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Research Article

Neutrophil to Lymphocyte Ratio as a predictor of myocardial contractility post-ST Segment Elevation Myocardial Infarction in young and middle-aged patients

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Abstract

Background: NLR is known to be more elevated in patient with worse LV systolic function**.** **Aim:** to detect relation between NLR and GLS in young and middle-aged STEMI patients. **Methods:** We investigated NLR & GLS in 56 patients during CCU STEMI admission and follow up NLR & GLS were done month later. Percentage of change of follow-up NLR in relation to the baseline admission NLR is calculated in each patient. **Results**: Baseline NLR shows moderate positive correlation with admission GLS ($p<0.001$ & $r= 0.593$) and fair positive correlation with follow-up GLS ($p<0.001$ & $r= 0.481$). Follow-up NLR shows moderate positive correlation with follow-up GLS ($p<0.001$ & $r= 0.613$). Percentage of NLR decline was related to GLS improvement state with means being about 56.52% in the group whose GLS improved after 1 month and only about 10.16% in the group whose GLS not improved. From. NLR was significantly higher in GLS>-13.8% group 6.06 (5.43±3.45) compared to (2.39±1.05) in GLS≤-13.8% group, $p<0.001$. By ROC curve, the cut-off value of NLR was 2.547. Multivariate analysis showed patients with higher NLR >2.547 are more probable to develop GLS>-13.8%. **Conclusion:** STEMI patients with high NLR have a greater chance to have worse LV systolic function at MI presentation and after one month

Keywords: LV GLS, NLR & ST elevation myocardial infarction

Introduction

The most severe form of coronary heart disease, which is the primary cause of death in Egypt and around the world, is myocardial infarction (MI). Myocardial necrosis brought on by unstable ischemia syndrome is known as MI. MI is a cardiac emergency with a high risk of morbidity and death, whether it is accompanied with ST segment elevation (STEMI) or not (NSTEMI)[1]. Necrotic cells cause a powerful inflammatory response by activating non-specific immunological pathways, and producing danger signals. which encourage leukocytes and endothelial cells to interact with each other, which results in extravasation of neutronphils and monocytes[2]. Neutrophils infiltrate the infarcted myocardium and coronary plaque, and by releasing reactive oxygen species and matrix-degrading enzymes, they cause tissue injury [3]. In patients with coronary heart disease (CHD), the ratio of neutrophils to lymphocytes (NLR) has recently been found to be a more reliable indicator of death and myocardial infarction than the total leukocytic count or neutrophil count alone[4]. High NLR is linked to a low left ventricular ejection fraction (LVEF%) in MI patients [5].

Due to its accessibility and ability to be performed in emergency situations, echo-cardiography is the modality of choice for determining risk stratification following MI [6]. When assessing the extent of myocardial infarction, GLS evaluation following MI appear to be more useful than LVEF% [7]. In healthy individuals, NLR increases with age [8] and previous studies testing the relation between GLS and NLR does not make the age-NLR relationship into account, so relation between NLR and GLS exclusively in young and middle-aged patients with STEMI has not yet well-established and needed to be addressed clearly.

Our study aim is to detect whether there is a relation between the left ventricular global longitudinal strain (GLS) and neutrophil-to-lymphocyte ratio (NLR) in young and middle-aged patients admitted by ST segment elevation myocar-dial infarction (STEMI).

Methods

This Analytic Prospective study was conducted on 56 patients with STEMI admitted to CCU of Cardio-thoracic hospital of Minia University during the period from October 2022 to May 2023**.** The study was explained to all patients, written consent was obtained from all participants. We excluded male patients more than 45 years old, post-menopausal females, patients with atrial fibrillation, severe valvular heart disease, chronic heart failure, prior acute coronary syndrome, cerebrovascular stroke, cancer, impaired renal function, chronic inflammatory disease, patients with sepsis or acute infection.

On admission, every patient was submitted to full history taking, clinical examination and the following investigations: ECG to detect STEMI, CBC, NLR, Serum creatinine, other routine laboratory tests were done and echocardiography.

