Original Research Article

Hematological Parameters and Short Term Clinical Outcome After ST-Elevation Myocardial Infarction

Abstract

Background: ST-Elevation myocardial infarction is a major public health problem and a leading cause of death both in developed and developing countries. The values of hematological biomarkers were evaluated as predictors of in hospital mortality and complications, in patients with acute coronary syndromes (ACS). This study aimed to draw a relationship between different hematological parameters and short-term clinical result in STEMI cases treated by primary percutaneous coronary intervention.

Methods: This prospective research involved 100 participants that had a STEMI and were receiving primary PCI and they were subjected to detailed history, general and local examination, resting surface ECG, baseline laboratory tests, reperfusion, transthoracic echocardiogram and follow up of clinical outcome.

Results: After STEMI, there was not significantly different between cases with low and high NLR ($P < 0.4$). There was significantly different between cases with low and high NLR after STEMI, low and high MPV [in heart failure, death and rehospitalization] and after STEMI, in diabetic patients and in NLR between low and high MPV groups after STEMI ($P < 0.05$), there was no significant statistical difference between patients with low and high PDW and after STEMI. LVEF were significant less in participants had MACE ($P < 0.036$). In stepwise multivariate regression analysis of hematological parameters, NLR and MPV were significant predictive factors of MACE ($P < 0.05$).

Conclusions: Hematological and coagulation parameters may be utilized as diagnostic and prognostic indicators. Early risk classification enables doctors to closely monitor and treat high-risk patients, as well as schedule them for regular follow-ups, helping to the reduction of mortality.

Keywords: Hematological parameters, Short-term clinical outcome, ST-Elevation, Myocardial infarction.

Abbreviation List:

AHA: American Heart Association

CAD: Coronary artery disease

ECG: Electrocardiogram

FMC: First medical contact

IHD: Ischemic heart disease

IRA: Infarct related artery

LBBB: Left bundle branch block

LVEF: Left ventricular ejection fraction

MACE: Major adverse cardiovascular events

MLR: Monocyte-to-lymphocyte ratio

MPV: Mean platelet volume

NLR: Neutrophil-to-lymphocyte ratio

PDW: Platelet distribution width

PPCI: Primary percutaneous coronary intervention

Introduction:

Ischemic heart disease (IHD) is the main cause of death on a global scale, and its prevalence is rising. However, throughout Europe during the last three decades, there has been a general trend toward decreasing IHD mortality. IHD now accounts 20% of all deaths in Europe which correlates to 1.8 million annual deaths, with large variations between countries [1].

Numerous recent researches have shown a decrease in acute and long-term mortality after STEMI as a result of increased utilization of reperfusion treatment as primary percutaneous coronary intervention (PPCI), current antithrombotic therapy, and secondary prophylaxis [1-3]. However, mortality remains high; in-hospital mortality for unselected STEMI patients in the ESC nations' national registries ranges between 4% and 12%, while reported 1-year mortality among STEMI cases is roughly 10% [4].

Inflammation has a significant role in the development and evolution of coronary atherosclerosis. Peripheral hematological markers, especially leukocytes subtypes, as the neutrophil-to-lymphocyte ratio (NLR) and the monocyte-to-lymphocyte ratio (MLR) can be utilized as a barometer of systemic inflammation. Clinical and epidemiological research shows a probable relationship between hematological indicators and coronary artery disease (CAD). Lymphocytes, neutrophils, and monocytes perform a critical function in the inflammatory response. The NLR is widely used as a prominent indicator for inflammation and coronary atherosclerosis**.** However, few studies have assessed the association [5, 6].

It is a well-known that larger platelets recognized to be metabolically and enzymatically more active and have been demonstrated to have a significant role in the development of atherosclerotic lesions and associated impacts and have a greater risk of thrombosis than smaller ones. Moreover, they serve as a marker of platelet activation, and so are associated with and also appear clinically with (CAD). Platelet indicators like as mean platelet volume

(MPV) and platelet distribution width (PDW) can be used to determine the degree of platelet activation [7].

According to the American Heart Association (AHA), The short-term mortality rate of individuals with STEMI is between 5% and 6% during the first hospitalization and between 7% and 18% after one year. The highest risk of ischemic complications following MI occurs within 180 days, after which the risk becomes fairly linear [8].

