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Predictive Value of Stress Hyperglycemia Ratio on Intracoronary Thrombus Burden in Patients with ST Segment Elevation Myocardial Infarction

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ABSTRACT

Background: Primary percutaneous coronary intervention (PCI) is the recommended treatment for ST-segment elevation myocardial infarction (STEMI). Substantial coronary thrombus during primary PCI is associated with a poor prognosis. While stress hyperglycemia is common in STEMI patients, its association with intracoronary thrombus extent is uncertain.

Patients and Methods: The study included 100 STEMI patients eligible for primary PCI within 12 hours of symptom onset. SHR was calculated by dividing admission glucose by the estimated average glucose derived from HbA1c. Intracoronary thrombus burden was angiographically assessed using the Thrombolysis in Myocardial Infarction (TIMI) thrombus grade scale. Patients with thrombus grades 1 to 3 were categorized as the small thrombus burden (STB) group, and those with grades 4 and 5 as the large thrombus burden (LTB) group.

Results: SHR was significantly higher in the LTB group compared to the STB group (p=0.016), regardless of diabetic status. SHR demonstrated significant predictive value for LTB when the cut-off value exceeded 1.37, with a sensitivity of 67.24% and a specificity of 54.76%. Both univariate and multivariate analyses showed that LTB was independently predicted by SHR and admission blood glucose levels. No relationship was found between SHR and post-primary PCI TIMI flow grade or in-hospital clinical outcome.

Conclusions: SHR and admission hyperglycemia were independent predictors for LTB incidence in STEMI patients.

Keywords: Stress hyperglycemia ratio, ST-segment elevation myocardial infarction, Intracoronary thrombus burden.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) arises from the erosion of atherosclerotic plaques, generating the coagulation cascade and platelet activation. This results in the development of intraluminal thrombus, resulting in coronary artery occlusion and subsequent myocardial necrosis [1].

STEMI is a medical emergency that can be fatal because of the potential for serious complications, including arrhythmias, mechanical complications, and both acute and long-term heart failure [1].

Early diagnosis and prompt reperfusion are critical components in the effective management of ST-segment elevation myocardial infarction (STEMI), as they are directly associated with improved patient outcomes, including reduced infarct size and mortality. Primary percutaneous coronary intervention (PCI) has developed as the preferred reperfusion strategy in STEMI, demonstrating superior outcomes compared to thrombolytic therapy. This advantage is especially pronounced in settings with skilled interventional cardiologists and well-established systems for rapid patient transport, where timely PCI can restore coronary blood flow more efficiently than thrombolysis. PCI not only facilitates rapid recanalization of occluded arteries but also reduces the risk of complications such as recurrent ischemia and hemorrhagic transformation, which are more common with thrombolytic treatments [2].

The presence of bulky coronary thrombi in STEMI patients has been consistently associated with increased cardiovascular morbidity and mortality, establishing it as a significant prognostic factor. Large thrombi are particularly detrimental, as they occlude major coronary arteries, leading to impaired myocardial perfusion. This occlusion can also cause distal embolization, where thrombotic material is carried downstream, obstructing smaller coronary vessels and exacerbating ischemic injury. The resulting disturbances in epicardial blood flow further compromise the viability of the myocardium, increasing the likelihood of adverse clinical outcomes such as heart failure, arrhythmias, and prolonged hospitalizations. Moreover, bulky thrombi are often linked to difficulties in achieving successful reperfusion during PCI, contributing to suboptimal angiographic results and increased procedural complexity [3-5]. Thus, the identification and management of large thrombus burdens are essential for optimizing treatment strategies and improving survival rates in STEMI patients.

Patients who have been diagnosed with ST-segment elevation myocardial infarction (STEMI) frequently experience a phenomenon known as stress hyperglycemia, which is characterized by elevated blood glucose levels in response to acute physiological stress. This condition is linked to a higher death rate and a higher incidence of in-hospital complications, regardless of the patient's pre-existing diabetic status. Consequently, the early recognition and effective management of stress hyperglycemia hold significant clinical implications for improving outcomes in STEMI management. Prompt therapeutic interventions aimed at normalizing blood glucose levels may reduce adverse

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events and enhance recovery trajectories in this vulnerable patient population [6].

