Platelet/Lymphocyte Ratio and Risk of In-Hospital Mortality in Patients with ST-Elevated Myocardial Infarction - A Prospective Observational Study from KIMS, Hubli, Karnataka

Uday Subhash Bande[1], Kalinga Bommanakatte Eranaik[2], Basawantrao Kailash Patil[3], Manjunath Shivalingappa Hiremani[4], Sushma Shankaragouda Biradar[5]

[1, 2, 3, 4, 5]Department of General Medicine, Karnataka Institute of Medical Sciences, Hubli, Karnataka, India.

ABSTRACT

BACKGROUND

Cardiovascular disease is a significant health problem in India with an estimate 3.7 million deaths each year. Mechanisms of myocardial ischemia include inflammation, endothelial dysfunction, platelet aggregation and coagulation. Acute coronary syndrome occurs due to rupture of atherosclerotic plaque. Platelets play a role in both development and rupture of the atherosclerotic plaque. Lymphocytes play a role in chronic inflammation of atherosclerosis. Lower lymphocyte count has increased mortality after acute myocardial infarction.

METHODS

The study was conducted in Department of General Medicine, Karnataka Institute of Medical Sciences, Hubli from February 2019 to December 2020. It is a prospective observational study. Patients aged ≥ 18 years with ST-elevated myocardial infarction (STEMI) were included in the study. Total 156 cases were selected based on inclusion and exclusion criteria. Cardiovascular events during the in-hospital period were noted. The study population was divided into tertiles based on the platelet-lymphocyte ratio (PLR) values. The low PLR group (n = 104) was defined as having values in the lower 2 tertiles (PLR ≤ 148.4) and the high PLR group (n = 52) was defined as having values in the highest tertile (PLR > 148.4). A 'P' value < 0.05 was considered statistically significant.

RESULTS

Out of 156 patients, 103 (66 %) were males and 53 (34 %) cases were female. Mean age group was 59 ± 10 years. Percentage of patients who underwent thrombolysis was higher in high PLR group (65.38 % vs. 48.07 %, P = 0.041). Death rate was higher in high PLR group (28.84 % vs. 8.65 %, P = 0.001). PLR > 148.4 was found to be an independent predictor of in-hospital cardiovascular mortality in multivariate analyses (hazard ratio: 13.222 (2.113-21.749) P = 0.006 with 95 % confidence interval). Receiver operating curve (ROC) analyses, a PLR value of 148.4 for in-hospital mortality rate had sensitivity of 62.5 % and a specificity of 72 % (area under the curve = 0.627, 95% confidence interval 0.485 – 0.769).

CONCLUSIONS

In our study, higher PLR had significant association with in-hospital mortality in patients with STEMI.

KEYWORDS

ST Elevation Myocardial Infarction (STEMI), Platelet/Lymphocyte Ratio (PLR), Ischemic Heart Disease (IHD)

Corresponding Author:
Dr. Uday Subhash Bande,
Karthik, Vijayanagar,
Dharwad-580008,
Karnataka, India.
E-mail: udayphysici@yahoo.co.in

DOI: 10.18410/jebmh/2021/640

How to Cite This Article:
Bande US, Eranaik KB, Patil BK, et al. Platelet/lymphocyte ratio and risk of in-hospital mortality in patients with ST-elevated myocardial infarction - a prospective observational study from KIMS, Hubli, Karnataka. J Evid Based Med Healthc 2021;8(41):3528-3533. DOI: 10.18410/jebmh/2021/640

Submission 14-09-2021,
Peer Review 21-09-2021,
Acceptance 13-10-2021,
Published 30-10-2021.

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BACKGROUND

Cardiovascular disease is a significant health problem in India with an estimate 3.7 million deaths each year. Cardiovascular disease is a major cause of death occurring in developing country.[1] Mechanisms of myocardial ischemia include inflammation, endothelial dysfunction, platelet aggregation and coagulation.[1] About half of the death with STEMI occur before individual reaches to the hospital and most of the death due to myocardial infarction occur in first month.[2] STEMI occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery previously affected by atherosclerosis and there is insufficient collateral formation.[2] Most common cause of myocardial infarction is the atherosclerosis of epicardial coronaries.[3] Ischaemic heart disease (IHD) is a condition in which there is a decreased supply of blood and oxygen to a part of the myocardium. It usually occurs when there is an imbalance between myocardial oxygen supply and demand.[3]

Common risk factors for atherosclerosis include cigarette smoking, high level of plasma low-density lipoprotein (LDL), low level of plasma high-density lipoprotein (HDL), systemic hypertension and diabetes mellitus. Factors that prevent atherosclerosis include anti-thrombotic state, endothelial lining and suppression of inflammation. Diabetes mellitus leads to macro vascular complication and thus predispose the patient to development of coronary artery disease. Hypertension predispose to atherothrombosis by causing endothelial damage and exposing sub endothelial surface.