The study aimed to draw a relationship between different hematological parameters and short-term clinical result in STEMI patients treated by primary PCI.

Methods:

This prospective study included 100 participants who had STEMI and were having primary PCI. They had persistent chest pain or other symptoms of ischemia such as shortness of breath, nausea/vomiting, exhaustion, palpitation, or syncope, as well as ST-elevation in at least two infectious leads. After approval by the local ethics committee, Faculty of Medicine, Tanta University, the research was carried out at the cardiovascular department, Tanta University Hospital and conducted over the period of 12 months in the period from September 2019 till August 2020.

Any cases in cardiogenic shock, cases with any oncotic disease, cases with end stage renal disease on hemodialysis, cases with chronic liver disease, cases with prior acute coronary syndrome within last 6 months, cases with prior PCI and cases with prior coronary artery bypass graft were excluded.

Prior to their involvement in the research, all individuals provided written informed consent, showing the study's value, plus procedures that have been performed.

According to ESC recommendations, The criteria for diagnosing myocardial infarction include the identification of an increase and/or decrease in cardiac parameters (ideally troponin) with at least one value greater than the 99th percentile of the upper reference limit. ,

together with Myocardial ischemia is manifested by at least one of the following: clinical manifestations of ischemia, electrocardiogram (ECG) changes indicating new ischemia (new ST-T changes or new left bundle branch block (LBBB), development of pathological Q-wave changes in the ECG, and imaging evidence of new loss of viable myocardium or new regional wall motion abnormality).

The following procedures were done for all participants:

History taking: were collected from all patients including: demographic data (age, sex, residence and educational level), medical history in general and related comorbidities, history of risk factors for (CAD) as Diabetes Mellitus, hypertension, smoking and past history and family history of CAD, renal impairment, recent surgery and trauma and rheumatoid arthritis and cerebrovascular events.

Clinical examination: Was done to all patients including vital heart rate, blood pressure, temperature and respiratory rate, general examination which was done with special attention to signs of heart failure and local cardiac examination as abnormal pulsation, Heart sounds & murmurs.

Resting 12 leads ECG: Standard 12-lead ECG was obtained during ten minutes of first medical contact (FMC) according to ESC guidelines including: (limb leads I, II, III, aVR, aVL, aVF, and Chest originates from V1to V6) for all patients on admission to the hospital. Right pericardial leads (V3R, V4R, V5R, V6R) and posterior chest leads (V7 to V9) were done for some patients to detect posterior wall and right ventricular infarction.

Baseline laboratory tests: including serum urea and creatinine, cardiac enzymes include serum troponin and CK-MB and complete blood count. CBC including: the NLR and the LMR were estimated as the ratio of neutrophils to lymphocytes and as the ratio of lymphocytes to monocytes, respectively. Platelet parameters, including count, MPV, PDW were determined using an automated cell counter device (Abbott Cell-Dyn4.000, Abbott

Park, IL, USA). Venous blood was directly drawn into a tube with a light blue top (contain anticoagulant -sodium citrate 3.2%). After then, the tube was twisted a few times gently and as soon as possible, for proper mixing with the anticoagulant. The entire period between collection of the sample and testing should not exceed 24 hours.

Reperfusion: was through Primary percutaneous intervention for infarct related artery (IRA).

Imaging: Left coronary imaging and right coronary imaging. Reperfusion success is measured by TIMI blood flow grade: Reperfusion was considered successful (TIMI 3) or abnormal (TIMI 0-1-2) according to the TIMI blood flow grade.

Echocardiography: All studies were carried out by using (a GE vivid seven cardiac ultrasound phased array system with tissue Doppler imaging using M4S transducer 4 MHz). 2D echocardiographic assessment was done during admission after successful PCI in left lateral decubitus position to: Assess LV systolic function using Biplane Simpson Method: Modified Simpson method (biplane method of disks) is a modality requiring area tracings of LV cavity.

