Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2021.67250.2169 Volume 30, Issue 1.1, -January 2024, Supplement Issue
Manuscript ID ZUMJ-2103-2169 (R6)
DOI 10.21608/ZUMJ.2021.67250.2169

Original Article

Predictors of High Thrombus Burden in Patients with Acute Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention.

Mohamed Ibrahim Mostafa Alawdi, Ahmed Mohamed Alzayat, Ismail Mohamed Ibrahim and Mohamed Taha Elsaid

Cardiology Department, Faculty of Medicine, Zagazig University

Corresponding author
Mohamed Taha Elsaid
Cardiology resident, Zagazig, Egypt

E-mail:
mtd010151610@gmail.com

Submit Date	2021-03-19
Revise Date	2021-05-10
Accept Date	2021-05-19

ABSTRACT

Objectives: To assess predictors of high thrombus burden in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention, We sought to study simple clinical and laboratory values.

Methods: This cross-sectional study was conducted in cardiology department, Faculty of medicine, Zagazig university hospitals. We studied patients with acute myocardial infarction underwent primary percutaneous coronary intervention attending our coronary care unit in Zagazig university hospitals. We enrolled 180 patients for the study in the period between January 2019 and January 2020.

Results: Patients were classified into two groups: Group 1: Low thrombus burden of culprit vessel, this group included 74 consecutive participants who had thrombus grade less than 4, with age 56.53 ± 5.435 years and were 39 males and 35 females. Group 2: High thrombus burden culprit vessel, which included 106 cases. Patients who had thrombus grade more than or equal 4 with age 57.32 ± 6.6 years and were 57male patients and 49 females. All Patients had PPCI

Conclusion: Initial troponin, pain to balloon time and RDW can be used as predictors of high thrombus burden in acute STEMI patients undergoing primary PCI.

Key words: Primary Percutaneous Coronary, Acute Myocardial Infarction, Infarction Related Artery

INTRODUCTION

Cardiovascular diseases (CVD) such as ischemic heart disease (IHD) represent the leading cause of morbidity and mortality worldwide. In (2005), 17.5 million people died from CVD (30% of all global disease-related deaths), including 7.6 million due to Acute Myocardial Infarction (AMI) [1].

Acute myocardial infarction (MI) is a leading cause of death all over the world and continues to be a significant public health problem in both developed and developing countries. Cardiogenic shock complicates 7-10% of cases of acute myocardial infarction and is associated with 70-80% mortality [2].

It is widely accepted that the most common cause of ACS events is an episode of acute thrombosis on top of coronary atherosclerotic disease. Myocardial infarction may occur without obstructive coronary atherosclerotic disease (MINOCA). But these cases have different treatment and prognosis [3].

Intracoronary thrombus management is still challenging, in spite of recent major pharmacological and invasive improvements, such as glycoprotein IIb/IIIa antagonists or thrombectomy, respectively, in percutaneous coronary intervention (PCI) [4].

Intracoronary thrombus burden is still a risk factor for long-term adverse cardiovascular events, stent thrombosis, no reflow, and distal embolization [5].

PATIENTS AND METHODS

The study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.Patients were classified into two groups according to thrombus burden of culprit lesion: Group 1with Low thrombus burden of culprit vessel, this group consisted of 74 consecutive participants who had thrombus grade less than 4, with age 56.53 ± 5.435 years and were 39 males and 35 females and Group 2: High thrombus burden of culprit vessel, which included 106 cases. Patients who had thrombus grade more than or equal 4 with age 57.32 ± 6.6 years and were 57 male patients and 49 females.

Inclusion Criteria: All patients diagnosed as STEMI and Presented within the first 12 hours from the onset of chest pain. According to **ESC guidelines 2018**, STEMI is defined by STEMI that qualifies patients for primary PCI was defined as persistent chest discomfort for > 20min or other symptoms suggestive of ischaemia and ST-segment elevation >2 mm in at least two contiguous leads or new onset LBBB.

Exclusion Criteria: The patients with baseline anemia (Hb<13 g/dL for males, 12 g/dL for females) were excluded. Anemia on admission was defined as a baseline hemoglobin concentration of less than 13 g/dL in men and less than 12g/dL in women, in accordance with the World Health Organization criteria. Patients who were ineligible for PPCI and patients with severe systemic co-morbidity such as severe liver and kidney diseases were excluded from the study.**Ethical Considerations:** Subjects of the study received full explanation of the study, and a written consent was obtained from each subject. The study was approved by the medical research and ethics committee.