It is important to note that admission blood glucose levels may not provide a comprehensive evaluation of the acute elevation in glucose experienced by patients who have chronic hyperglycemia. To more accurately evaluate the extent of stress hyperglycemia, the stress hyperglycemia ratio (SHR) is utilized. The SHR is calculated by dividing the patient's admission glucose level by the estimated average blood glucose level, as determined by hemoglobin A1c (HbA1c) measurements. This ratio serves as a more reliable indicator of acute glycemic derangement, allowing medical professionals to more accurately identify individuals who are at risk and adjust their care approaches accordingly [7,8].

Extensive research has elucidated the detrimental effects of both stress hyperglycemia and chronic hyperglycemic states on post-procedural outcomes following primary PCI. Studies have indicated that these glycemic conditions are correlated with impaired myocardial reflow, significantly increasing the risk of no-reflow phenomena even after seemingly successful revascularization efforts. The underlying mechanisms are thought to involve adverse effects on endothelial function and platelet activity, coupled with the induction of microvascular dysfunction. Collectively, these pathophysiological disturbances hinder efficient coronary reflow, thereby exacerbating ischemic injury and worsening clinical outcomes in patients undergoing PCI [9, 10]. Addressing these metabolic derangements is critical in optimizing revascularization strategies and improving overall patient prognosis in the context of STEMI management.

Aim of the study: To assess the association between the stress hyperglycemia ratio (SHR), calculated using acute and chronic glycemic levels, and the extent of intracoronary thrombus burden in STEMI patients.

PATIENTS AND METHODS

This prospective observational study was conducted on hundred patients presented to Menoufia University Hospital and Mahalla Cardiac Center with STEMI, aged > 18 years old and eligible for primary PCI twelve hours after chest pain onset.

Ethical approval:

The study was done after approval from the Ethical Committee of Menoufia University Hospitals, Menoufia, Egypt (approval code: 10/2022 – CARD 20). An informed written consent was obtained from the patients. The Helsinki Declaration was followed throughout the study's conduct.

Exclusion criteria: patients with severe hepatic or renal insufficiency, active infection, malignancy, autoimmune disease, blood dyscrasias, previous thrombolytic therapy and other forms of acute coronary syndromes (ACS) (NSTEMI and unstable angina) were excluded.

Every patient had a thorough history taking, a clinical examination, standard laboratory testing, and 12 leads surface electrocardiogram (ECG).

STEMI was diagnosed when a patient had typical anginal chest pain and a new ST-segment elevation measured at the J point, found in two contiguous leads, with ST-segment elevation ≥ 1.5 mm in women, ≥ 2.5 mm in men under 40, or ≥ 2 mm in men over 40 in leads V2–V3 and/or ≥ 1 mm in the other leads (without left ventricular (LV) hypertrophy or left bundle branch block (LBBB) pattern) [11].

For each patient, the stress hyperglycemia ratio (SHR) was computed by dividing the estimated average glucose level by acute hyperglycemia (admission blood glucose) [4,12]. A blood glucose level more than 198 mg/dl at admission was considered acute hyperglycemia [13]. The following formula was used to estimate the average glucose level: [(28.7×HbA1c %)-46.7] **[14]**.

Primary PCI techniques, decisions and the medical treatment of patients were administered in line with the European Society of Cardiology (ESC) guidelines [15-17].

The severity of thrombus burden in myocardial infarction (MI) [3] was assessed using the Thrombolysis in Myocardial Infarction (TIMI) thrombus grade scale. This scale categorizes thrombus burden into six grades:

- **Grade Zero:** No visible thrombus.
- **Grade One:** Possible thrombus, but difficult to confirm.
- **Grade Two:** Thrombus present, but less than half the diameter of the vessel.
- **Grade Three:** Thrombus occupies more than half but less than the entire diameter of the vessel.
- **Grade Four:** Thrombus occupies the entire diameter of the vessel.
- **Grade Five:** Total occlusion of the vessel by thrombus.

Following that, patients were split into 2 groups according to their thrombus burden:

1. **Large Thrombus Burden (LTB) group:** Included patients with thrombus grades four and five.
2. **Small Thrombus Burden (STB) group:** Included patients with thrombus grades one, two, three.