These risk factors can be modifiable or non-modifiable. Primary pathophysiology is endothelial damage that leads formation of atheromatous plaque.[3] When the stenosis is 50 %, there will be limitation to increase coronary blood flow during exercise. When the stenosis is 80 %, there will be limitation to coronary blood flow even with mild exertion or even at rest. Critical obstruction of left main coronary artery and left anterior descending artery are more dangerous than other arteries obstruction. Acute obstruction of coronary arteries is harmful than chronic obstruction. Patients with chronic coronary obstruction usually will have development of collaterals that will supply the myocardium. Common symptoms of myocardial infarction include chest pain, breathlessness, palpitation and excessive sweating.[3] Acute coronary syndrome occurs due to rupture of atherosclerotic plaque.[4] Acute coronary syndrome includes unstable angina, non STEMI and STEMI.[5]

In unstable angina, patient will have chest pain at rest without development of myocardial infarction and thus troponin I level is usually within normal range. In Non STEMI cases, there will be sub endocardial infarction with elevated troponin I level but without ST elevation in electrocardiogram (ECG). In patients with STEMI, there is complete obstruction of coronaries, transmural infarction with ST elevation in ECG and raised troponin I level.[5] STEMI is defined as > 30 minutes of continuous typical chest pain and ST-segment elevation ≥ 2 mm in 2 contiguous electrocardiography leads within 12 hours of symptom onset or within up to 18 hours if there was evidence of ongoing ischemia or hemodynamic instability and with raised troponin I levels.[6]

About 76 % of all fatal coronary thrombi are due to rupture of a plaque. Rupture of plaque is the most common cause of coronary thrombosis in males and in females. Ruptured plaques are characterized by a lipid-rich central core, a thin fibrous cap in the periphery which contains few smooth muscle cells and large number of macrophages, angiogenesis, adventitial inflammation, and outward remodeling. Commonest cause for coronary thrombosis is due to plaque rupture. Platelets play a major role in both development and rupture of the atherosclerotic plaque.[7] Lymphocytes also play a role in chronic inflammation of atherosclerosis and in contrast to platelet, lower lymphocyte count has increased mortality after acute myocardial infarction.[8,9] A study performed in Turkey by Ahmet Temiz et al. showed higher PLR was associated with increased in-hospital mortality in patients with STEMI.[10] Current study aims to assess reliability of platelet/lymphocyte ratio to predict in-hospital mortality in patients with ST-elevated myocardial infarction.

Aims and Objectives

To assess the relationship between platelet/lymphocyte ratio and risk of in-hospital mortality in patients with ST-elevated myocardial infarction.

METHODS

This study was conducted in the Department of General Medicine, Karnataka Institute of Medical Sciences, Hubli from February 2019 to December 2020. It is a prospective observational study. Total 156 cases were selected based on inclusion and exclusion criteria.

Inclusion Criteria

1. Patients aged ≥ 18 years with ST elevation myocardial infarction (STEMI).
2. A diagnosis of ST elevation myocardial infarction (STEMI) was defined as > 30 minutes of continuous typical chest pain and ST-segment elevation ≥ 2 mm in 2 contiguous electrocardiography leads within 12 hours of symptom onset or within up to 18 hours if there was evidence of ongoing ischemia or hemodynamic instability and with raised troponin I levels.

Exclusion Criteria

1. Patient aged < 18 years.
2. Unstable angina.
3. Stable angina.
4. Prinzmetal angina.
5. Non STEMI.
6. Established IHD.

Sample Size Estimation

It is calculated based on the below formula. Sample size is calculated as follows.

Sample size $=4pq/d^2$,

Where P = prevalence, q = (1-p) and d = 0.05. Prevalence (p) of STEMI patients admitted to KIMS, Hubli is 10.95 % (P = 0.1095), q = 1-p = 1-0.1095 = 0.8905, d = absolute precision, 'd' value is considered 0.05 to produce good precision and smaller error of estimate. By substituting p, q, and d value in above equation we get sample size of 156.