Follow up of clinical outcome:

Follow up of clinical status of each patients'group for 1 month after discharge from hospital to evaluate complication related to MI as: Major adverse cardiovascular events (MACE) including (heart failure, cardiovascular death, hospitalization for acute coronary syndrome or heart failure, acute myocardial infarction and non-fatal stroke). Arrhythmias and conduction abnormalities are frequent in the early stages of STEMI and are also significant prognostic variables. Mechanical problems such as free wall, ventricular septal, and papillary muscle rupture are possible in the initial days after STEMI. Pericarditis, both early and late (Dressler syndrome), is often linked with late or unsuccessful coronary reperfusion, as well as with a higher infarct size.

Statistical analysis

The SPSS (Statistical Package for the Social Sciences) version 22 for Windows® was utilized to code and analyze the acquired data (IBM SPSS Inc, Chicago, IL, USA). The Shapiro Walk test was utilized to assess the data's normal distribution. Frequencies and percentages were used to characterize qualitative data. Fisher exact and Chi square tests ($\chi 2$) were employed to determine the difference between qualitative variables as specified. Quantitative data were described as mean ± SD (Standard deviation). A set of independent tests to compare two independent groups of regularly distributed variables (parametric data), the t-test was employed, whereas the Mann Whitney U test was used for non-normally distributed data (non-parametric data). Univariate and multivariate logistic regression analysis was used to estimate the dependent and independent risk predictor of categorical outcome.

Results

Characteristics of patients with acute myocardial infarction were discussed in the follwing table. **[Table 1]**

Regarding demographic data, hematological parameters and clinical outcomes in comparison between low and high NLR groups after STEMI. There was significantly different between two groups for MLR, MPV, troponin, heart failure, death and Rehospitalization. **[Table 2]**

Regarding hematological parameters in comparison between low and high MLR groups after STEMI, there was significantly different between two groups for diabetic patients. age, MPV, urea and creatinine. **[Table 3]**

Regarding hematological parameters in comparison between low and high MPV groups after STEMI, there was significant difference between two groups for diabetic patients, NLR, heart failure, death and Rehospitalization. **[Table 4]**

Regarding demographic data, hematological parameters and clinical outcomes in comparison between low and high PDW groups after STEMI, there was significantly different between two groups for age, MPV and urea. **[Table 5]**

Diabetes, NLR, MLR, MPV were significantly higher in patients had MACE. LVEF were significantly lower in patients had MACE. Age, male gender, hypertension, family History, active smoker, PDW, troponin, urea and creatinine were insignificantly different between patients had MACE and patients who hadn't MACE. **[Table 6]**

Discussion

As regarding Neutrophil to lymphocyte ratio, the results demonstrated that on comparison between low and high NLR groups after STEMI regarding clinical outcome, in heart failure, mortality, and rehospitalization, there was a significant variation between the groups. Our results supported by **Gul et al.** [9] study conducted on 145 patients and reported that high NLR group had higher rate of complications as heart failure and death. Similar to our study **Wang et al.** [10] study conducted on 840 patients with STEMI in whom PCI and reported that NLR was found to be a predictor of all-cause death and cardiovascular events in cases having angiography or cardiac revascularization.

As regarding Monocyte to Lymphocyte ratio, the results showed that on comparison between low and high MLR groups after STEMI regarding outcome and complications, there was not significantly different between the two groups. In disagreement with our results, **Kiris et al.** [11] assessed MLR 48h after admission in 318 STEMI participants who received primary PCI and discovered that it was independently associated with long-term mortality. **Wang et al.** [12] registered 306 STEMI participants and discovered that MLR was strongly associated with long-term major adverse cardiac and cerebrovascular events. This may be explained by short term follow up in our study. In **Cai et al.** [13] study conducted on 1369 STEMI patients and reported that MLR continued being significantly correlated with the endpoints of the study in patients with STEMI which suggests that MLR can be utilized to predict a types of adverse results after STEMI.