Strain analyses

Speckle tracking echocardiography analysis was performed from apical views (apical four chambers, apical two chambers and apical long axis). Standard grayscale 2D images were obtained at a frame rate of 50–90 frames/s with electrocardiogram gating during three consecutive cardiac cycles and software package (Siemens Healthineers, USA was used for offline analysis.

Imaging of the LV myocardium in apical four chambers, apical two chambers and apical three chambers' views to estimate the global longitudinal strain of the myocardium.

LV endocardium was traced at each apical view at the end-systolic frame. A region of interest was automatically defined between the endo and epicardial borders and manually adjusted to include the LV myocardium. Global longitudinal strain was then corrected for LV end-diastolic volume and end -systolic volume [9].

After one month of medical treatment patients attended Cardiovascular clinic for evaluation of their symptoms, adjustment of medical treatment, follow up level of NLR and follow up echocardiography to assess GLS. The samples were divided into two groups based on the GLS values: GLS≤-13.8% and GLS>-13.8% [10].

Results

Our study included 56 young and middle-aged patients presented to our hospital by STEMI. From 56 participants, 50 patients (89.3%) were males, 28 patients (50%) were smokers, 24 patients (42.9%) were hypertensive, 16 patients (28.6%) were diabetics. Mean±SD of their age was 39.86±5.94. At time of MI presentation at our hospital, Mean±SD of TLC was 11.6 ±3.99, Mean±SD of Neutrophil-to-lymphocyte ratio (NLR) was 4.07 ±3.05, Mean±SD of Global Longitudinal Strain (GLS) was -11.9 ±3.29, and Mean±SD of LV ejection fraction (LVEF%) was 41.79 ±12.94 **(Table 1)**.

We found that LVEF% measurements significantly increased at follow-up in relation to baseline while GLS measure-

ments & NLR values significantly decreased as shown in **(Table 2).**

Included patients were divided according to systolic function improvement (indicated by GLS) at follow-up in relation to baseline at presentation into two groups (improved and not improved). NLR values decrease significantly in both groups as shown in **(Table 3)**.

Percentage of NLR values decline at follow-up in relation to baseline was calculated in each patient, and we found that NLR values significantly decrease more in those with improving GLS as shown in **(Table 4)**.

When included patients were divided according to GLS at presentation -those with GLS ≤ -13.8 are of better systolic function and those with GLS> -13.8 are of worse systolic function-, there was a significant difference between the two groups as regard DM, MI site, TLC & NLR, but no significant difference is present as regard age, sex, HTN, and smoking status as shown in **(Table 5).**

There was a significant positive correlation between NLR at presentation and GLS at presentation (p= <0.001 and r = 0.593) & follow-up GLS (p= <0.001 and r = 0.481). Also, there was a significant negative correlation between NLR at presentation and LVEF% at presentation (p= <0.001 and r = -0.572) & LVEF% at follow-up (p= <0.001 and r = -0.456) **(Figure 1)**.

There was also a significant moderate positive correlation between NLR and GLS (p= <0.001 and r = 0.613) and there was a significant negative moderate correlation (p= <0.001 and r = -0.538) between NLR and LVEF% as shown in **(Table 6)**.

Stepwise logistic regression analysis was done for age, HTN, diabetes, smoking status, MI site, TLC, and NLR for prediction of worse LV systolic function (GLS > -13.8) during MI presentation, we found that NLR & TLC showed significant statistical results for prediction of worse LV systolic function, but NLR was the strongest predictor for worse LV systolic function with p= 0.001, AOR= 2.19 & 95% CI= 1.27 – 3.38 as in **(Table 7)**.

ROC curve analysis was done for NLR as the most significant variable that predicts worse LV systolic function **(Figure 2)**. As regard NLR, optimal cut off point > 2.547 could predict LV systolic function impairment with sensitivity of 90.3% and specificity of 68.3 % and accuracy of 80.49% (AUC= 0.835, PPV= 77.93, NPV= 85 as in **(Table 8)**.

