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Laboratory Predictors of High Thrombus Burden in Acute ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Intervention

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ABSTRACT

Background and Aim : A risk factor for stent thrombosis, no-reflow, and unfavorable outcomes is still intracoronary thrombus. Hematological markers' diagnostic utility in cardiovascular diseases (CVDs) is an important field for research. So our aim is to ascertain the association between various hematological markers and the high thrombus burden.

Methods : This cross-sectional study included 124 cases of acute ST-elevation myocardial infarction (STEMI) and was classified into two groups ; 54 cases in the group (I) had a low thrombus burden, whereas 70 cases in group (II) had a high thrombus burden. Electrocardiography (ECG), echocardiography, complete blood count (CBC) sample, lipid profile, and troponin were gathered.

Results : Group II had lower high-density lipoprotein (HDL-C) with a significant difference of $p<0.001$. Significant higher white cell counts (WBC) count, monocytes, relative distribution width (RDW), neutrophile to lymphocytes ratio (NLR), monocytes to high-density lipoprotein ratio (MHR), and troponin were also in favor of group II. MHR at odds ratio 0.35 with 95% confidence interval (CI) and troponin at odds ratio 0.659 were significant predictors of high thrombus burden. MHR sensitivity was 78% , specificity was 65%, and $p<0.001$, while troponin sensitivity was 74%,specificity was 56% , and $p<0.001$. The cutoff values were 0.026 and 3.2 for MHR and troponin respectively.

Conclusion: Troponin and MHR were simple, sensitive, and low-cost markers, might be helpful in predicting the high thrombus burden.

Keywords: Predictors; Monocytes to high-density lipoprotein (MHR); High thrombus burden

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) has been associated with a high rate of morbidity and mortality [(1)]. The pathophysiology behind acute coronary syndrome is made up of intracoronary thrombus formation on top of atherosclerotic plaque rupture and a decrease in coronary blood flow [(2)]. Intracoronary thrombus burden remains a risk factor for long-term adverse cardiovascular events, stent thrombosis, no-reflow, and distal embolization [(3)]. Hematological markers, predictive and even diagnostic value in cardiovascular diseases (CVDs) is a major field of research [(4)].

Decreased relative distribution width (RDW) is related to a lower likelihood of adverse events in patients with acute myocardial infarction [(5)]. Leukocytes are considered essential for thrombus formation as well as plaque activation in acute coronary syndrome (ACS). The most reliable indicator of unfavorable outcomes has been proven to be the neutrophil-lymphocyte ratio (NLR) among the markers of inflammation in ACS [(6)]. Monocytes play a critical role in atherosclerosis by releasing cytokines that are pro-inflammatory and pro-oxidant [(7)].

Additionally, high-density lipoprotein-cholesterol (HDL-C) has a cardioprotective role by preventing the oxidation of low-density lipoprotein (LDL) and monocyte activation [(8)]. High values of the monocyte to high-density lipoprotein cholesterol ratio (MHR) are associated with unfavourable outcomes and severe coronary artery disease [(9)]. So, we aimed to ascertain the relationship between the different hematological indices and the high thrombus burden in acute STEMI managed by primary percutaneous coronary intervention (PPCI).

PATIENTS AND METHODS

This cross-sectional study was conducted at Zagazig University Hospital from (July 2020 to December 2022) on acute STEMI patients presented within the first twelve hours after the chest pain beginning and then managed with PPCI. STEMI was defined as typical cardiac chest pain and new ST-elevation in 2 contiguous leads with more than 1 mm in all leads except leads V_2–V_3 or the presence of new-onset left bundle branch block with a rise of (CK-MB or troponin) [(10)]. Participants who had blood transfusions in the last three months, who had any type of inflammatory disease or any type of blood disorder such as anemia or patients who had received thrombolytic therapy within the previous 24 hours were all excluded from the study.

Electrocardiogram parameters (ECG):

A twelve-lead ECG was done before the procedure and sixty minutes after the PPCI to assess the infarct site

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and degree of ST-segment resolution, a value of more than or equal to seventy percent was considered successful [11].

Echocardiographic data:

Within the first 24 hours of admission, a transthoracic echocardiographic examination was carried out utilizing a 1.5-3.6 MHz multifrequency phased array probe and a commercial Vivid E9 ultrasound scanner with phased-array transducers from Horton, Norway, for assessment of the left ventricular ejection fraction.