As regarding (MPV), on comparison between low and high MPV groups after STEMI regarding clinical outcomes there was significantly different between the two groups in heart

failure, death and Rehospitalization. Similar to our study **Monteiro Júnior et al.** [14] included 466 patients and reported that the mean MPV value was 10.9 ±0,9 (HR 2.97, 95% CI: 1.15–7.67, p = 0.024) and reported increased in (MPV) was associated with higher mortality. Furthermore, **Chu et al.** [15] study on 2809 patients and discovered that elevated MPV was independently related with an incidence of acute MI & mortality following MI which concordant to our study, but discordant to our study in restenosis following coronary intervention. Similar to our study **Adam et al.** [16] study conducted on 250 patients with ACS indicated that the increase in MPV may present an independent mortality risk factor. On the other hand, **Klovaite et al., 2011**[17] study conducted on 1300 patients developed MI indicated that there is no relationship between raised the rate of MPV and death in CAD's patients. **Lopez-Cuenca et al. [18]** study conducted on 329 patients and reported that MPV was of poor independent prognostic significance at 6 months' follow-up in the patients with non-STEMI. This may be attributed to different sample size as it was larger than our study sample size as mentioned before.

As regarding (PDW), on comparison between low and high PDW groups after STEMI regarding outcome and complications there was not significantly different between the two groups. In disagreement with our study **Bekler et al. [19]** study conducted on 502 patients and reported that an increased level of PDW (>17%) was related to the severity of CAD in patients with ACS. In disagreement with our study **Cetin et al. [20]** study conducted on 260 patients and reported that, PDW was greater in patients with STEMI than in those with stable CAD. This difference in results may be regarded to the difference in characteristic of the patients.

As regarding patients who had MACE, In the current study, diabetes, NLR, MLR, MPV were significantly higher in patients had MACE (P <0.05). LVEF were significantly lower in patients had MACE (P <0.05). Age, male gender, hypertension, family History, active

smoker, PDW, troponin, urea and creatinine were insignificantly different between patients had MACE and patients who hadn't MACE. In disagreement with our study **Adam et al.** [16] reported there was significantly different between the two groups regarding MPV and LVEF. The percentages of the left ventricular ejection fraction were lower in death group than in survival group**.** This may be attributed to different sample size as it was larger than our study sample size.

Conclusions:

Hematological and coagulation parameters may be employed as diagnostic and prognostic indicators. Early classification of high-risk patients enables doctors to closely monitor and treat them, as well as schedule them for regular follow-ups, so contributing to mortality reduction.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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Tables

Table legends

Table 1: Comparison between low NLR (Group A) and high NLR (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Table 2: Comparison between low MLR (Group A) and high MLR (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Table 3: Comparison between low MPV (Group A) and high MPV (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Table 4: Comparison between low PDW (Group A) and high PDW (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Table 5: Relationship between demographic data and hematological parameters with major adverse cardiovascular events (MACE)

Table 1: Comparison between low NLR (Group A) and high NLR (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Variables		NLR (%)		p-value
		Group A Low < 3.7 n= 51	**Group B High ≥ 3.7 n= 49**	
Age (years)	Mean ± SD	59.4 ± 10.1	61.12 ± 8.9	0.3
Male Gender	n (%)	43 (84.3%)	41 (83.7%)	0.9
Hypertension	n (%)	30 (58.8%)	19 (38.8%)	0.8
Diabetes	n (%)	29 (56.9%)	29 (59.2%)	0.8
Family History	n (%)	14 (27.5%)	13 (26.5%)	0.9
Active smoker	n (%)	31 (60.8%)	32 (65.3%)	0.6
MLR (%)	Mean ± SD	0.33 ± 0.1	0.39 ± 0.17	**0.02***
MPV (fl)	Mean ± SD	7.7 ± 0.8	9.4 ± 1.6	**0.001***
PDW (%)	Mean ± SD	13.02 ± 3.9	12.8 ± 1.7	0.7
Troponin (ng/ml)	Mean ± SD	1.04 ± 1.2	1.8 ± 1.3	**0.03***
Urea (mg/dl)	Mean ± SD	34.08 ± 10.3	37.14 ± 13.5	0.2
Creatinine (mg/dl)	Mean ± SD	1.1 ± 0.2	1.15 ± 0.4	0.6

Heart Failure	n (%)	1 (7.7%)	27 (31%)	**0.001***
Death	n (%)	0 (0%)	6 (12.2%)	**0.01**
Stroke	n (%)	0 (0%)	2 (4.1%)	0.14
Reinfarction	n (%)	0 (0%)	1 (2%)	0.3
Rehospitalization	n (%)	4 (7.8%)	24 (49%)	**0.001***
LVEF (%)	Mean ± SD	48.3 ± 22.1	46.6 ± 15.1	0.6

