBCs, WBCs specially Neutrophils to lymphocytes ratio, platelet count.
- Cardiac enzymes using (Beckman Coulter AU 480).
- Troponine was done by (ABON cTnI) a one-step Troponine I test device, qualitative immunoassay based on a membrane for the detection of cTnI in whole blood, serum and plasma. This protein is the most commonly used biomarker. Troponin I is extremely heart-specific and remains up longer than creatine kinase-MB.
- CK, CK-MB, and CRP.
- Urea, creatinine and random blood sugar were done according to the standard lab methods.
- Echocardiography: Gain settings, sector width, and frame rate 24 were modified to maximize endocardial definitions in regular grayscale 2D imaging. During end-expiratory apnea, typical apical and parasternal images at a depth of 12-20 cm were taken. LV end diastolic volume, end systolic volume, and ejection percent were derived from apical 2- and 4-chamber images using a changed bi-plane Simpson's approach.All measures were performed in >3 consecutive cardiac cycles and >5 cycles if the case's rhythm wasAF, and the final analysis utilized the average results.
- Color Doppler was used to estimate the degree of mitral reguirge (MR) using vena contracta width measurement. MR was classified according to the following criteria
    - Mild :< 0.3cm
    - Moderate: .3 - .69cm
    - Severe: >.7 cm

Coronary angiography

Coronary angiography was done within 48 hours of admission through femoral arterial methodutilizing 6F catheterwhich had non-ionic, low-osmolar, iodinated contrast agent. The number of diseased arteries and places of lesion were determined. A luminal diameter narrowing >70% regarded as a major lesion.

Further investigations & conservative treatment

Patients who are not meeting the criteria for invasive treatment, continued on medication and further noninvasive investigations done, starting from stress ECG, stress echocardiography, or CCTA.

Statistical analysis

Windows® version 22 performed the statistical analysis (SPSS Inc, Chicago, IL, USA). Using the Shapiro-Wilks normality test and histograms, the distribution of numerical data was examined in order to identify the appropriate kind of statistical testing: parametric or nonparametric. The mean and standard deviation (SD) of parametric datawere used to compare the three groups using an ANOVA test, resulted by a post hoc (Tukey) test to compare each pair of groups. Categorical variables were reported in terms of frequency and percentage, and the Chi-square test was utilized to evaluate their statistical significance. At the appropriate cut off, estimations of the test's sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy were obtained using receiver operating characteristic (ROC) analysis. The Linear Correlation Coefficient was utilised to discover the correlation between two quantitative data in one group. A two-tailed P value of less than or equal to 0.05 was considered statistically significant.

Results:

There was insignificantly differentamong studied groups as regard demographic data (age, gender, residence, previous medical history and smoking) (table 1).

Table 1: Demographic data among the studied groups

		Group I (n = 30)	Group II (n = 30)	Test statistic	P-value
Age (years)	**Mean ± SD**	68.40 ± 12.82	65.47 ± 13.97	0.847 [a]	0.400
	Min - Max	58 – 78	51– 79		
Gender	**Male**	12 (40%)	19 (63.33%)	3.270 [b]	0.071
	Female	18 (60%)	11 (36.67%)		
Residence	**Rural**	15 (50 %)	16 (53.33%)	0.067 [b]	0.796
	Urban	15 (50%)	14 (46.67%)		
Previous medical history	**DM**	13 (43.3%)	14 (46.7%)	0.067 [b]	0.795
	Hypertension	12 (40%)	10 (33.3%)	0.287 [b]	0.592
	Dyslipidemia	11 (36.7%)	8 (26.7%)	0.693 [b]	0.405
Smoking		7 (23.3%)	8 (26.7%)	0.089 [b]	0.766

a: Independent samples T-test test; b: Pearson's Chi-square test; Max: maximum; Min: minimum; SD: standard deviation.