Laboratory data:

Upon admission to the hospital, blood samples were taken. The levels of baseline lipid panel, creatinine, troponin in EDTA-anticoagulated tubes, and samples for the complete blood count assay were gathered. Hemoglobin, white blood cells, neutrophils, lymphocytes, monocytes, RDW, platelet counts, and mean platelet volume were all analyzed using an automated Beckman Counter analyzer, *California.*

Coronary Angiography and PPCI procedure:

At the time of hospital admission, all patients received a loading dose of 600 mg of clopidogrel and 300 mg of aspirin as well as 70 IU/kg of unfractionated heparin during PCI. The culprit artery only was accessed by PPCI unless there was a cardiogenic shock. The coronary angiography was carried out utilizing the conventional Judkins technique. PPCI was carried out utilizing a six- or seven-French catheter and a traditional radial or femoral route was used. All patients had their stents deployed. Drug-eluting stents, balloon pre-dilatation or post-dilatation, the use of aspiration catheter, and the administration of glycoprotein IIb/IIIa inhibitors were up to the decision of the cardiologist.

The thrombus grade was assessed after restoring antegrade flow through balloon dilatation or guidewire. Angiographic thrombus burden was classified according to the thrombolysis in myocardial infarction (TIMI) study [12]. Grade 0: no evidence of thrombus, grade one: suspected thrombus (low contrast density), grade two: definite thrombus and the largest thrombus diameter is less than or equal to half vessel diameter, grade three: definite thrombus and the largest thrombus diameter is more than half to less than two vessel diameters, grade four: definite thrombus and the largest thrombus diameter more than two vessel diameters, and grade five: completely occlusive thrombus.

Our participants were divided into 2 groups based on the aforementioned grades group I of low thrombus burden (grades 0-three) and group II of high thrombus burden (grades four and five).

Ethical standards:

Our study was approved by our university and it took official approval (NO. ZU-IRB # 623112-7-2020). Before participants were enrolled in our study, we obtained written informed consent from each participant after telling them of the study's purpose. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Sample size and technique:

A previous study **Wang *et al.*** [13] reported that the monocyte count in groups with low versus high thrombus burden was (0.53± 0.2 versus 0.61 ±0.2) with a confidence interval (CI) of 95%. Based on this, the sample size was calculated to be 124 patients using open EPI.

Statistical analysis

The SPSS (21) program was used to analyze the data. Quantitative data were displayed as mean and standard deviation (SD), and were compared by the Student t-test.

Mann-Whitney test was used for data which were not normally distributed. Qualitative data were displayed as frequency and percentage and were compared by chi-square test. For the purpose of predicting characteristics related to a high thrombus burden, multinomial logistic regression was applied. Using a receiver operating characteristic curve analysis, a cutoff for the predictive factors was established. P value less than 0.05 was considered significant.

RESULTS

We enrolled 124 cases of acute STEMI who were divided into two groups, group (I) had 54 cases with low thrombus burden whereas 70 cases in group (II) had high thrombus burdens. No significant difference as regard age, sex, or risk factors was found (Table 1).

Table 1: Demographic and risk factors of the study groups

Variable		Group I Low Thrombus Burden (n= 54)	Group II High Thrombus Burden (n= 70)	P value
Age (years)	Mean ± SD	57.7± 8.5	55.9± 11.8	0.345
Gender	Male, n (%)	38 (70.4)	40 (57.1)	0.131
	Female, n (%)	16 (29.6)	30 (42.9)	
Hypertension	No, n (%)	23 (42.6)	34 (47.8)	0.508
	Yes, n (%)	31 (57.4)	36 (52.2)	
Diabetes	No, n (%)	31 (57.4)	39 (55.1)	0.850
	Yes, n (%)	23 (42.6)	31 (44.9)	
Dyslipidemia	No, n (%)	26 (48.1)	10 (14.3)	<0.001
	Yes, n (%)	28 (51.9)	60 (85.7)	
Family history of premature CAD	No, n (%)	25 (46.3)	39 (55.1)	0.298
	Yes, n (%)	29 (53.7)	31 (44.9)	
Smoking	No, n (%)	31 (57.4)	39 (55.7)	0.850
	Yes, n (%)	23 (42.6)	31 (44.3)	

CAD: coronary artery disease.

Group II had lower ST-segment resolution, ejection fraction (EF), and HDL-C with a significant difference but higher WBC count, monocytes, RDW, NLR, MHR, and troponin were also in favor of group II with a significant difference (Table 2).