All patients were subjected to **Complete history taking**: Age, sex , History of hypertension: is defined as According to ESC Guidelines it was defined as elevated systolic blood pressure ≥ 140 mmHg, and/or elevated diastolic blood pressure ≥ 90mmHg on three separate measurements, Diabetes: The diagnosis of diabetes mellitus (DM) is based on previous history of DM treated with or without drug therapies or on basis listed by American Diabetes Association,(2010) as: Fasting blood sugar ≥ 126 mg%, 2 hours post-prandial blood sugar ≥ 200 mg% and HbA1C≥6.5%. , Hyperlipidemia: Dyslipidemia is demonstrated by higher levels of small, dense low-density lipoprotein (LDL), lower levels of high-density lipoprotein (HDL) and higher levels of triglycerides are risky. (Smoking status, previous PCI or coronary artery bypass graft (CABG),Family history, presence of pre-infarction angina was defined as one or more occurrences of chest pain similar to the STEMI pain that occurred within 24 hours of infarct onset, pain to balloon time and door to balloon time were calculated. Pain to balloon time is defined as the time interval beginning from the episode of chest pain that led the patient to present to the emergency department to the time of the first balloon inflation and Door to balloon time: The interval starts with the patient's arrival in the emergency department, and ends when a catheter guidewire crosses the culprit lesion in the cardiac cath. lab. Positive family history of coronary artery disease is defined as documented evidence of premature coronary artery disease in a close relative (men<55 and women <65 years of age) Medications such as statins and antiplatelet. CHA2D-VAS2C score was calculated as **Fig. (1)**

General and local examination: Blood pressure, body mass index, heart rate and Killip class as Table. (1).Incidence of Heart Failure in Acute Myocardial Infarction Presence of cardiogenic shock is defined as the presence of peripheral hypoperfusion signs (cold, shivering, paleness, oliguria, loss of consciousness, etc.) accompanied by low-systemic blood pressure (<90 mm Hg) that are resistant to fluid administration and required inotropic therapy and/or intra-aortic balloon pump). Local examination of the heart for cardiomegaly, pulsations, thrills, heart sounds and murmurs.

Electrocardiographic Examination was done on admission by Comen CM 100 device at a paper speed of 25mm/s and amplification of 10mm/mv. 12-lead ECG with the right leads (V3R and V4R) and posterior leads (V7-V9) also were made to view all the surfaces of the heart. If the initial ECG was not diagnostic but the patient remained symptomatic and there was high suspicion for ACS, serial ECG and continuous monitoring was performed. ST-segment shifts were measured 20 ms after j point for ST-segment elevation using the preceding TP segment as the baseline. Presence of ST segment elevation at least 1 mm (2 mm for V1-V3) in 2 or more contiguous leads in electrocardiogram (ECG) or new onset of left bundle-branch block is reported. Calculation of number of leads with ST segment elevation and summation of ST elevation are done**CBC** Blood samples were obtained on admission or shortly after admission. Hemoglobin, White blood cells, Neutrophil–lymphocyte (NLR) ratio .Red cell distribution width (RDW) was defined as a measure of the range of variation of red blood cell (RBC) volume that is reported as part of a standard complete blood count. Usually red blood cells are a standard size of about 6-8 μm in diameter, and ranged between (11.5:14.5)%.Mean platelet volume was defined as is a machine-calculated measurement of the average size of platelets found in blood and is typically included in blood tests as part of the CBC, the typical range of platelet volumes is 9.4–12.3 fL. Since the average platelet size is larger when the body is producing increased numbers of platelets, the Mean platelet volume (MPV) test results can be used to make inferences about platelet production in bone marrow or platelet destruction problems. Platelet Counts.Platelet distribution width was defined as a measurement of platelet anisocytosis calculated from the distribution of individual platelet volumes, typical range (8.3-25.0) fL. Thrombocrit (or plateletcrit) is the percentage of blood volume occupied by platelets and is an assessment of circulating platelet mass. All CBC parameters were measured as part of the

automated complete blood count (CBC) using a Coulter LH 780 Hematology.

Kidney Function Test including serum uric acid,An estimated glomerular filtration rate (GFR) calculated from serum creatinine using the Cockcroft- Gault equation ([140 _ age] _ [weight in kg] _ [0.85 if female]/ [72 _ creatinine],Cardiac Enzymes including Initial and peak hs-**troponin and CK-MB Lipid Profile**, Non fasting lipogram within 24 hours from the onset of chest pain,HbA1C and RBG were done for all patients .Femoral approach was used **for Coronary Angiography and PPCI** using the standard Judkins technique. Angiographic coronary thrombus burden is scored based on 5grades: as in **Table (2),**After restoring antegrade flow through guide wire or small balloon dilatation in patients with TIMI thrombus grade 5 and coronary angiogram enabled re-stratification of the underlying residual thrombus (final TIMI thrombus grade) .All patients received dual antiplatelet (aspirin and clopidogrel) as pre-medications before PPCI. Use of aspiration (thrombectomy) devices was left to the discretion of the interventional cardiologist. After PPCI, all patients were moved to CCU and received the standard post-STEMI medications as per guidelines.

Statistical analysis

Statistical analysis for the collected data had performed using statistical package for the social sciences (SPSS) version 23. Normally distributed numerical data had described as mean±standard deviation (SD), non-normally distributed numerical data had described as median, range, while categorical data had described as number, percentage.

RESULT

There was statistically nonsignificant difference between both groups regarding age, sex, DM, HTN and dyslipidemia. There was statistically highly significant difference between the two studied groups as regard pain to balloon time and Statistically significant difference as regard Killip class >II, family history of premature coronary artery disease and CHA2DS2-VASC score but regarding previous PCI, previous CABG, pre-infarction angina and door to balloon time there was statistically non-significant difference between both groups in **Table 3**.