They were enrolled in the study only after obtaining written consent from the patient, in case where patient is not in state to give consent, consent was obtained from the patient's relatives. We collected demographic details like name, age and sex. We noted cardiovascular risk factors like diabetes mellitus, systemic hypertension and smoking history. Treatment received during the in-hospital stay was recorded. Patients whose blood pressure exceeded 140/90 mmHg at admission or those who had been treated with antihypertensive medication were considered to be hypertensive (HTN). Diabetes mellitus (DM) was defined as fasting blood sugar level above 126 mg/dl or post prandial blood sugar level above 200 or HbA1c ≥ 6.5 or the use of anti-diabetic drugs. Acute kidney injury (AKI) was defined as an absolute increase in serum creatinine by 0.3 mg/dL or more within 48 hours or a relative increase of atleast 1.5 times baseline that is known or presumed to have occurred in past 7 days.

Lab parameters like complete blood count, differential count, renal function test, troponin I level and random blood sugar (RBS) values were obtained. From complete blood count, platelet and lymphocyte count was obtained and PLR ratio was calculated. Blood sample was obtained by venipuncture and complete blood count and differential count was measured using Xp300 haematology analyser (Sysmex) and biochemical analysis was done using standard method.

Standard 12 lead electrocardiogram was done at admission and during hospital stay. 2-dimensional echocardiography was done for required patients. The patients with STEMI received thrombolysis if they were in window period and had no contraindication for thrombolysis. Those patients presenting late or those who had contraindication for thrombolysis were not thrombolysed. Cardiovascular events during the in-hospital period like ventricular tachycardia, complete heart block and death were recorded. Advanced heart failure was defined as Killip classification ≥ 3 and cardiovascular death was defined as death due to acute myocardial infarction, heart failure, or arrhythmia. The follow-up period was for 7 days. Killip classification was used to classify patients with heart failure.[11] Killip class 1 patients had no evidence of heart failure. Killip class 2 patients had mild heart failure with crepitation involving one third or less of the posterior lung fields and systolic blood pressure of 90 mm Hg or more. Killip class 3 patients had pulmonary edema with rales involving more than one third of the posterior lung fields and systolic blood pressure of 90 mm Hg or higher. Killip class 4 patients had cardiogenic shock with any rales and systolic blood pressure of less than 90 mm Hg.

Statistical Analysis

All the above data was filled in a proforma and master chart was prepared in an Excel sheet. The study population was divided into tertiles based on the PLR values. The low PLR group (n = 104) was defined as having values in the lower 2 tertiles (PLR ≤ 148.4) and the high PLR group (n = 52) was defined as having values in the highest tertile (PLR > 148.4). Quantitative variables are expressed as the mean value ± SD or median (interquartile range), and qualitative variables are expressed as percentages (%). A comparison of parametric values between high and low PLR groups was performed using the t test. Categorical variables were compared by the likelihood-ratio c^2 test. A P value < 0.05 was considered statistically significant. All statistical studies were carried out with the Statistical Package for Social Sciences (SPSS) program (version 21).

RESULTS

A total of 156 patients were studied, majority of the cases were of males 103 (66 %) and 53 (34 %) cases were of females. Mean age group was 59 ± 10 years. Among study population, 52 patients had history of smoking, 68 patients had diabetes mellitus, 55 patients were hypertensive. Out of 156 STEMI patients, 84 patients underwent thrombolysis. During follow up period of 7 days, 13 patients developed ventricular tachycardia, 31 patients developed heart failure Killip class 3 ≥, 21 patients developed acute kidney injury and 6 patients developed complete heart block. In the study population, 24 patients had died, 15 patients died in high PLR group and 9 patients died in low PLR group. Overall mortality rate was 15.38 %, mortality was higher in high PLR group when compared with low PLR group. Table 1 shows the patient's clinical characteristics, laboratory findings and in-hospital cardiovascular events of the groups. Figure 1 shows bar diagram representing the clinical characteristics, laboratory findings and in-hospital cardiovascular events of the groups.

Percentage of patients who underwent thrombolysis was higher in high PLR group (65.38 % vs. 48.07 %, P = 0.041). Death rate was higher in high PLR group (28.84 % vs. 8.65 %, P = 0.001). Sex, systemic hypertension, diabetes mellitus, smoking history and acute kidney injury were not significantly different between the groups. Heart failure Killip class ≥ 3, ventricular tachycardia and heart block were not significantly different between the groups. There was no significant difference between the groups who received aspirin, clopidogrel, statins and low molecular weight heparin (LMWH).