*: significant as P value < 0.05; NLR, neutrophil to lymphocyte ratio; MLR, monocyte to lymphocyte ratio; LVEF, left ventricular ejection fraction; PDW, platelet distribution width; MPV, mean platelet volume

Table 2: Comparison between low MLR (Group A) and high MLR (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Variables		**MLR (%)**		**p-value**
		Group A Low < 0.32 n= 40	**Group B High ≥ 0.32 n= 60**	
Age (years)	Mean ± SD	56.6 ± 9.5	62.6 ± 8.8	**0.002***
Male Gender	n (%)	33 (82.5%)	51 (85%)	0.7
Hypertension	n (%)	17 (42.5%)	32 (53.3%)	0.2
Diabetes, n (%)	n (%)	15 (37.5%)	43 (71.7%)	**0.001***
Family History	n (%)	10 (25%)	17 (28.3%)	0.7
Active smoker	n (%)	25 (62.5%)	38 (63.3%)	0.9
NLR (%)	Mean ± SD	5.6 ± 3.4	6.09 ± 2.6	0.4
MPV (fl)	Mean ± SD	8.8 ± 0.8	9.8 ± 1.3	**0.001***
PDW (%)	Mean ± SD	12.7 ± 4.4	13.02 ± 1.5	0.6
Troponin (ng/ml)	Mean ± SD	1.2 ± 1.3	1.5 ± 1.3	0.29
Urea (mg/dl)	Mean ± SD	32.6 ± 9.6	37.5 ± 13.1	**0.04***
Creatinine (mg/dl)	Mean ± SD	1.02 ± 0.18	1.1 ± 0.3	**0.01***
LVEF (%)	Mean ± SD	48.7 ± 17.9	46.7 ± 19.7	0.5
Heart Failure	n (%)	9 (22.5%)	19 (31.7%)	0.3
Death	n (%)	2 (2.5%)	4(8.3%)	0.2
Stroke	n (%)	0 (0%)	2 (3.4%)	0.2
Reinfarction	n (%)	0 (0%)	1 (1.7%)	0.4
Rehospitalization	n (%)	8 (20%)	20 (33.3%)	0.14

*: significant as P value < 0.05; NLR, neutrophil to lymphocyte ratio; MLR, monocyte to lymphocyte ratio: LVEF, left ventricular ejection fraction; PDW, platelet distribution width; MPV, mean platelet volume

Table 3: Comparison between low MPV (Group A) and high MPV (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Variables		MPV		p-value
		Group A Low < 8 n= 43	**Group B High ≥ 8 n = 57**	
Age (years) (Mean ± SD)	Mean ± SD	58.4 ± 11.3	61.6 ± 7.8	0.1
Male Gender	n (%)	36 (83.7%)	48 (84.2%)	0.9
Hypertension	n (%)	24 (55.8%)	25 (43.9%)	0.2
Diabetes, n (%)	n (%)	19 (44.2%)	39 (68.4%)	**0.015***
Family History	n (%)	11 (25.6%)	16 (28.1%)	0.7
Active smoker	n (%)	27 (62.8%)	36 (63.2%)	0.9
NLR (%)	Mean ± SD	3.7 ± 1.7	5.3 ± 2.5	**0.001***
MLR (%)	Mean ± SD	0.34 ± 0.16	0.38 ± 0.12	0.18
PDW (%)	Mean ± SD	13.02 ± 425	12.8 ± 1.5	0.7
Troponin (ng/ml)	Mean ± SD	1.1 ± 1.3	1.6 ± 1.3	0.06
Urea (mg/dl)	Mean ± SD	34.19 ± 10.8	36.6 ± 12.8	0.3
Creatinine (mg/dl)	Mean ± SD	1.10 ± 0.2	1.14 ± 0.39	0.5
LVEF (%)	Mean ± SD	46.8 ± 22.7	48.06 ± 15.7	0.7
Heart Failure	n (%)	4 (9.3%)	24 (42.1%)	**0.0001***
Death	n (%)	0 (0%)	6 (10.5%)	**0.028***
Stroke	n (%)	1 (2.3%)	1 (1.8%)	0.8
Reinfarction	n (%)	0 (0%)	1 (1.8%)	0.3
Rehospitalization	n (%)	5 (11.6%)	23 (40.4%)	**0.002***

*: significant as P value < 0.05; NLR, neutrophil to lymphocyte ratio; MLR, monocyte to lymphocyte ratio; LVEF, left ventricular ejection fraction; PDW, platelet distribution width; MPV, mean platelet volume

Table 4: Comparison between low PDW (Group A) and high PDW (Group B) regarding demographic data, hematological parameters and clinical outcomes.