Table 1: Demographic data, risk factors, laboratory & echocardiographic data of studied population

	Total (N=56)
Age	
Mean±SD	39.86±5.94
(Range)	(21-48)
Sex	
Male	50 (89.3%)
Female	6 (10.7%)
Smoking	
Smokers	28 (50%)
HTN	
Hypertensive	24 (42.9%)
Non-hypertensive	32 (57.1%)
DM	
Diabetic	16 (28.6%)
Non-diabetic	40 (71.4%)
MI site	
Anterior	39 (69.6%)
Inferior	10 (17.9%)
Lateral	7 (12.5%)
Serum creatinine(mg/dl)	
Mean±SD	1.02±0.12
(Range)	(0.8-1.4)
Serum urea (mg/dl)	
Mean±SD	32.75±4.75
(Range)	(20-45)
TLC	
Mean±SD	11.6 ±3.99
(Range)	(5-22)
NLR	
Mean±SD	4.07±3.05
(Range)	(0.94-17.6)
GLS	
Mean±SD	-11.9±3.29
(Range)	(-18 - -5)
LVEF%	
Mean±SD	41.79±12.94
(Range)	(18-70)

TLC: Total leucocytic count NLR: Neutrophil to lymphocyte ratio
GLS: Global longitudinal strain LVEF %: Left ventricular ejection fraction
MI: Myocardial Infarction DM: Diabetes Mellitus
HTN: Hypertension

Table 2: Comparison of GLS, LVEF%, & NLR values in each patient between values at presentation & those at follow-up

	At presentation (Mean±SD)	After follow-up (Mean±SD)	p value
GLS	-11.9±3.29	-13.16±3.38	**0.001**
LVEF%	41.79±12.94	50.25±12.38	**<0.001**
NLR	4.07±3.05	2.44±2.29	**<0.001**

GLS: Global Longitudinal Strain LVEF%: Left Ventricular Ejection Fraction
NLR: Neutrophil to Lymphocyte Ratio

Table 3: NLR change between admission and follow-up in both groups of GLS change (improved or not improved)

NLR	At presentation (Mean±SD)	After follow-up (Mean±SD)	p value
In patients whose GLS improved (N= 31)	4.56±2.58	1.91±0.75	**<0.001**
In patients whose GLS not improved (N=25)	3.47±3.5	3.09±3.24	**0.003**

NLR: Neutrophil to Lymphocyte Ratio GLS: Global Longitudinal Strain

Table 4: NLR decline % in both groups of GLS change (improved or not)

	In patients whose GLS improved Median & (Range)	In patients whose GLS not improved Median & (Range)	p value
Percentage of NLR decline	56.52% (14%-76%)	10.16% (-19%-32%)	**<0.001**

NLR: Neutrophil to Lymphocyte Ratio GLS.: Global Longitudinal Strain

Table 5: Comparison of different risk factors and laboratory markers among systolic function state at presentation

	LV GLS		p value
	GLS≤ -13.8 (N=25)	**GLS> -13.8 (N=31)**	
Age (Mean±SD)	40.16±5.1	39.61±6.62	0.921
Sex			
Male	23 (46%)	27 (54%)	0.682
Female	2 (33.3%)	4 (66.7%)	
DM			
Diabetic	3 (18.8%)	13 (81.2%)	**0.014**
Non-Diabetic	22 (55%)	18 (45%)	
HTN			
Hypertensive	9 (37.5%)	15 (62.5%)	0.352
Not-hypertensive	16 (50%)	16 (50%)	
Smoking			
Smoker	16 (57.1%)	12 (42.9%)	0.06
Non-smoker	9 (32.1%)	19 (67.9%)	
MI site			
Anterior	12 (30.8%)	27 (69.2%)	**0.003**
Inferior	9 (90%)	1 (10%)	
Lateral	4 (57.1%)	3 (42.9%)	
TLC (Mean±SD)	9.67±3.8	13.15±3.47	**<0.001**
NLR (Mean±SD)	2.39±1.05	5.43±3.45	**<0.001**

NLR: Neutrophil to Lymphocyte Ratio GLS: Global Longitudinal Strain
TLC: Total Leucocytic Count

Table 6: Correlation between NLR & (GLS and LVEF%) at follow-up

	Follow-up NLR	
	R	**P**
Follow-up GLS	0.613	**<0.001**
Follow-up LVEF%	-0.538	**<0.001**