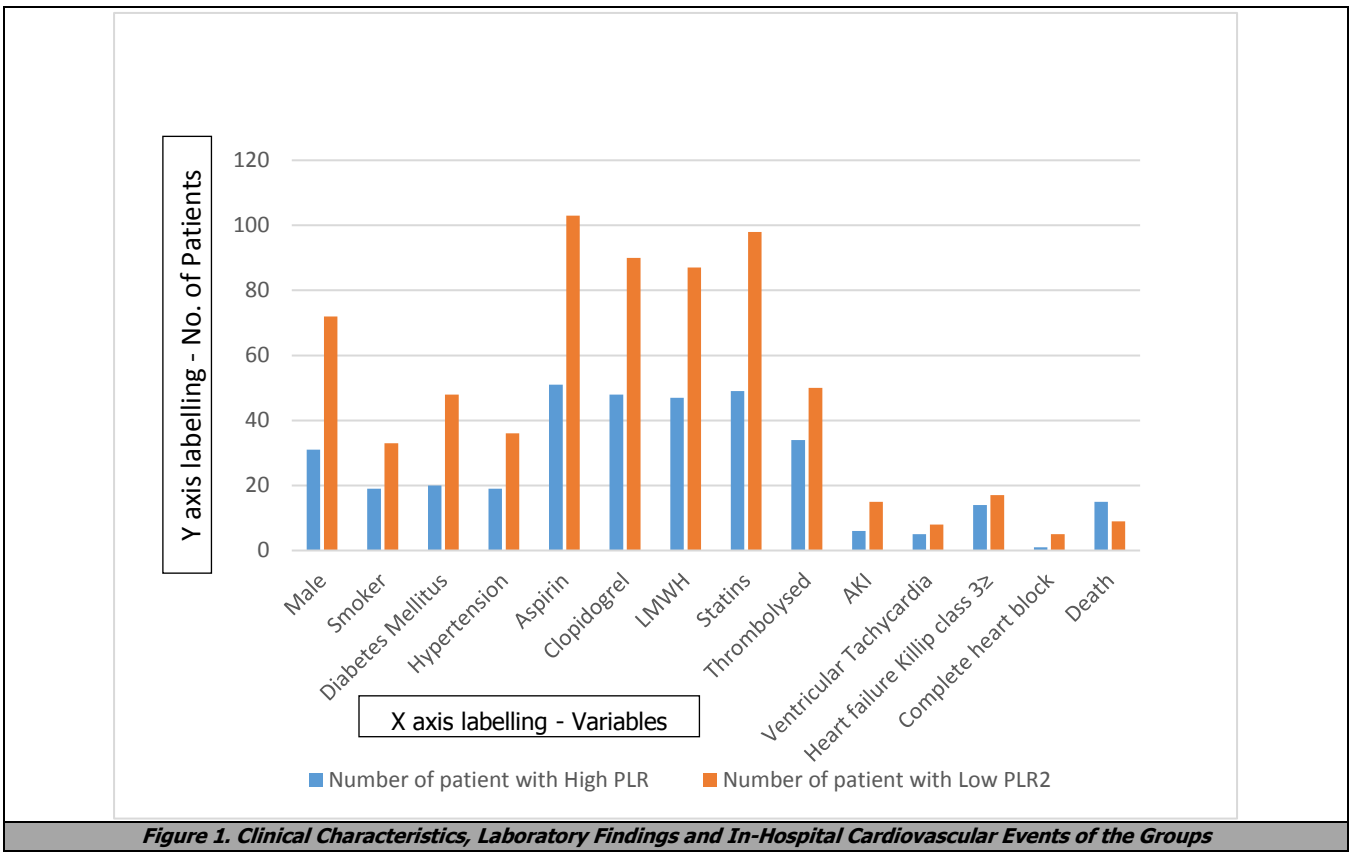

Figure 1. Clinical Characteristics, Laboratory Findings and In-Hospital Cardiovascular Events of the Groups

Variable	High PLR (n = 52) n (%)	Low PLR (n = 104) n (%)	P Value
Male	31 (59.61)	72 (69.23)	0.232
Current smoker	19 (36.53)	33 (31.73)	0.548
Diabetes mellitus	20 (38.46)	48 (46.15)	0.361
Hypertension	19 (36.53)	36 (34.61)	0.813
Aspirin	51 (98.07)	103 (99.03)	0.615
Clopidogrel	48 (92.3)	90 (86.53)	0.288
Low molecular weight heparin	47 (90.38)	87 (83.65)	0.255
Statins	49 (94.23)	98 (94.23)	1
Thrombolysed	34 (65.38)	50 (48.07)	0.041
Acute kidney injury	6 (11.53)	15 (14.42)	0.619
Ventricular tachycardia	5 (9.61)	8 (7.69)	0.682
Heart failure Killip class 3 ≥	14 (26.92)	17 (16.34)	0.119
Complete heart block	1 (1.92)	5 (4.80)	0.377
Death	15 (28.84)	9 (8.65)	0.001