Table 2: Electrocardiographic, echocardiographic, and laboratory data of the study groups

Variable		Group I Low Thrombus Burden (n= 54)	Group II High Thrombus Burden (n= 70)	P value
Site	Anterior	34 (63)	46 (65.7)	0.494
	Inferior	20 (37)	22 (31.4)	
	Lateral	0	2 (2.9)	
STR> 70%	No	0	24 (34.3)	**<0.001**
	Yes	54 (100)	46 (65.7)	
LVEDD (mm)	Mean ± SD	55.1± 6.6	64.8± 3.7	**<0.001**
LVESD (mm)	Mean ± SD	32.8± 4.2	43.6± 3.8	**<0.001**
EF (%)	Mean ± SD	53.7± 6.5	42.9± 2.6	**<0.001**
Hb (g/dl)	Mean ± SD	13.6± 0.7	13.4± 0.5	0.066
WBC ($X10^3$)	Mean ± SD	9.5± 0.9	11.3± 1.5	**<0.001**
Platelet ($X10^3$)	Mean ± SD	320.6± 74.8	332.2± 69.6	0.373
Mean platelet Volume (fl)	Mean ± SD	9.1± 1.1	9.3± 0.6	0.198
Neutrophil (per mm^3)	Mean ± SD	7.3± 0.7	7.6± 0.8	0.031
Monocytes (per mm^3)	Mean ± SD	1.1± 0.7	1.5± 0.9	**0.002**
RDW (%)	Mean ± SD	14.8± 0.8	15.1± 0.8	**0.041**
Lymphocytes ($X10^3$/µL)	Mean ± SD	3.8± 0.8	3.6± 0.7	0.141
MHR	Mean ± SD	0.02± 0.01	0.04± 0.03	<**0.001**
NLR	Mean ± SD	2.0± 0.6	2.3± 0.5	**0.027**
HDL-C (mg/dl)	Mean ± SD	67.8 ± 2.6	52.8 ± 6.2	**0.001**
Triglyceride (mg/dl)	Mean ± SD	162.5 ± 5.2	196.7 ± 5.3	0.050
LDL (mg/dl)	Mean ± SD	122.8± 8.9	153.6± 6.8	0.152
Troponin (ng/ml)	Mean ± SD	2.8± 0.3	4.1± 1.0	**<0.001**

STR: ST-segment resolution; **EF**: ejection fraction; **LVEDD**: left ventricular end-diastolic diameter; **LVESD**: left ventricular end-systolic diameter, **Hb**: hemoglobin; **WBC**: white blood cell count; **RDW**: relative distribution width; **MHR**: monocytes high-density lipoprotein ratio; **NLR**: neutrophil-lymphocyte ratio; **HDL**: high-density lipoprotein; **LDL**: low-density lipoprotein

Group II had significantly more prevalent three-vessel diseases than group I. Pre-intervention TIMI 0\I was significantly more prevalent in group II than in group I. Post-intervention TIMI III was significantly lower in group II than in group I. Thrombus aspiration, balloon dilatation, and GBII B/3A use; all were more prevalent in group II in comparison to the group I (Table 3)

Table 3: Angiographic data of the study groups

Variable		**Group I Low Thrombus Burden (n= 54)**	**Group II High Thrombus Burden (n= 70)**	**P value**
Culprit lesion	LAD	32 (59.3)	42 (60)	0.287
	LCX	2 (3.7)	4 (5.7)	
	LAD+ LCX	0	2 (2.9)	
	LM	0	2 (2.9)	
	LM+ LAD	2 (3.7)	0	
	RCA	18 (33.3)	30 (28.6)	
No. of vessels	SVD	30 (55.6)	14 (20)	**<0.001**
	DVD	20 (37.0)	30 (42.9)	
	TVD	4 (7.4)	26 (37.1)	
TIMI flow (pre)	0\I	19 (35.1)	60 (85.7)	**<0.001**
	II	13 (24.1)	3 (4.3)	
	III	22 (40.7)	7 (10)	
TIMI flow (Post)	II	4 (7.4)	19 (27.1)	**0.005**
	III	50 (92.6)	51 (72.9)	
MBG	1	1 (1.9)	2 (2.9)	0.148
	2	13 (24.1)	28 (40)	
	3	40 (74.1)	40 (57.1)	
Direct stents	N %	22 (40.7)	2 (2.9)	**<0.001**
Thrombus aspiration	N %	10 (18.5%)	50 (71.4%)	**<0.001**
Balloon use	N %	32 (59.3)	68 (97.1)	**<0.001**
GBIIb/IIIa	N %	8 (14.8)	40 (57.1)	**<0.001**

TIMI: thrombolysis in myocardial infarction; **MBG**: Myocardial Blush grade, **GBIIb/IIIa**: glycoprotein IIb/IIIa; **LAD**: left anterior descending; **LCX**: left circumflex; RCA: right coronary artery; **LM**: left main; **SVD**: single vessel disease; **DVD**: double vessel disease; **TVD**: triple vessel disease.