By comparing between both groups, WBCs, neutrophil, NLR, monocytes, CRP, troponin I and CK-MB were significant increase in group I than group II (P<0.05) while eosinophil was significantlydecreased in group I than group II (P <0.05).Lymphocytes and basophils were insignificant difference between both groups (table 2).

Table 2:Laboratory investigations among the study groups

		Group I (n=30)	Group II (n=30)	Test	P-value
WBCs ($x10^9$/L)	**Mean ± SD**	10.38 ± 1.23	8.87 ± 1.35	4.513 [a]	**<0.001***
	Min - Max	8.48 – 12.58	6.51 – 10.74		
Neutrophil ($x10^9$/L)	**Mean ± SD**	7.32 ± 1.56	5.23 ± 0.73	6.645 [a]	**<0.001***
	Min - Max	5.03 – 9.77	3.93 – 6.47		
Lymphocyte ($x10^9$/L)	**Mean ± SD**	1.91 ± 0.28	2.01 ± 0.25	1.351 [a]	0.182
	Min - Max	1.41 – 2.34	1.48 – 2.40		
N/L ratio	**Mean ± SD**	3.93 ± 1.12	2.66 ± 0.54	5.615 [a]	**<0.001***

	Min - Max	2.23 – 5.90	1.67 – 3.75		
	Normal	2 (6.67%)	11 (36.67%)	7.954 [b]	**0.005***
	Abnormal	28 (93.33%)	19 (63.33%)		
Monocyte ($x10^9$/L)	**Mean ± SD**	0.72 ± 0.13	0.58 ± 0.11	4.230 [a]	**<0.001***
	Min - Max	0.51 – 0.94	0.40 – 0.76		
Eosinophil ($x10^9$/L)	**Mean ± SD**	0.13 ± 0.06	0.23 ± 0.07	5.802 [a]	**<0.001***
	Min - Max	0.03 – 0.21	0.12 – 0.31		
Basophil ($x10^9$/L)	**Mean ± SD**	0.05 ± 0.01	0.05 ± 0.02	0.633 [a]	0.529
	Min - Max	0.03 – 0.07	0.03 – 0.08		
CRP (mg/L)	**Mean ± SD**	32.2 ± 10.35	26.13 ± 11.66	2.096 [a]	**0.04***
	Min - Max	11.6 – 45.56	4.26 – 42.35		
Troponin I	**Mean ± SD**	10.57 ± 4.2	5.81 ± 1.95	5.533 [a]	**<0.001***
	Min - Max	5.21 – 18.3	2.43 – 9.54		
CK-MB (U/L)	**Mean ± SD**	92.18 ± 30.97	23.21 ± 4.88	11.852 [a]	**<0.001***
	Min - Max	40.28 – 139.39	15.05 – 30.06		

Max: maximum; Min: minimum; SD: standard deviation; a: Independent samples T-test test; b: Pearson's Chi-square test; * statistically significant ($p \leq 0.05$)

The mean EF was insignificantly changed between both groups. Number of cases with EF 45 - 50% was insignificantly different between both groups, Number of cases with EF 47 - 60% was significant increase in group I compared to group II while Number of cases with EF 55 - 63% was significantly decreased in group I than group II (P = 0.006, 0.010 respectively) (table 3)

Table 3: Comparison between the study groups regarding EF:

	RWMA	Group I	Group II	Test statistic	P value
EF (%)	**Mean ± SD**	53.43 ± 8.09	55.37 ± 7.57	0.076 [a]	0.343
	Min-Max	41 - 65	42 - 66		
	(55-63 %)	5 (16.7%)	15 (50.0%)	7.50 [b]	**0.006***
	(47-60 %)	20 (66.7%)	10 (33.3%)	6.667 [b]	**0.010***
	(45-50 %)	5 (16.7%)	5 (16.7%)	0.0 [b]	1.00

a: Independent samples T-test test; b: Pearson's Chi-square test, * statistically significant ($p \leq 0.05$)