There was statistically nonsignificant difference between both groups regarding number of leads with ST segment elevation (STE) and sum of STE. There was statistically highly significant difference between the two studied groups as regard RDW and NLR but regarding Hb, Hematocrit, MPV, Platelet count and WBCs there was statistically nonsignificant difference between both groups **in Table 3**.

There was statistically nonsignificant difference between both groups regarding HDL-C and LDL-C. There was statistically highly significant difference between the two studied groups as regard Initial troponin, Peak troponin, Initial Ck-MB, and Peak CKMB **in Table 3**.

There was statistically highly significant difference between the two studied groups as regard serum uric acid but regarding eGFR there was statistically non-significant difference between both groups. There was statistically nonsignificant difference between both groups regarding RBG and HbA1c.

Regarding univariate logistic regression in **Table 4**, predictors of high thrombus burden that showed high statistically significance include pain to balloon time, RDW, NLR, serum uric acid, initial troponin, initial CKMB, and that showed statistically significance include CHA2DS2-VASC score, family history of premature coronary artery disease, and Killip class >II.

Regarding multivariate logistic regression in **Table 5,** predictors of high thrombus burden that showed statistically significance include pain to balloon time, RDW, initial troponin, and initial CK-MB but that showed statistically non-significance include CHA2DS2VASc score, NLR, Killip class>II, family history of premature CAD, and serum U. A. Roc curve showed cut-off value (1110.5) pg./mL for initial troponin to predict high thrombus burden with sensitivity (89.60%) and specificity (79.40%) and AUC (0.853). Roc curve showed cut-off value (140) minutes for pain to balloon time to predict high thrombus burden with sensitivity (81%) and specificity (82%) and AUC (0.853). Roc curve showed cut-off value (17.6) %for RDW to predict high thrombus burden with sensitivity (80%) and specificity (60.80%) and AUC (0.696)

Table (1) Incidence of Heart Failure in Acute Myocardial Infarction (Mello et al. 2014)

Killip Class	Characteristics	Patients (%)
	No evidence of congestive heart failure	**85**
	Rales, ↑ jugular venous distention, or S_3	**13**
	Pulmonary edema	**1**
	Cardiogenic shock	**1**

Table (1): Grades of thrombus burden:

Grade	Description	
Zero	No angiographic characteristics of thrombus are present.	
One	Possible thrombus is present, with such angiography characteristics: -reduced contrast density, haziness, irregular lesion contour, -or a smooth convex meniscus at the site of total occlusion suggestive but not diagnostic of thrombus; in thrombus.	Small thrombus burden
Two	Definite thrombus, with greatest linear dimensions<1\2 vessel diameter.	
Three	Definite thrombus, with greatest linear dimensions>1\2 vessel diameter but <2vessel diameters.	
Four	Definite thrombus, with greatest linear dimensions> 2vessel diameters.	Large thrombus burden
Five	Total occlusion (unable to assess thrombus burden due to total vessel occlusion).	

Table 3. Baseline Clinical and Laboratory Characteristics According to Thrombus Burden:

		Group (Total =180)		X2 value	P
		low thrombus burden n=76	high thrombus burden n=106		
Age, years		56.53±5.435	57.32±6.686	.845	.399
Sex	Male, n (%)	39(52.7%)	57(53.8%)	0.02	0.887
	Female, n (%)	35(46.2%)	49(46.2%)		
Smoking, n (%)		34(45.9%)	39(36.8%)	1.1515	0.218
Dyslipidemia, n (%)		37(50%)	52(41.1%)	0.016	0.901
HTN, n (%)		48(64.9%)	57(53.8%)	2.206	0.138
DM, n (%)		41(55.4%)	47(44.3%)	2.136	0.144
Killip class (>II), n (%)		32(43.2%)	67(63.2%)	7.018	0.008
Family history of premature coronary artery disease, n (%)		26(35.1%)	56(52.8%)	5.501	0.019
Previous PCI, n (%)		17(23%)	35(33%)	2.141	0.143
Previous CABG, n (%)		8(10.8%)	5(4.7%)	2.415	0.12
Pre-infarction angina, n (%)		36(48.6%)	43(40.6)	1.156	0.282
Pain to balloon time, minutes		100.53±62.906	201.23±63.386	10.52	<.001
Door to balloon time, minutes		29.49 ±3.393	30.34±3.564	1.611	0.109
CHA2DS2-VASC score,		4.04 ±.883	4.46±.886	3.148	0.002
Number of leads with STE,		4.72 ±1.854	4.43±1.847	1.007	0.315
Summation of STE, mv,		3.393±1.716	6.18±1.851	0.708	0.48
Hb, g/dL		13.85 ±.917	13.62±1.046	1.517	0.131
Hematocrit, %		3.992±3.992	39.64±4.164	1.209	0.228
RDW, %		15.28 ±2.854	17.92±2.079	6.795	<0.001
NLR		5.32 ±2.571	8.18±2.189	7.808	<0.001
MPV, fl		8.53 ±.503	8.52±.502	0.107	0.915
Platelet count, x1000/uL		254.76 ±24.475	257.57±22.905	0.787	0.432
PDW, %		16.99 ±4.72	17.69±4.576	1	0.319
WBCS, x1000/uL		12.8 ±1.526	13.21±1.596	1.728	0.086
HDL-C, mg/dL		32.91 ±3.570	33.43±2.801	1.065	0.289
LDL-C, mg/dL		127.65 ±18.760	132.27±18.375	1.647	0.101
Initial troponin, pg./mL		735.19 ±595	1553.78±511.594	9.868	<0.001