Table 1. Clinical Characteristics, Laboratory Findings and In-Hospital Cardiovascular Events of the Groups

Both univariate and multivariate analysis were done to assess correlation. Backward stepwise multivariate logistic regression was used to predict the factors associated independent in-hospital mortality. Heart failure Killip class 3 ≥, high PLR and acute kidney injury were found to be associated with increased in-hospital cardiovascular mortality in a univariate logistic regression analysis. Table 2 shows univariate analyses for risk factors of in-hospital cardiovascular mortality.

PLR >148.4 was found to be an independent predictor of in-hospital cardiovascular mortality in multivariate analyses (hazard ratio: 13.222 (2.113 -21.749) P = 0.006 with 95 % confidence interval). Other independent predictors of cardiovascular mortality were AKI, ventricular tachycardia and heart failure Killip class 3 ≥. Acute kidney injury had hazard ratio of 15.034 (5.584 -15.412) P = 0.001 with 95 % confidence interval. Ventricular tachycardia had hazard ratio of 9.014 (2.141 -17.014) P = 0.001 with 95 % confidence interval.

Variable	HR (% 95CI)	P Value
Age ≥ 60 years	2.000 (0.801-4.992)	0.137
Male	1.200 (0.487-2.958)	0.692
Diabetes mellitus	0.911 (0.378-2.200)	0.836
Systemic hypertension	0.721 (0.279-1.861)	0.499
Smoking history	1.531 (0.628-3.728)	0.349
> 12 hours of symptoms	1.050 (0.427-2.581)	0.915
No thrombolytic therapy	0.985 (0.411-2.357)	0.973
Acute kidney injury	6.000 (2.169-16.596)	0.001
Ventricular tachycardia	2.733 (0.768-9.724)	0.120
Heart failure Killip class ≥ 3	9.462 (9.261-12.428)	< 0.0001
High PLR	4.279 (1.723-10.626)	0.002

Table 2. Univariate Analyses for Risk Factors of In-Hospital Cardiovascular Mortality

HR: hazard ratio, PLR: platelet and lymphocyte ratio, CI: confidence interval.

Variable	HR (% 95 CI)	P Value
Age ≥ 60 years	1.077 (0.232-5.000)	0.924
Acute kidney injury	15.034 (5.584-15.412)	0.001
Ventricular tachycardia	9.014 (2.141-17.014)	0.001
Heart failure Killip class ≥3	14.495 (2.727-19.041)	<0.001
High PLR	13.222 (2.113-21.749)	0.006

Table 3. Independent Predictors of In-Hospital Cardiovascular Mortality

HR: hazard ratio, PLR: platelet and lymphocyte ratio, CI: confidence interval.

Heart failure Killip class ≥ 3 had hazard ratio of 14.495 (2.727 - 19.041) P < 0.001 with 95 % confidence interval.

Table 3 shows independent predictors of cardiovascular mortality in the hospital. In ROC curve analyses, a PLR value of 148.4 for in-hospital mortality rate had sensitivity of 62.5 % and a specificity of 72 % (area under the curve = 0.627, 95 % confidence interval 0.485 – 0.769). Figure 2 shows receiver operating characteristic (ROC) curve for the platelet/lymphocyte ratio.

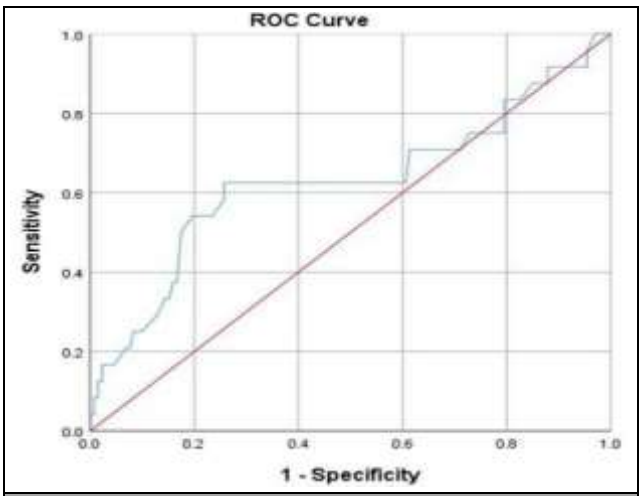

Figure 2. Receiver Operating Characteristic (ROC) Curve for the Platelet/Lymphocyte Ratio