Regarding group I, NLR had a significant positive association with neutrophil (r=0.856, P<0.001), CRP (r=0.382, P=0.037), Troponin I (r=0.417, P=0.022), CK-MB (r=0.378, P=0.039) and a significantly negative association with lymphocyte (r=-0.684, P<0.001) and EF (r=-0.401, P=0.028) while insignificant association was presented between NLR and (age, BP, WBCs, monocyte, eosinophil and basophil.Regarding group II, NLR had a significant positive association only with neutrophil (r=0.780, P<0.001), and had a significant negative association with lymphocyte (r=-0.745, P<0.001) and EF (r=-0.601, P<0.001) while insignificant correlation was presented between NLR and (age, BP, WBCs, monocyte, eosinophil, basophil, CRP, troponin I and CK-MB)(Table 4)

Table 4:Correlations between N/L ratio and other markers in the study groups

	N/L ratio			
	Group I (n=30)		Group II (n=30)	
	r	p- value	r	p- value
Age	0.078	0.680	-0.048	0.800
SBP	-0.217	0.249	-0.339	0.067
DBP	-0.251	0.180	0.296	0.112
WBCs ($x10^9$/L)	-0.109	0.565	-0.336	0.070
Neutrophil ($x10^9$/L)	0.856	**<0.001***	0.780	**<0.001***
Lymphocyte ($x10^9$/L)	-0.684	**<0.001***	-0.745	**<0.001***
Monocyte ($x10^9$/L)	-0.112	0.557	0.188	0.321
Eosinophil ($x10^9$/L)	0.097	0.609	-0.124	0.514
Basophil ($x10^9$/L)	0.287	0.124	-0.089	0.640
CRP (mg/L)	0.382	**0.037***	0.205	0.277

Troponin I	0.417	**0.022***	0.034	0.86
CK-MB (U/L)	0.378	**0.039***	0.221	0.241
EF%	-0.401	**0.028***	-0.601	**<0.001***

r: coefficient of Pearson's correlation (weak: r<0.3; moderate: r =0.3-0.7; strong: r>0.7, irrespective of the sign);
* statistically significant ($p \leq 0.05$)

Cases with abnormal NLR had significant increase in CRP, troponin I, CK-MB and more abnormal ECG findings compared to those with normal NLR (P <0.05). EF was significantly decreased in cases with abnormal NLR than those with normal NLR (P <0.001) (Table 5)

Table 5:Relationship between NLR and Cardiac evaluation

Cardiac evaluation		N/L ratio		Statistical tests	
		Normal (n = 13)	**Abnormal (n = 47)**	**Test statistic**	**P-value**
Groups	**I**	2 (6.67%)	28 (93.33%)	FE	**0.01***
	II	11 36.67%	19 63.33%		
CRP	**Mean ± SD**	22.91 ± 11.25	30.89 ± 10.87	2.287[b]	**0.026***
	Min – Max	4.26 – 41.69	4.5 – 45.56		
Troponin I	**Mean ± SD**	6.04 ± 2.45	8.78 ± 4.19	2.216[b]	**0.031***
	Min – Max	3.25 – 12.64	2.43 – 18.3		
CK-MB	**Median [IQR]**	26.53 [18.11 – 27.75]	58.09 [25.86 – 108.36]	133[c]	**0.002***
	Min – Max	15.55 – 55	15.05 – 139.39		
ECG Findings	**ST depression**	12 (92.3%)	25 (53.2%)	FE	**0.011***
	St elevation	1 (7.7%)	22 (46.8%)		
	Inverted T wave & pathological Q wave	8 (61.5%)	12 (55.3%)	FE	**0.028***
	Peaked T wave	2 (15.4%)	21 (44.7%)		
EF (%)	**Mean ± SD**	61.77 ± 4.17	52.36 ± 7.28	4.386[b]	**<0.001***
	Min – Max	51 - 66	41 – 65		

a: Pearson's Chi-square test; b: Independent samples T-test; c: Mann-Whitney test; d: Fisher-Freeman-Halton exact test; FE: Fisher's exact test; IQR: interquartile range; * statistically significant ($p \leq 0.05$).