After culprit lesion revascularization, TIMI (Thrombolysis in MI) flow was graded; TIMI 0: Complete occlusion and no perfusion. TIMI I: Penetration of obstruction by contrast but no distal perfusion. TIMI II: Perfusion of entire artery but delayed flow. TIMI III: Complete perfusion and normal flow [18].

Patients were observed after primary PCI for 48 hours of their hospital stay to assess complications as

congestive heart failure, reinfarction, post infarction angina and death.

Statistical analysis

Data analysis was performed using SPSS version 26. Continuous variables were summarized as mean and standard deviation, and compared between groups using an unpaired t-test. Frequencies and percentages representing categorical variables were shown, and the chi-square test or Fisher's exact test, as applicable, was used for analysis. Kruskal-Wallis test used to compare two or more groups for a continuous or discrete variable. Statistical significance was defined as a p-value of less than 0.05.

Using receiver operating characteristic (ROC) curve analysis, the optimal cut-off value of the stress hyperglycemia ratio (SHR) for predicting a high thrombus load was determined. Sensitivity, specificity, and area under the curve (AUC) were calculated.

Univariate regression was used to estimate the relationship between a dependent variable and one independent variable. Multivariate regression was also used to estimate the relationship between a dependent variable and more independent variables.

RESULTS

A hundred patients with STEMI were enrolled in the study. Patients were classified into 2 groups based on the intracoronary thrombus burden grades. Small thrombus burden (STB) group included patients with thrombus burden grade 1 to grade 3 and consisted of 42 patients. Large thrombus burden (LTB) group included patients with thrombus burden grade 4 and grade 5 and consisted of 58 patients.

Demographic data, lipid profile, HbA1c and average glucose level were not substantially different among both groups. Admission blood glucose and SHR increased remarkedly in LTB than STB **(Table 1).**

Table 1: Comparison of the two groups under study according to laboratory parameters and demographic information

		STB (n=42)	LTB (n=58)	Test of Sig.	P
Demographic data					
Age (years)		60.02±10.74	60.03±9.92	t=0.004	0.996
Sex	**Male**	37(88.1%)	53(91.38%)	χ^2=0.291	0.589
	Female	5(11.9%)	5(8.62%)		
Hypertension		16(38.1%)	24(41.38%)	t=0.109	0.741
Diabetes mellitus		26(61.9%)	34(58.62%)	t=4.105	0.741
Smoking		11(26.19%)	17(29.31%)	t=0.117	0.732
Hyperlipidemia		42(100%)	57(98.28%)	t=0.731	1
Family history of CAD		8(19.05%)	12(20.6%)	t=0.041	0.839
Laboratory parameters					
Lipid profile	**Total cholesterol (mg/dl)**	182.79±8.37	179.97±38.76	U=1213	0.972
	LDL (mg/dl)	108.5±4.65	106.55±4.91	U=1266.0	0.798
	HDL (mg/dl)	42.02±8.24	43.12±8.1	U=1322	0.465
	Triglycerides (mg/dl)	153.74±7.31	148.62±7.12	U=1201.5	0.715
Admission blood glucose (mg/dl)		236.05 ± 35.18	271.71±66.98	U=1608.0	**0.002***
HbA1c (%)		7.92±1.05	7.8±1.21	U=1177.50	0.779
Average glucose level (mg/dl)		180.58±8.91	177.06±6.4	U=1177.500	0.779
Stress hyperglycemia ratio		1.43±0.25	1.68±0.26	U=1551.55	**0.020***

Data are presented as mean ± SD or frequency (%). *: Significant P, STB: Small thrombus burden, LTB: Large thrombus burden, CAD: Coronary artery disease, LDL: Low density lipoprotein, HDL: High density lipoprotein, HbA1c: Glycated hemoglobin, χ^2: Chi square test, t: student t test, U: Mann whiteny test.

SHR in patients with or without DM was significantly higher in LTB than STB **(Table 2).**

Table 2: Comparison of the two groups under study based on SHR in patients with diabetes mellitus and those without

	Total	STB	LTB	P
SHR in DM patients	(n=60) 0.24 ± 1.24	(n=26) 1.15±0.21	(n=34) 1.31±0.23	**0.008***
SHR in non-DM patients	(n=40) 2.08 ± 0.44	(n=16) 1.88±0.35	(n=24) 2.22±0.44	**0.002***

Data are presented as mean ± SD. *: Significant P, STB: Small thrombus burden, LTB: Large thrombus burden, SHR: Stress hyperglycemia ratio DM: Diabetes mellitus.