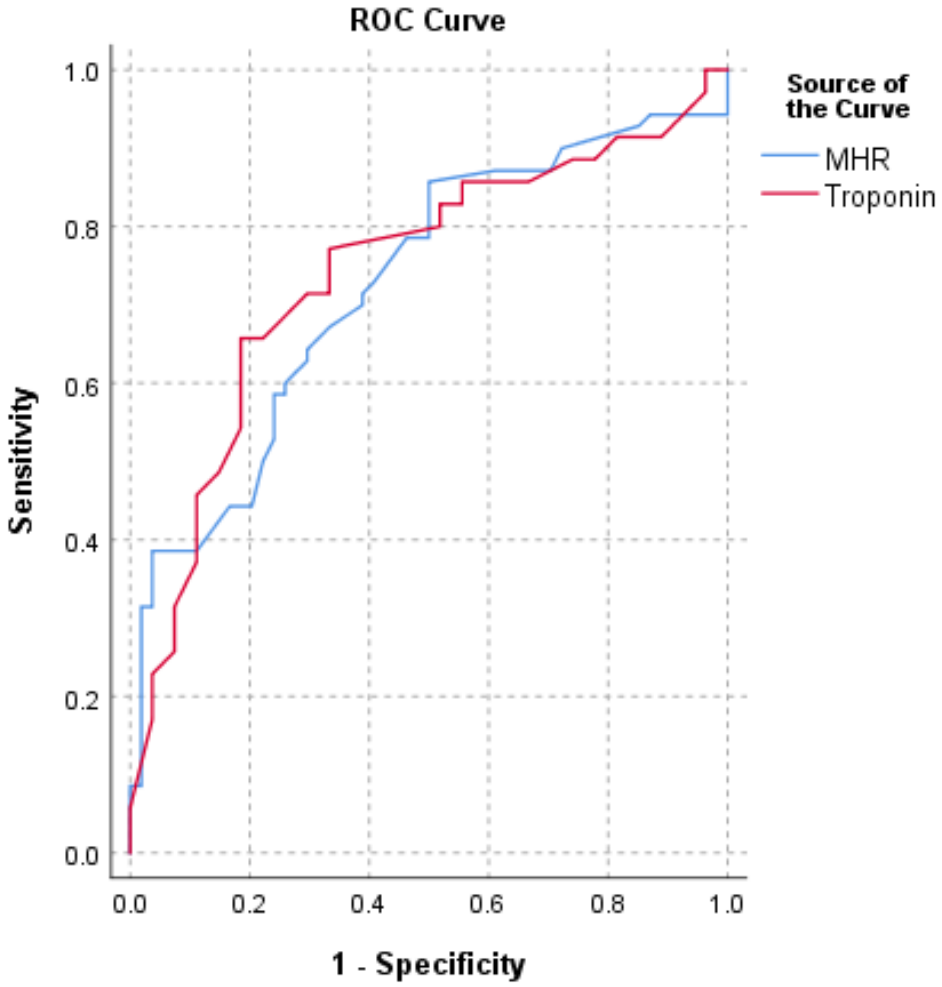
According to multivariate regression, MHR at odds ratio 0.35 with 95% confidence interval (CI) and troponin at odds ratio 0.659 were significant predictors of high thrombus burden (Table 4)

Table 4: Multivariate logistic regression analysis to detect predictors of high thrombus burden

Variable	**Odds ratio (95% CI)**	**P value**
WBCs	0 (0-9226.1)	0.112
Monocytes	1.95 (0.451- 8.44)	0.371
RDW	849 (0.458- 1.574)	0.604
MHR	0.35 (0.218- 0.579)	**0.002**
Lymphocytes	0.739 (0.145- 3.751)	0.715
NLR	0.270 (0.028- 2.606)	0.258
Troponin	0.659 (0.465- 0.936)	**0.020**
HDL-C	0.979 (0.946-1.015)	0.248

WBC: white blood cell count; **RDW**: relative distribution width; **MHR**: monocytes to high-density lipoprotein ratio; **NLR**: neutrophil-lymphocyte ratio; **HDL**: high-density lipoprotein.