NLR: Neutrophil to Lymphocyte Ratio GLS: Global longitudinal Strain
LVEF%: left Ventricular Ejection Fraction

Table 7: Stepwise logistic regression analysis showing that NLR & TLC can independently predict LV systolic function state

Model		AOR	95% CI	p value
1	NLR	2.39	1.4-4.09	0.001
2	TLC	1.22	1.01-1.47	0.04
	NLR	2.19	1.27-3.38	0.005

NLR: Neutrophil to Lymphocyte Ratio TLC: Total Leucocytic Count

Table 8: ROC curve analysis data for NLR as a predictor of GLS> -13.8

	AUC	Optimal cut off point	Sensitivity	Specificity	Accuracy	PPV	NPV
NLR	0.835	2.547	90.3	68.3	80.49	77.93	85

NLR: Neutrophil to Lymphocyte Ratio

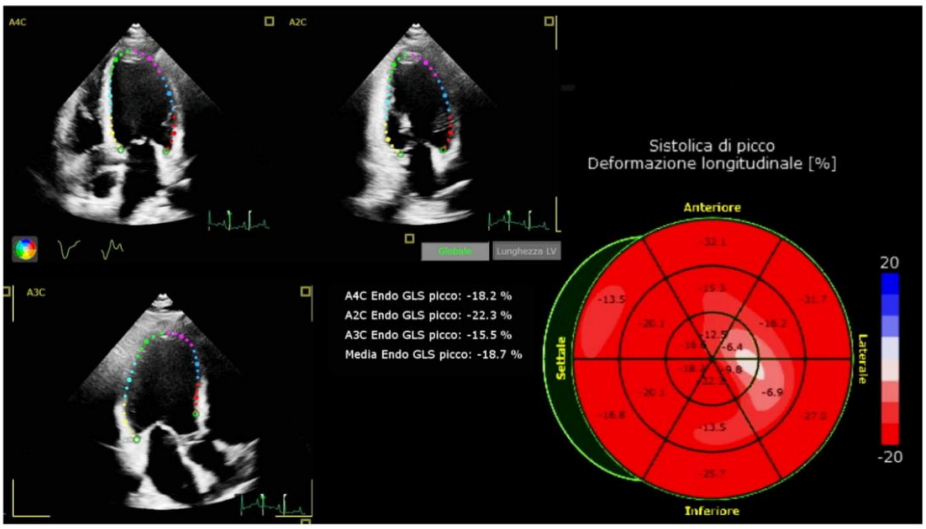

Figure 1: Longitudinal Strain analysis of different myocardial segments [27]