	Group (Total =180)		X2 value	P
	low thrombus burden n=76	high thrombus burden n=106		
peak troponin, pg/mL	4814.84 ±3940	8547.5±2548.773	7.168	<0.001
Initial CKMB, ng/mL	31.43 ±11.259	45±7.851	8.957	<0.001
Peak CKMB, ng/mL	187.45 ±78.408	255.18±46.456	6.666	<0.001
eGFR, mg/min/1.73 m2	86.88 ±9.395	86.54±8.511	0.253	0.8
Serum uric acid, mg/dL	6.18 ±2.388	8.08±1.276	6.24	<.001
Hb A1c, %	8.64 ±1.731	8.22±1.764	1.564	0.12
RBS, mg/dL	264.12 ±97.778	282.32±93.453	1.261	0.209

*DM= diabetes mellitus, HTN= hypertension STE = ST segment elevation, Hb= hemoglubin RDW=red cell destribution width NLR=neutrophils to lymphocytics ratio MPV= Mean Platelet Volume PDW= Platelet Distribution Width WBCS= White Blood Cells.

Table 4. Multivariate Logistic Regression Analysis to Detect the Independent Predictors of High TIMI Thrombus Burden:

	Univariate logistic regression					Multivariate logistic regression				
	B	p value	OR	95% CI for OR		B	p value	OR	95% CI for OR	
				lower	Upper				Lower	upper
Pain to balloon	0.02	<0.001	1.02	1.015	1.025	0.016	<0.001	1.016	1.009	1.023
RDW	0.387	<0.001	1..473	1.295	1.676	0.584	0.050	0.558	0.308	.1.009
NLR	0.428	<0.001	1.534	1.345	1.751	0.103	0.668	0.902	0.563	.1.446
Serum Uric acid	0.534	<0.001	1.706	1.417	2.054	0.348	0.209	0.705	0.41	1.215
Initial troponin	0.002	<0.001	1.002	1.002	1.003	0.003	0.006	1.003	1.001	1.006
Initial CKMB	0.125	<0.001	1.133	1.094	1.175	0.147	0.027	1.159	1.017	1.32
CHA2DS2-VASC score	0.531	0.002	1.701	1.206	2.4	0.222	0.385	1.249	0.757	2.06
Family history of premature coronary artery disease	0.726	0.02	0.484	0.263	0.891	0.828	0.126	2.288	0.793	6.601
Killip class >II	0.813	0.009	0.443	0.242	0.813	0.426	0.416	1.532	0.548	4.279

OR: odds ratio
CI: confidence interval

Table 5. The validity of initial troponin, pain to balloon time and RDW with area under the ROC curve (AUC) as predictor for high thrombus burden:

predictor of High thrombus burden	Area under curve (AUC)	Std. Error	p-value	95% Confidence Interval		cut-off	Sensitivity	Specificity
				lower bound	upper bound			
Initial troponin	0.823	0.034	<.0001	0.756	0.89	1110.5	89.60%	79.40%

predictor of High thrombus burden	Area under curve (AUC)	Std. Error	p-value	95% Confidence Interval		cut-off	Sensitivity	Specificity
				lower bound	upper bound			
pain to balloon time	0.853	0.031	<.0001	0.793	0.914	140	81%	82%
RDW	0.696	0.042	<.0001	0.613	0.778	17.6	80%	60.80%

CHA_2DS_2-VASc risk factor	Points
Congestive heart failure Signs/symptoms of heart failure or objective evidence of reduced left ventricular ejection fraction	+1
Hypertension Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	+1
Age 75 years or older	+2
Diabetes mellitus Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	+1
Previous stroke, transient ischaemic attack, or thromboembolism	+2
Vascular disease Previous myocardial infarction, peripheral artery disease, or aortic plaque	+1
Age 65–74 years	+1
Sex category (female)	+1

CHA_2DS_2-VASc = Congestive Heart failure, hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, and Sex (female).

Fig. (1) CHA2D-VAS2C score(2016 ESC Guidelines for the management of atrial fibrillation)