Variables		PDW		p-value
		Group A Low < 11.7 n= 32	**Group B High ≥ 11.7 n= 68**	
Age (years)	Mean ± SD	56.9 ± 8.2	61.8 ± 9.8	**0.015***
Male Gender	n (%)	28 (84.8%)	56 (83.6%)	0.8
Hypertension	n (%)	12 (36.4%)	37 (55.2%)	0.07
Diabetes)	n (%)	18 (54.5%)	40 (59.7%)	0.6

Family History	n (%)	10 (30.3%)	17 (25.4%)	0.6
Active smoker,	n (%)	20 (60.6%)	43 (64.2%)	0.7
LVEF (%)	Mean ± SD	50.1 ± 18.8	46.2 ± 19.02	0.3
NLR (%)	Mean ± SD	5.2 ± 1.6	6.2 ± 3.4	0.1
MLR (%)	Mean ± SD	0.37 ± 0.19	0.35 ± 0.1	0.5
MPV (fl)	Mean ± SD	8.6 ± 1.19	9.8 ± 1.12	**0.001***
Troponin (ng/ml)	Mean ± SD	1.1 ± 1	1.6 ± 1.4	0.06
Urea (mg/dl)	Mean ± SD	28.8 ± 7.5	38.9 ± 12.4	**0.001**
Creatinine (mg/dl)	Mean ± SD	1.05 ± 0.18	1.1 ± 0.38	0.1
Heart Failure				
Death	n (%)	2(6.3%)	4(5-9%)	0.9
Stroke	n (%)	0(0%)	2 (3%)	0.3
Reinfarction	n (%)	0 (0%)	1 (1.5%)	SS0.4
Rehospitalization	n (%)	6 (18.8%)	22 (32.4%)	0.15

*: significant as P value < 0.05; NLR, neutrophil to lymphocyte ratio; MLR, monocyte to lymphocyte ratio: LVEF, left ventricular ejection fraction; PDW, platelet distribution width; MPV, mean platelet volume

Table 5: Relationship between demographic data and hematological parameters with major adverse cardiovascular events (MACE)

Variables		**MACE (n = 38)**	**No MACE (n = 62)**	**p-value**
Age (years)	Mean ± SD	62.79 ± 7.219	58.69 ± 10.512	0.095
Male Gender	n (%	33 (86.8%)	51 (82.3%)	0.544
Hypertension	n (%	20 (52.6%)	29 (46.8%)	0.570
Diabetes	n (%	27 (71.1%)	31 (50.0%)	**0.038***
Family History	n (%	12 (31.6%)	15 (24.2%)	0.419
Active smoker	n (%	24 (63.2%)	39 (62.9%)	0.980
LVEF (%)	Mean ± SD	44.48 ± 13.11	49.40 ± 21.70	**0.036***
NLR (%)	Mean ± SD	6.53 ± 2.816	3.61 ± 0.817	**<0.001***
MLR (%)	Mean ± SD	0.13 ± 0.343	0.06 ± 0.248	**0.025***
MPV (fl)	Mean ± SD	9.89 ± 1.573	7.79 ± 0.727	**<0.001***
PDW (%)	Mean ± SD	13.21 ± 1.695	12.73 ± 3.604	0.797
Troponin (ng/ml)	Mean ± SD	1.97 ± 1.619	1.13 ± 1.208	0.120
Urea (mg/dl)	Mean ± SD	39.53 ± 13.928	33.16 ± 10.114	0.233
Creatinine (mg/dl)	Mean ± SD	1.16 ± 0.547	1.10 ± 0.298	0.131

***: significant as P value < 0.05**