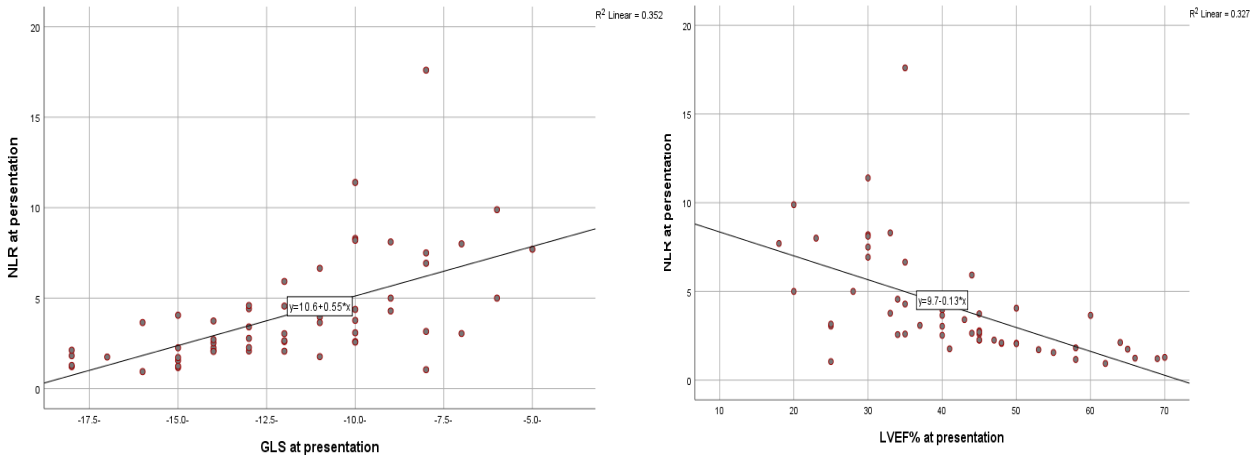

Figure 2: Correlation between NLR & (GLS and LVEF%) at presentation

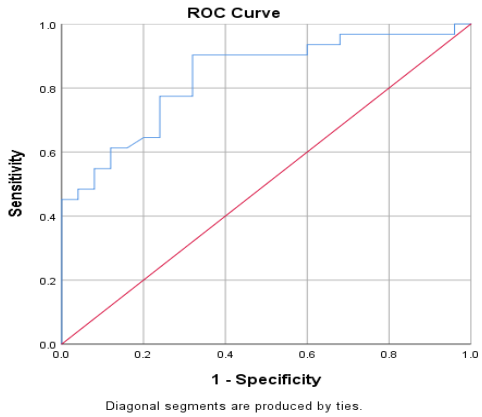

Figure 3: Roc curve of NLR as a predictor of GLS> -13.8

Discussion

Former studies have reported that neutrophil-to-lymphocyte ratio (NLR) could predict myocardial damage and clinical outcome after myocardial infarction (MI). Our results confirmed that NLR can predict LV systolic function in young and middle-aged patients with ST segment elevation myocardial infarction (STEMI).

In the current study, which was performed in a population of young and middle-aged STEMI patients, we found that NLR is correlated to left ventricle (LV) systolic function at MI presentation with a significant moderate negative correlation with LV ejection fraction (LVEF%) and LV global longitudinal strain (GLS). Also, we found that higher NLR (Above 2.55) can predict worse LV systolic function (GLS less than 13.8) independently of other factors like diabetes and site of MI (p=0.001). This cutoff point of GLS (-13.8) was stated by Eek at al study[10] which found that GLS > -13.8% is associated with an area of infarction > 12%. Such area of infarction is known to be associated with higher mortality rate in short-term observations[11].

Our current study has also found that admission NLR not only correlates with GLS and LVEF% during hospital admission but also correlates with GLS and LVEF% after 1 month. Also, follow-up NLR after 1 month can be significantly correlated with follow-up GLS and LVEF%. Furthermore, we found that the percentage of change of NLR from admission to follow-up is higher in patients whose follow-up GLS is better than basal one. Thus, we can say that NLR can be used as a useful biomarker for determining which patients may show poor prognosis even after successful revascularization.

Leukocytosis is a common finding in MI, and leucocyte differential analysis may provide more information for identifying high-risk patients, which is a crucial issue in day-to-day practice.

In an emergency room, the total leucocyte count can be quickly assessed within the first hour, making it a valuable diagnostic and prognostic indicator[12]. The predictive significance of the leucocyte count in patients with AMI has been shown in recent studies[13, 14]. Furthermore, recent study by Carbone et al., has demonstrated that some leucocyte subtypes are more predictively significant when assessing overall cardiovascular risk. In MI, neutrophilia follows myocardial injury. Additionally, neutrophils are known to release specific substances, including superoxide radicals and proteolytic enzymes, which may contribute to the rupture of atherosclerotic plaque and worsen the inflammatory process[15]. In patients with ACS, leucocyteplatelet aggregates may cause vascular occlusion which leads to an increase in size of infarcts[16]. Moreover, the most severe myocardial injury may result from vasoconstriction and micro-vascular blockage with neutrophil-platelet

plugs. Avanzas et al., showed that the size of the infarct area in MI is correlated with a high neutrophil count[17].

Regarding lymphocyte apoptosis, lymphopenia indicates the presence of a highly inflammatory process[18], and lymphopenia in acute conditions is caused by lymphocyte apoptosis, which causes the apoptotic cells to release pro-inflammatory cytokines. Thus, a high NLR is linked to the severity of CAD and is a sign of a high inflammatory process[19], and increased cardiovascular risk is linked to both relative neutrophilia and lymphopenia[20]Moreover, MI patients who show both neutrophilia and lymphopenia demonstrate a highly inflammatory process, and inflammation plays a significant role in myocardial injury in these patients.