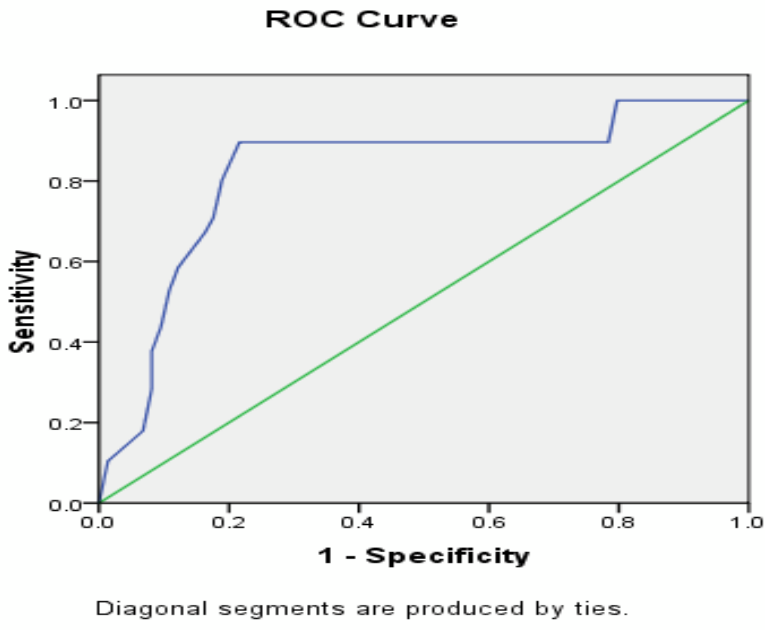

Fig (2): ROC curve of initial troponin with area under the ROC curve (AUC) as a predictor for high thrombus burden with cut-off value 1110.5 pg./mL

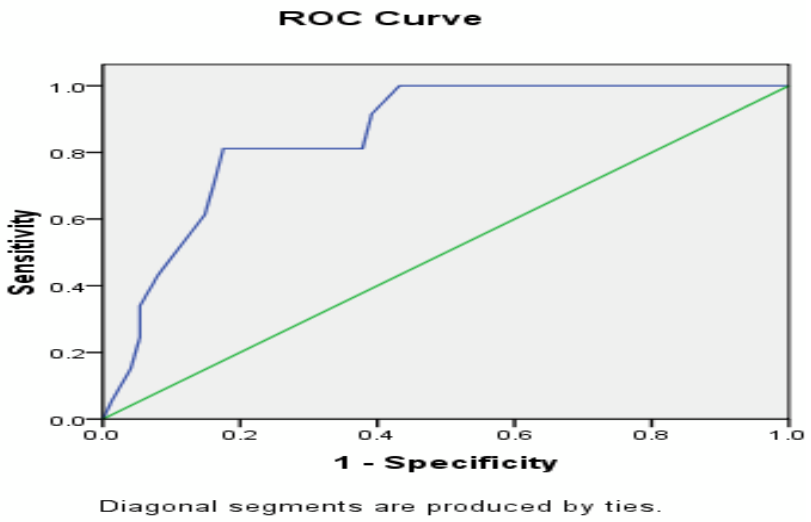

Fig (3): ROC curve of pain to balloon time with area under the ROC curve (AUC) as a predictor for high thrombus burden with cut- off value 240 minutes.

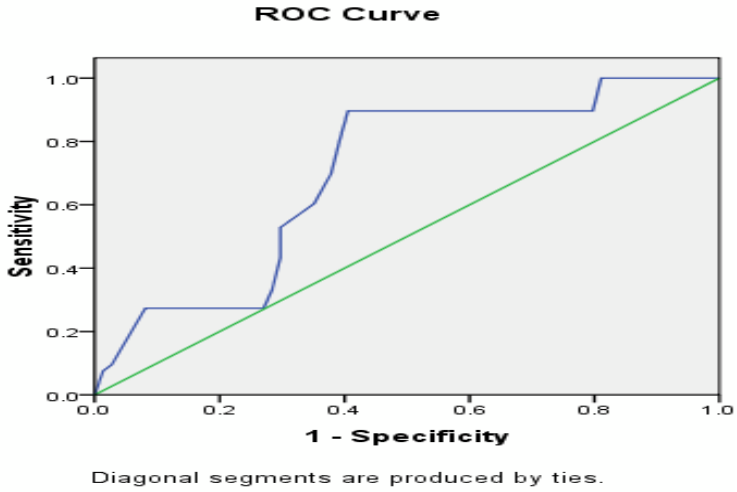

Fig (4): ROC curve of RDW with area under the ROC curve (AUC) as a predictor for high thrombus burden with cut-off value 17.6%

DISCUSSION

Acute myocardial infarction (MI) is a leading cause of death all over the world and continues to be a significant public health problem in both developed and developing countries. Cardiogenic shock complicates 7-10% of cases of acute myocardial infarction and is associated with 70-80% mortality [2].

Treatment of myocardial infarction has undergone profound changes since the bed rest era of the 1960s. At that time, there was debate whether coronary thrombosis was the cause or the consequence of MI. In the 1960s and 1970s, discussion focused on how long to keep patients in bed (a month was not uncommon) and whether coronary care units reduced mortality or not. Until early 1980s, no treatment had been shown to reduce mortality or morbidity in the acute phase of MI or to prevent recurrent events[2].

Coronary angioplasty, known as "percutaneous trans-luminal coronary angioplasty" (PTCA), was first developed in 1977 by Andreas Gruentzig. This is then; adopted throughout the world as a treatment for coronary artery disease (CAD) [6].

Achievement of TIMI III flow after thrombolytic therapy in myocardial infarction was reported not to exceed 50% to 60%. The first reported application of primary mechanical reperfusion for acute MI with balloon angioplasty was in 1982. Sooner; catheter-based reperfusion has become increasingly more popular and constitutes an excellent alternative to the more widely available fibrinolytic therapy with 34% relative reduction in mortality in favor of PCI (6.5% vs. 4.4%), a 47%

relative reduction in non/fatal re-infarction (5.3% vs. 2.9%)[7].