NLR can significantly predict acute MI with AUC of 0.844 (P value<0.001), at cut off >2.69, it's a significant predictor for MI with 90% sensitivity, 63.33% specificity. Troponin I can significantly predict acute MI with AUC of 0.828 (P value<0.001), at cut off >6.64, it's a significant predictor for MI with 76.67% sensitivity, 63.33 specificity. CK-MB can significantly predict acute MI with AUC of 1.00 (P value<0.001), at cut off >30.06, it's a significant predictor for MI with 100% sensitivity, 100% specificity.

By comparing between NLR, troponin I and CK-MB, CK-MB was the best diagnostic of acute MI with AUC of 1.00, the diagnostic accuracy of NLR and troponin I for acute MI was comparable (Table 6)

Table 6:ROC curve analysis of NLR, troponin I and CK-MB for diagnosing acute MI

	N/L ratio	Troponin I	CK-MB
AUC	0.844	0.828	1.00
95% CI of AUC	0.727 to 0.925	0.709 to 0.913	0.940 to 1.00
p-value (Null hypothesis AUC=0.5)	**<0.001***	**<0.001***	**<0.001***
Cut-off value	>2.69	>6.64	>30.06
Sensitivity (%)	90	76.67	100
Specificity (%)	63.33	63.33	100
Pairwise comparisons of AUCs			
N/L ratio vs. Troponin I	0.813		
N/L ratio vs. CK-MB	**0.002***		
Troponin I vs. CK-MB	**<0.001***		

AUC: area under the curve, CI: confidence interval, *: significant as P value ≤ 0.05.

Discussion

In our work, there was insignificantly differentamong studied groups as regard demographic data. Our findings were supported by **Tahto et al.,**[11]as they described that cases in the AMI group were older than cases in the UA group, however without significant differences.

However, in the study of **Demir et al.,**[12]the CAD group had much older average ages. ($p = 0.01$). The ratio of male patients in the CTO and CAD groups was increased than in the controls. The CTO group had a considerably greater incidence of cardiovascular risk factors like diabetes mellitus, hyperlipidemia, and a family history of coronary artery disease (CAD) than other groups ($p < 0.05$), However, hypertension and smoking were comparable across all groups.

However, in the study of **Lin et al.,**[13] there were insignificantly different in heart rate, diastolic blood pressure, period of hospitalization, and the number of implanted stents at entrance.

Our findings were supported by **Sönmez et al.,**[14] as they described that cases with CAD had a significant increase value of NLR ($p<0.001$).

Also, **Mansiroglu et al.,**[15] revealed that Neutrophil count and NLR were significantly different ($p <0.001$). A total of 426 cases (102 unstable angina pectoris (USAP), 223 non-STEMI, 103 STEMI were assessed.

In the study of **Demir et al.,**[12], The groups had comparable Hb values and platelet counts. WBC and neutrophil count were significant increase in the CTO group; in contrary, the lymphocyte count was significant decrease in the CTO group ($p < 0.05$). NLR levels were significant increase in the CTO group ($p < 0.001$).

Furthermore, **Tahto et al.,**[11] demonstrated that the acquired outcomes demonstrated that the average peripheral blood NLR levels in the AMI group were considerably greater than those seen in the UA group ($p = 0.001$). Also, average leukocyte count ($p = 0.001$), neutrophils ($p < 0.0005$) and monocytes ($p = 0.03$) in the peripheral blood were significant increase in the AMI group than the values in the UA group.