MHR's area under the curve (AUC) was 0.725, sensitivity was 78% , specificity was 65%, p<0.001 ,and positive predictive value (PPV) 89%, negative predictive value (NPV) was 77%while troponin's(AUC) was 0.741,sensitivity was 74%,specificity was 56% , PPV was 87%, (NPV) was 65% ,p<0.001. The cutoff values were 0.026 and 3.2 for MHR and troponin respectively were presented in (Figure 1)



MHR: monocytes to high-density lipoprotein

Figure 1: ROC curve for MHR and troponin for predicting high thrombus burden.

DISCUSSION

Our study's main findings demonstrated that MHR and troponin were significant predictors of high thrombus burden in STEMI patients. High thrombus burden was associated with higher WBC count, monocytes, RDW, MHR, NLR, and troponin level, low HDL, more 3VD prevalence, impaired TIMI grade pre-procedural, impaired epicardial reperfusion in the form of lower TIMI III flow post-procedural, more balloon dilation, and more GBIIb/IIIa usage.

Even with aspiration catheter and GB IIb/IIIa inhibitors usage, the management of myocardial infarction with a significant thrombus burden remains challenging. In managing STEMI, addressing the variables that affect the burden of intracoronary thrombi may be helpful [14].

Oxidative stress and inflammation are significant contributors to atherosclerosis.

Monocytes had significant breaking points in the development of atherosclerosis by activation and cholesterol oxidation [15] , activated monocytes involve with damaged endothelium and release adhesion molecules and pro-inflammatory cytokines [16]. Several studies [17-19] reported that high monocyte count was related to the advancement of atherosclerotic plaque and ACS. Similarly, we observed that the high thrombus burden group had more circulating monocytes than the low thrombus group.

Multiple research studies have shown that HDL-C has cardioprotective properties, and enhances endothelial function by reducing inflammation and oxidative stress. Additionally, HDLC controls the activation, adhesion, and transmigration of monocytes [20].

Tran-Dinh *et al.* [21] revealed that HDL-C blocks the migration of monocytes into the subendothelium. **Murphy *et al.*** [22] reported the same finding. Therefore, the development, progression, and severity of the atherosclerotic process depend on the quantity of circulating HDL-C and how it relates to the number of monocytes. These studies encouraged us to propose the hypothesis that the intracoronary thrombus burden in STEMI patients may be related to the monocyte count and MHR.

Our study reported low HDL in the high thrombus burden group in comparison to the low thrombus, which indicates that the patients weren't cardioprotected, **Arisoy *et al.*** [23] reported the same finding as ours.

Additionally, our results revealed that MHR and troponin were independent predictors of a high thrombus burden. **Arisoy *et al.*** [23] was parallel to our results. **Ya-Fei *et al.*** [24] also reported that MHR was an accurate predictor of high thrombus burden. **Cetin *et al.*** [25] concluded that the MHR was a significant indicator of stent thrombosis. Also, it worths mentioning that we observed that high thrombus burden was associated with poor epicardial reperfusion in the form of post-procedural poor TIMI III, more balloon dilation, and more GBIIb/IIIa inhibitor usage. This was concordant with **Tanboga *et al.*** [26]

CONCLUSIONS

Troponin and MHR were simple, sensitive, and low-cost markers, that might be helpful in predicting the high thrombus burden. At a cutoff of 3.2 and 0.026 respectively we potentially can identify patients liable to high risk of developing high intracoronary thrombus burden and adjust our treatment to take into account any potential adverse events of the high thrombus burden in STEMI. High thrombus burden was associated with higher WBC count, monocytes, RDW, MHR, NLR, and troponin level, low HDL, more 3VD prevalence, impaired TIMI grade pre-procedural, impaired epicardial reperfusion in the form of lower TIMI III flow post-procedural, more balloon dilation and more GBIIb/IIIa inhibitors usage.

LIMITATIONS

We assessed baseline laboratory markers at admission time only, MHR and other markers might change following the acute period of STEMI. The intracoronary thrombus burden was assessed visually only, and techniques such as intravascular ultrasound or optical coherence tomography would be better. The small sample size was also a limitation.

RECOMMENDATIONS

Future large sample size multi-center studies to confirm our results. Clinical follow-up studies of the association of MHR/troponin with clinical adverse events.

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