If immediately available, primary PCI should be performed in patients with STEMI who can undergo PCI of the infarct artery within 12 hours of symptom onset, with balloon inflation within 90 minutes of presentation by certain personal and laboratory prerequisites[8].

Intracoronary thrombus management is still challenging, despite recent major pharmacological and invasive improvements, such as glycoprotein IIb/IIIa antagonists or thrombectomy, respectively, in percutaneous coronary intervention (PCI). Intracoronary thrombus burden is still a risk factor for long-term adverse cardiovascular events, stent thrombosis, no reflow, and distal embolization. Therefore, treatments such as glycoprotein IIb/IIIa inhibitors, or procedures such as thrombectomy, which are reducing thrombus burden, improve both epicardial and myocardial perfusion[4].

CHA2DS2-VASc score is easily applied in daily practice to predict thromboembolic risk in atrial fibrillation patients. Furthermore, it predicts major adverse cardiac events after PPCI, and it is associated with increased 1 y mortality rate in patients with Acute Coronary Syndrome (ACS) [9]. Establishing the predictors of intracoronary thrombus and associated clinical and angiographic conditions, therefore, may provide improvements in its management. To address this issue, we aimed to assess predictors of high thrombus burden in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention.

The current study is a cross-sectional study involving 180 patients with acute myocardial infarction undergoing primary percutaneous coronary intervention, admitted to the Coronary Care Unit in Zagazig University Hospitals in the period between January 2019 and January 2020.

They were classified into two groups:

- **Group 1 (Low thrombus burden of culprit vessel**): This group consisted of 74 consecutive participants who had thrombus grade less than 4, with age of 56.53 ± 5.435 years and were 39 males and 35 females.
- **Group 2 (High thrombus burden of culprit vessel),** which included 106 cases. Patients who had thrombus grade more than or equal 4 with age of 57.32 ± 6.6 years and were 57 male patients and 49 females.

In our study, there were statistically nonsignificant differences between both groups regarding age, sex, DM, HTN and dyslipedimia. So, our patients in both groups were matched for age and sex.

This result was in agreement with **Tanboga et al.,** [10] who investigated the determinants of angiographic thrombus burden in patients with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (pPCI). Study population consisted of 662 patients with STEMI (mean age 54.5 + 11.9 years and 87.3% males).

In our study, there was statistically highly significant difference between the two studied groups as regard pain to ballon time and Statistically significant difference as regard killip class >II, family history of premature coronary artery disease and CHA2DS2-VASC score but regarding previous PCI, previous CABG, pre-infarction agina and door to ballon time there was statistically non significant difference between both groups.

Regarding CHA2DS2-VASc score, **Seyis et al.** [11] suggested that CHA2DS2-VASc score can predict thrombus burden in STEMI patients undergoing PPCI. CHA2DS2-VASc score has an easy to remember formula and can be applied quickly in emergent patients.

Hudzik et al., [12] have evaluated whether CHA2DS and CHA2DS2-VASc score can be used to predict the risk of adverse events among ACS patients. Similar findings were reported. CHA2DS and CHA2DS2-VASc score had prognostic value in ACS patients both with and without atrial fibrillation.

A retrospective analysis of 15681 patients with acute myocardial infarction revealed that CHA2DS2-VASc score was associated with long-term cardiac events such as myocardial infarction and all-cause death and it was found to be a more important predictor in STEMI patients than NSTEMI patients[13].

In a study of 12785 consecutive patients who underwent PCI, CHA2DS2-VASc score predicted all-cause mortality and death and nonfatal myocardial infarction in a significant and linear fashion. CHA2DS2- VASc score correlated significantly with the number of diseased vessels and the severity of coronary artery disease. Also, it is an independent predictor of no-reflow in STEMI patients[14].

Tanboga et al., [10] showed that patients with high-thrombus burden had more family history of coronary artery disease (CAD), longer pain to balloon time, higher Killip class (> II) and pre-infarction agina as different result to our study but regarding previous PCI, previous CABG, pre-infarction angina and door to balloon time there was statistically non-significant difference between both groups.

This is explained by the that longer pain to balloon time leads to more and more thrombus formation

and propagation leading to more myocardial injury and stunning or necrosis leading to heart failure and cardiogenic shosk and more Killip class.

In our study, there was statistically nonsignificant difference between both groups regarding number of leads with STE and sum of STE. This could be explained by the fact that ST elevation reflects transmural ischemia secondary to occlusive thrombus and the evolution of elevation may vary in time. Number of leads with STE and summation of STE may also depend on location of culprit lesion and vessel, independently from thrombus burden.

Our study showed that there was statistically highly significant difference between the two studied groups as regard RDW and NLR but there were statistically non-significant differences between both groups regarding Hb, Hematocrit, MPV, Platelet count and WBCs.

This is explained by the fact that RBCS are important element in throbus formation so increase in their size leads to high thrombus burden and high thrombus burden leads to more myocardial injury leading to increase in neutrophils.