This study's neutrophil-to-lymphocyte ratio is consistent with the findings of a previous investigation by**Zazula and associates**[16], who also observed a significantly different in the

NLR among the following four groups of cases: cases with chest pain that was not the outcome of a cardiac disease, in whom the average NLR was the lowest; cases with unstable angina pectoris; cases with NSTEMI; and cases with STEMI in whom the average NLR was the highest. Nevertheless, a research done by **Meissner et al**[17], the NLR was shown to be greater in cases with acute MI (STEMI, NSTEMI) and UAP than those diagnosed with nonspecific chest pain; however, insignificantly different in NLR levels between STEMI and NSTEMI cases was discovered.

In the study of **Lin et al.,**[13], NLR, WBC, RBCs, Hb RDW, blood glucose, creatinine, D-dimer, fibrin degradation products, CRP, and Nt-pro-BNP were significantly different between the two groups.NLR, white blood cell, erythrocyte, Hb, RDW, blood glucose, creatinine, D-dimer, fibrin degradation products, CRP, and Nt-pro-BNP were significantly different between the two groups. RBCs volume, platelet count, lymphocyte counts, total bilirubin, triglyceride, total cholesterol, HDL, LDL, uric acid, troponin, glycosylated hemoglobin, and thyroid stimulating hormone were insignificantly different between the two groups.

Acet 's, et al.,[18] study showed increased NLR levels were substantially associated with lower patency of infarct-related blood vessels prior to PCI, indicating that NLR might be a more accurate indication of infarct-related arterial blood flow in STEMI cases prior to PCI.

According to **Zuin et al.,**[19], in STEMI and NSTEMI groups, significantly increased neutrophils and SXs (SYNTAX score) were detected ($p < 0.001$) in upper vs. lower among NLR tertiles and a significantlyassociation was observedamong the NLR and SXs.

Our results were supported by study of **Tahto et al.,**[11] as by assessing the link between the examined parameters in patients with AMI and UA, they found substantial negative correlations between NLR and lymphocyte count, supporting the notion that lymphopenia is

prevalent in cases with ACS. Additionally, they detected substantial positive connections between NLR and CK-MB activity.

In accordance with the findings of our research done by **Altun et al.,**[20]. a significantly positive associationamong NLR and CRP was noted, consistent with the outcomes of the research undertaken by **Akpek et al.,**[21] which were in contrast with our research.

In the study of **Chen et al.,** [22], in every case group, there was a positive linear regression of NLR vs. CK-MB ($p < 0.001$), This outcome was consistent with ours, yet they found negative linear regression of NLR vs. EF ($p < 0.001$) which in contrast with ours. The findingsrecommend that the associations of NLR with these parameters were significant.

According to **Dur et al.,**[23], a positive association was observed between high NLR and SSS (syntax severity score) ($p=0.001$) and demonstrated. The associations between high NLR and CKMB ($p=0.009$) and troponin ($p=0.049$) were observed and demonstrated.

Also,**Gao et al.,**[24] explored the clinical significance and association of NLR in cases with AMI. The study approved that the AUC of NLR was 0.868 (95% CI: 0.830~0.906). At cut off <3.04, NLR was a significant predictor for AMI with 84.66% sensitivity, 77.17% specificity.

Furthermore, **Nalbant et al.,**[25]evaluatedperforming of NLR in diagnosing AMI among caseswith higher serum creatinine. They concluded that troponin, and NLR were noticed to be higher in cases with AMI, than cases without AMI.

However, **Pyati et al.,**[26] showed that at cut off >6.32 ng/mL, troponin I have highest diagnostic accuracy (AUC = 0.982), with 96.2 % sensitivity100% specificity and CK-MB at cut off >24 IU/L (AUC = 0.64) is a significant predictor for MI with 23.1 % sensitivity 61.5 % specificity.

Conclusions:

NLR is a powerful marker of myocardial injury in cases with AMI. HIn all cases, high NLR is related with myocardial dysfunction. Severe inflammation (NLR) couldexpect the result of the heart in cases with coronary syndrome.

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