SHR can significantly predict LTB (P=0.016 and AUC=0.637) at cut-off > 1.37 with 67.24% sensitivity, 54.76% specificity, 67.2% positive predictive value and 54.8 % negative predictive value **(Figure 1).**

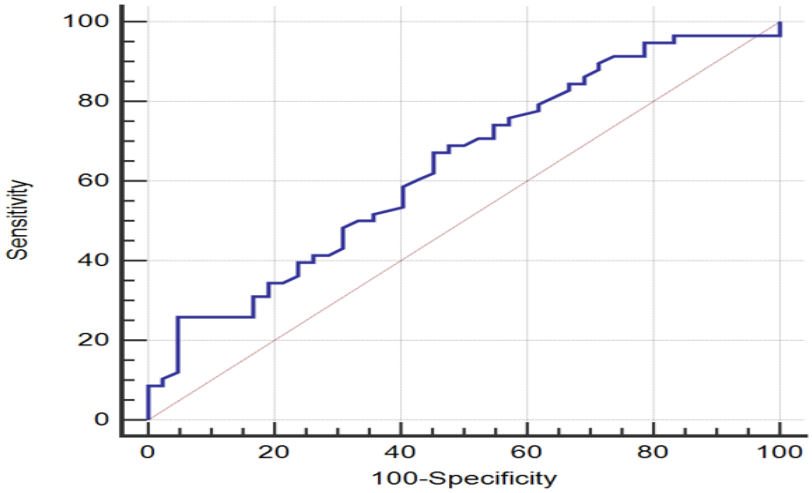

Figure 1: ROC curve for stress hyperglycemia ratio to predict LTB patients.

SHR had statistically insignificant relation with post procedural TIMI flow grades and in-hospital clinical complications as (heart failure, post infarction angina and death) **(Table 3).**

Table 3: Relation between SHR and both TIMI flow and clinical outcomes in total sample

		SHR	Test of Sig.	P
Post primary PCI TIMI flow				
TIMI I (n=4)		2±0.68	K=6.606	0.272
TIMI II (n=16)		1.5±0.39		
TIMI III (n=80)		1.6±0.54		
In-hospital clinical outcomes				
HF	**Yes(n=33)** **No(n=67)**	1.54±0.47 1.59±0.56	U=1099.500	0.965
Reinfarction	**Yes(n=0)** **No(n=100)**	- -	-	-
Angina	**Yes(n=24)** **No(n=76)**	1.59±0.54 1.57 ± 0.53	U=919.00	0.955
Death	**Yes(n=4)** **No(n=96)**	1.62±0.84 1.57 ± 0.52	U=176.00	0.793

Data are presented as mean ± SD. SHR: Stress hyperglycemia ratio, PCI: Percutaneous coronary intervention, TIMI: Thrombolysis in myocardial infarction, HF: Heart failure, K: Kruskal-Walllis test, U: Mann whiteny test.

The incidence of post procedural TIMI flow < III was significantly higher in LTB than STB (P<0.05). On the other side, the incidence of in-hospital clinical complications was insignificantly different between both groups **(Table 4).**

Table 4: Comparison of the two groups under study based on clinical outcomes and TIMI flow

		STB(n=42)	LTB(n=58)	Test of Sig.	P
Post primary PCI TIMI flow					
TIMI I (n=4)		0(0.0%)	4 (4%)	χ^2=7.943	**0.015***
TIMI II (n=16)		3 (3%)	13 (13%)		
TIMI III (n=80)		39 (39%)	41 (41%)		
In-hospital clinical outcomes					
HF	**Yes (n=33)** **No (n=67)**	11(26.2%) 31(73.8%)	22(37.9%) 36(62.1%)	χ^2=1.519	0.218
Reinfarction	**Yes(n=0)** **No(n=100)**	0(0.0%) 42(100.0%)	0(0.0%) 58(100.0%)	-	1
Angina	**Yes(n=24)** **No(n=76)**	10(23.8%) 32(76.2%)	14(24.2%) 44(75.8%)	χ^2=0.001	0.970
Death	**Yes(n=4)** **No(n=96)**	1(2.4%) 41(97.6%)	3(5.2%) 55(94.8%)	χ^2=0.494	0.637

Data are presented as frequency (%). *: Significant P. STB: Small thrombus burden, LTB: Large thrombus burden, PCI: Percutaneous coronary intervention, TIMI: Thrombolysis in myocardial infarction, HF: Heart failure, χ^2: Chi square test.