Regarding the RDW is a marker of variation in the size of circulating red cells (anisocytosis) and is routinely reported as a part of routine CBC analysis. The RDW is reported as high in patients with acute coronary syndrome and associated with adverse cardiovascular events[15].

Our study showed that there were statistically nonsignificant differences between both groups regarding HDL-C and LDL-C.

This result was in agreement with **Tanboga et al.,** [10],but showed that patients high-thrombus burden had lower triglycerides. This difference between last study and our study could be attributed to smaller sample size in our case and ethnic difference.

In our study, there was statistically highly significant difference between the two studied groups as regard initial troponin, peak troponin, initial CKMB, peak CKMB and peak troponin / Summation of STE.

This is explained by the fact that high thrombus burden leads to more myocardial injury leading to more elevation in cardiac enzymes.This result was in agreement with **Tanboga et al.,** [10] who showed that patients with high-thrombus burden had baseline CK-MB and baseline troponin; higher peak CKMB, and peak troponin.

Our study showed that there was statistically highly significant difference between the two studied groups as regard serum uric acid. But regarding eGFR, there was statistically non significant difference between both groups. This could be explained by the significant relationship between UA levels and CAD endothelial dysfunction[16], coronary reserve[16], and coronary blood flow in patients undergoing elective angiography[17] and in STEMI patients undergoing primary PCI[18].**Acet et al.,** [19] evaluated the relationship between PLR, NLR, and serum UA in STEMI patients with patent IRAs as confirmed by angiography before receiving primary PCI. They found a significant relationship between UA levels and the degree of myocardial perfusion before primary PCI. Admission UA and PLR values were independent predictors of impaired coronary blood flow.Multivariate logistic regression analysis was performed in order to determine independent predictors of high-thrombus burden (CHA2DS2 VAScscore, Pain to balloon time, RDW, NLR, Killip class >II, family history of premature CAD, initial troponin, troponin, initial CKMB, serum uric acid), initial troponin, pain to balloon time and RDW were determined as independent predictors in our analysis.

Tanboga et al., [10] performed multivariate logistic regression analysis in order to determine independent predictors of high-thrombus burden (family history of CAD, preinfarction angina, previous history of statin use, Killip class, pain to balloon time, door to balloon time, NLR, RDW, TG, baseline CK-MB, and baseline troponin). Only RDW was determined as an independent predictor in the analysis.

Seyis et al., [11] evaluated the role of CHA2DS2-VASc score in predicting the amount of intracoronary thrombus burden in STEMI patients undergoing PPCI. Logistic regression analysis revealed that one-point increament in CHA2DS2-VASc score was associated with three times higher risk of having high thrombus burden in IRA. ROC analysis revealed the cut-off value of CHA2DS2-VASc score>2 as a predictor of high thrombus burden with a sensitivity of 91% and a specificity of 82% (AUC:0.925, 95% CI 0.874-0.961; $p<0.001$).

In our study, the area under the ROC curve of the initial troponin was 0.823 to predict TIMI thrombus burden. The best cut-off value of the initial troponin to predict high TIMI thrombus burden was 1110.5 pg (89.6% sensitivity and 79.4% specificity). The area under the ROC curve of the pain to balloon time was 0.853 to predict high TIMI thrombus burden. The best cut-off values of the pain to balloon to predict high TIMI thrombus burden was 240 minutes (81% sensitivity and of 82% specificity). The area under the ROC curve of the RDW was 0.696 to predict high TIMI thrombus burden. The best cut-off value of the RDW to predict high TIMI thrombus burden was 17.6% (80% sensitivity and of 60.8% specificity).

Tanboga et al., [10] found that the area under the ROC curve of the RDW was 0.733 to predict high TIMI thrombus burden. The best cut-off values of the RDW to predict high TIMI thrombus burden was 15.2% (75% sensitivity and of 57% specificity). The differences between our results and **Tanboga et al.,** [10] may be due to difference in sample size and ethnic differences.

CONCLUSION

Initial troponin, pain to balloon time and RDW were determined as predictors of high thrombus burden in acute STEMI patients undergoing primary PCI.