In agreement with our results, Arbel et al., studied NLR, LVEF%, 30-day & 5-year all-cause mortality in 538 STEMI patients and found that higher NLR could be independently associated with lower LVEF% and higher 5-year mortality rates[5].

Similarly, Chen et al., who retrospectively analyzed NLR, CKMB, TGF-β, IL-10 & LVEF% in 715 patients with acute coronary syndrome undergoing percutaneous coronary intervention (PCI), have concluded that higher NLR can strongly predict LVEF% and other previously stated markers of myocardial damage[21].

Also, Azab et al., studied 619 patients with non–ST-segment elevation myocardial infarction (NSTEMI) to whom NLR was done inpatient. They found that those with average inpatient NLR above 4.7 had a higher 4-year mortality rate than those with average inpatient NLR below 3 [14].

Another Bekler et al., study included 405 NSTE-ACS patients who were analyzed for NLR and LVEF% and found that NLR could independently predict LV systolic dysfunction[22].
Kalkan et al., also studied 72 patients with anterior STEMI who performed echocardiography for LVEF% measurement, SPECT MPI with technetium Tc 99m sestamibi for assessment of myocardial perfusion defects & blood testing for NLR. They found that the higher is the NLR, the higher is the infarct size ratio, the lower is LVEF%, and the higher is the incidence of clinical heart failure in those patients[23].

Most of previously stated studies have used LVEF% as an indicative of LV systolic function. The assessment of LVEF% is known to be inherently variable and susceptible to measurement errors, off-axis imaging, and picture quality. Since speckle-tracking imaging software was developed, direct echocardiographic evaluation of cardiac fiber deformation has been accessible in standard clinical practice. GLS has recently been shown to have a good degree of consistency and agreement, and numerous studies have found that it can identify subclinical LV dysfunction[24]. Also, LVEF% barely detects earlier and minimal pathological changes in contrary to GLS[25]. GLS measurements after MI show specific benefits over LVEF% measurements of information on the infarction area[7].

Our study is not the first to relate GLS as a marker of LV systolic function to NLR. A study by Nursidiq et al.,[26] included 57 STEMI and NSTEMI patients who were divided according to GLS measurement results into GLS>-13.8% and GLS≤-13.8%. That study has found that NLR was significantly higher in those with GLS>-13.8% group compared to those with GLS≤-13.8%. However, mean age of patients participating in that study was 56.21±9.43 and it is known that NLR is positively associated with age with older people having a relatively higher NLR[8]. Hence, age may confound the relation between NLR & GLS and in our current study, we included only young and middle-aged STEMI patients. Also consequently, NLR cut-off value in our current study was 2.55 which is much lower than that of Nursidiq et al., study which was 4.69. Not only Nursidiq et al., disagreed with us on the matter of cut-off point value, but also

Azab et al., stated a different cut-off value of 4.7 for the same reason.

In addition to age issue, -unlike Nursidiq et al., study- in our study, follow-up NLR, GLS, and LVEF% measurements were done after 1 month, and we have found that admission NLR and follow-up NLR could be significantly associated with follow-up LVEF% and GLS. In addition to that, the site of MI was not observed by Nursidiq et al.

In brief, NLR is a cheap and widely available parameter in almost emergency care and our current study stated that higher NLR values can be used as an indicative of worse LV systolic function (GLS>-13.8%) -and subsequently larger infarct size- with NLR cut-off value of 2.55 (with a sensitivity of 90.3% and a specificity of 68.3%) in young and middle-aged patients with STEMI.

The present study has some limitations. First, our study was based on a relatively small group of patients. Second, failure to measure GLS of patients before onset of STEMI. Finally, short period of follow-up does not allow observation of major adverse cardiovascular events (MACE) and possible direct relation between NLR and MACE may need to be tested in future studies.

Conclusion

High NLR can independently predict worse LV systolic function and so can predict poor outcome in young and middle-aged patients with STEMI. NLR is correlated with LV GLS in those patients, where high NLR is associated with worse GLS.

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