DISCUSSION

Atherosclerotic coronary artery disease is the most common cause of mortality and morbidity in developed countries and even in developing countries. Most of STEMI occur due to rupture of atheromatous plaque, this exposes the sub endothelial surface and this is a pro-thrombotic state, there will be platelet adhesion, aggregation and clot formation.[12] STEMI occurs due to complete obstruction of the coronary arteries by thrombus whereas partial coronary obstruction leads to non STEMI or unstable angina. Activated platelets precipitate to produce inflammatory substances from endothelial cells and leukocytes leading to adhesion and transmigration, and thus increase the inflammatory process and progression of atherosclerotic plaque.[12]

Platelets play a major role in inflammation by releasing mediators of inflammation and play a role in formation and rupture of atherosclerotic plaque. Several studies have demonstrated the association between elevated platelet count and adverse cardiovascular outcomes.[12,13] Study conducted by Thaulow E et al.[13] showed an association between platelet concentration and aggregebility with long-term incidence of fatal coronary artery disease in a population of middle aged apparently healthy male. According to study conducted by Iijima R et al.[12] in patients with coronary artery disease who are undergoing per-cutaneous intervention (PCI) after pre-treatment with clopidogrel of 600 mg, baseline platelet count predicted the 30-day mortality.

According to study conducted by Lordkipanidze M et al. raised platelet count had been independently associated with an inadequate response in patients who were receiving aspirin.[14] Relative lymphocyte count is a simple, easily available and cost effective marker that can be used in high risk patients to determine the cardiovascular morality. Low peripheral blood lymphocyte count was associated with adverse cardiovascular outcomes. The exact mechanism by which low relative lymphocyte count is associated with cardiovascular mortality is unknown.[15] According to study conducted by Gary et al. showed that higher platelet volume may change blood viscosity and increase inflammation. This predisposes to thrombus formation especially after plaque rupture.[16] Davi et al. showed that elevated platelet count is associated with higher rates of cardiovascular events.[17] According to Basem Azab et al. PLR was independent predictor of long-term mortality after non ST elevation myocardial infarction.[18] According to study conducted by Vakili H et al. showed higher PLR was positively correlated with corrected thrombolysis in myocardial infarction (TIMI) frame count.[19]

A retrospective study performed in Turkey by Ahmet Temiz et al.[10] showed higher PLR was associated with increased in-hospital mortality in patients with ST elevation myocardial infarction. Our study adopted similar methodology of Ahmet Temiz et al.[10] however ours was a prospective observational study. Sample size of Ahmet Temiz et al.[10] was 636 whereas our sample size was 156. Mean age in our study was 59 ± 10 years whereas in Ahmet Temiz et al.[10] it was 62 ± 12 years. According to Ahmet Temiz et al.[10] independent predictors of cardiovascular mortality were high PLR (> 144), age ≥ 70, non-thrombolysed patients and glomerular filtration rate (GFR) < 60 ml/min.

In our study, independent predictor cardiovascular mortality were acute kidney injury, ventricular tachycardia, heart failure Killip class ≥ 3 and PLR > 148.4. In our study, there was no significant correlation between age and cardiovascular mortality. Similarly in our study there was no significant correlation between thrombolysed patients and cardiovascular mortality. Present study showed that PLR > 148.4 is an independent predictor of cardiovascular mortality in patients with ST-elevated myocardial infarction.

CONCLUSIONS

In our study, higher PLR had significant association with in-hospital mortality in patients with STEMI. In our study other independent factors associated with mortality were acute kidney injury (AKI), ventricular tachycardia and Killips class 3 and more. However, large multi-centric studies involving all patients with acute coronary syndrome are required to see association between PLR and mortality rate before it can be implemented in clinical practice. There is need for studies that correlate value of PLR ratio and mortality in patients with unstable angina and non ST elevation myocardial infarction.

Limitations of the Study

1. Small sample size of study.
2. It is a single centred study.
3. There was short duration of follow-up in the study.
4. Only STEMI patients were included in the study.

Data sharing statement provided by the authors is available with the full text of this article at jebmh.com.
Financial or other competing interests: None.
Disclosure forms provided by the authors are available with the full text of this article at jebmh.com.

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