REFERENCES

1. **Hahn, Sigrid A., and Charles Chandler.** Diagnosis, management of S.T. elevation myocardial infarction: a review of the recent literature, practice guidelines, The Mount Sinai journal of medicine, New York. 2006; 73: 469-81.
2. **Jean-Pierre Bassand, Nicolas Danchin, Gerasimos Filippatos, Anselm Gitt, Christian Hamm, Sigmund Silber, et al.** Implementation of reperfusion therapy in A.M.I. A policy statement from the European Society of Cardiology. Eur Heart J 2005; 26:2733.
3. **Thygesen, Kristian, Joseph S. Alpert, Allan S Jaffe, Bernard R Chaitman, Jeroen J Bax, et al.** Fourth universal definition of myocardial infarction. Journal of the American College of Cardiology, 2018; 72: 2231-64.
4. **Charis Costopoulos, Diana A. Gorog, Carlo Di Mario, and Neville Kukreja.** Use of thrombectomy devices in primary percutaneous coronary intervention: a systematic review, meta-analysis. Int J Cardiol. 2013; 163(3): 229-241.
5. **Georgios Sianos, Michail I. Papafaklis, Joost Daemen, Sofia Vaina, Carlos A. van Mieghem, Ron T. van Domburg, et al.** Angiographic stent thrombosis after routine use of drug-eluting stents in S.T- segment elevation myocardial infarction: the importance of thrombus burden.Journal of the American College of Cardiology, 2007; 50: 573-83.
6. **Grüntzig A, Kuhlmann U, Vetter W, Lütolf U, Meier B, and Siegenthaler W.** Transluminal dilatation of coronary artery stenosis. Lancet 1978; 1:263.
7. **Grech ED.** ABC of interventional cardiology, Percutaneous coronary intervention. I: History, development. BMJ 2003; 326: 1080-1082.
8. **Elliott M. Antman, Daniel T. Anbe, Paul Wayne Armstrong, Eric R. Bates, Lee A. Green, Mary Hand, et al.** ACC/AHA guidelines for the management of cases with S.T-elevation myocardial infarction: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines. 2004; 110: e82-e292.
9. **Katia Orvin, Tamir Bental, Abid Assali, Eli Israel Lev, Hana Vaknin-Assa, and Ran Kornowski.** Usefulness of the CHA2DS2-VASC score to predict adverse outcomes in cases having percutaneous coronary intervention. Am J Cardiol 2016; 117: 1433-1438.
10. **Ibrahim Halil Tanboga, Selim Topcu, Enbiya Aksakal, Kamuran Kalkan, Serdar Sevimli, and Mahmut Acikel.** Determinants of angiographic thrombus burden in cases with S.T-segment elevation myocardial infarction. Clinical, Applied Thrombosis/Hemostasis. 2014; 20(7): 716-722.
11. **Sabri Seyis, Özge Kurmuş, Salih Kiliç, Sezen Bağlan Uzunget and Ebru Akgül Ercan.** CHA2DS2-V.A.S.C score predicts intracoronary thrombus burden in cases with S.T-elevation myocardial infarction. Biomedical Research 2017; 28(17): 8050-8054.
12. **Bartosz Hudzik, Janusz Szkodziński, Michal Hawranek, Andrzej Lekston, Lech Poloński, Mariusz Gąsior, et al.** CHA2DS2-V.A.S.C score had useful in predicting poor 12-month outcomes following myocardial infarction in diabetic cases without atrial fibrillation. Acta Diabetol 2016; 53: 807-815.
13. **Kyung Hwan Kim, Wan Kim, Sun Ho Hwang, Won Yu Kang, Sang Cheol Cho, Weon Kim, et al.** The CHA2DS2VASc score could be used to stratify the prognosis of A.M.I cases irrespective of presence of atrial fibrillation. J Cardiol 2015; 65: 121-127.
14. **Gokturk Ipek, Tolga Onuk, Mehmet B. Karatas, Baris Gungor, Altug Osken, Muhammed Keskin, et al.** CHA2DS2-V.A.S.C Score had a predictor of no-reflow in cases with S.T-segment elevation myocardial infarction who underwent primary percutaneous intervention. Angiol. 2016; 67: 840-845.
15. **Gul, Mehmet, Uyarel, Huseyin, Ergelen, Mehmet, et al.** The relationship between red blood cell distribution width, the clinical outcomes in non-ST elevation myocardial infarction, unstable angina pectoris: a 3-year follow-up. Coron Artery Dis 2012; 23(5): 330-336.
16. **Flintholm Raft K., Frestad D., Michelsen M.M., Suhrs H.E., Rask A.B., Nilsson M., et al.** Peripheral endothelial function, coronary

flow velocity reserve is not associated in cases with angina, no obstructive coronary artery disease: the iPOWER study. Journal of vascular research. 2017; 54: 309-19.

17. **Hasan Aydin Bas, Fatih Aksoy, Atilla Icli, Ercan Varol, Abdullah Dogan, Dogan Erdogan, et al.** The association of plasma oxidative status, inflammation with the development of atrial fibrillation in cases presenting with S.T. elevation myocardial infarction, Scandinavian journal of clinical, laboratory investigation. 2017; 77: 77-82.
18. **Seoyoung C. Kim, Nishant R. Shah, James R. Rogers, Courtney F. Bibbo, Marcelo F.Di Carli, and Daniel H. Solomon.** Assessment of coronary vascular function with cardiac PET in relation to serum uric acid', PloS one. 2018; 13: e0192788.
19. **Halit Acet, Faruk Ertaş, Mehmet Ata Akıl, Ferhat Özyurtlu, Abdülkadir Yıldız, Nihat Polat, et al.** Novel predictors of infarct-related artery patency for S.T-segment elevation myocardial infarction: Platelet-to-lymphocyte ratio, uric acid, neutrophil-to-lymphocyte ratio. Anatol J Cardiol. 2015; 15: 648-56

How to cite

Darwish, M., El Zayat, A., abdelaziz, M., Al Awadi, M., Ibrahim, I. Predictors Of High Thrombus Burden In Patients With Acute Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention.. Zagazig University Medical Journal, 2024; (317-327): -. doi: 10.21608/zumj.2021.67250.2169