In univariate regression analysis, SHR and admission blood glucose were independent predictors of LTB. In multivariate regression analysis, SHR and admission blood glucose remained independent predictors of LTB **(Table 5).**

Table 5: Logistic regression analysis, both univariate and multivariate, for the variables influencing LTB patients

	Univariate		Multivariate	
	p	**OR (LL – UL 95% C.I)**	**P**	**OR (LL – UL 95% C.I)**
Age	**0.995**	1.0001(0.961– 1.039)		
Sex	**0.590**	0.6981(0.1886– 2.5842)		
Hypertension	**0.740**	1.1471(0.5088 – 2.586)		
Smoking	**0.731**	1.1685(0.479 – 2.846)		
Family history of CAD	**0.839**	1.1087(0.4085–3.009)		
Total cholesterol	**0.744**	0.998(0.989 – 1.007)		
LDL	**0.795**	0.998(0.988 – 1.009)		
HDL	**0.504**	1.07(0.967 – 1.068)		
Triglycerides	**0.711**	0.998(0.993 – 1.004)		
HbA1c	**0.776**	0.973(0.807– 1.173)		
Average glucose level	**0.776**	0.991(0.992 – 1.005)		
Admission blood glucose	**0.005***	1.014(1.004 – 1.024)	0.008*	1.013(1.003 – 1.022)
Stress hyperglycemia ratio	**0.020***	2.762 (1.172 – 6.509)	0.044*	2.446(1.023 – 5.848)

*: Significant P. OR: Odd`s ratio, CI: Confidence interval, LL: Lower limit, UL: Upper limit, CAD: Coronary artery disease, LDL: Low density lipoprotein, HDL: High density lipoprotein, HbA1c: Glycated hemoglobin.

DISCUSSION

The prevalence of stress hyperglycemia is notably high in patients with acute myocardial infarction (AMI), regardless of whether they have a preexisting diagnosis of diabetes mellitus. Increased mortality, greater infarct size, and prolonged hospital stays and other negative clinical consequences have been repeatedly linked to this phenomenon [19].

Stress hyperglycemia during AMI may result from the physiological response to the acute ischemic event, involving stress-induced hormonal fluctuations, such as elevated cortisol and catecholamines, which subsequently impair glucose metabolism. Studies have demonstrated that hyperglycemia in AMI patients, even in the absence of previous diabetes, is an independent predictor of worse prognosis. The pathophysiological mechanisms linking stress hyperglycemia to poor outcomes may involve both direct myocardial injury and secondary effects such as increased systemic inflammation, oxidative stress, and endothelial dysfunction. Therefore, managing glucose levels in AMI patients, regardless of diabetic status, is crucial to improving clinical outcomes [19].

Intracoronary thrombosis is one of the fundamental pathophysiological events in ST-segment elevation myocardial infarction (STEMI), where the rupture of an atherosclerotic plaque leads to the formation of a thrombus within the coronary artery. This thrombotic obstruction of the coronary artery severely impedes blood flow, contributing to myocardial ischemia and subsequent infarction. The presence of an intracoronary thrombus can significantly compromise both cardiac and epicardial perfusion, leading to detrimental consequences on myocardial viability. In addition, the mechanical interventions used during primary percutaneous coronary intervention (PCI), while designed to restore coronary patency, may be hindered by the thrombus. In particular, large thrombi are associated with suboptimal angiographic reperfusion, and they increase the risk of distal embolization, which can lead to further ischemic injury in microvascular territories. Distal embolization may exacerbate the myocardial damage by obstructing the small coronary vessels, worsening the prognosis of the patient. Therefore, understanding the burden and characteristics of intracoronary thrombus is essential in optimizing PCI strategies and improving patient outcomes in STEMI. [4].

In our study, STEMI patients were categorized into two distinct groups based on the burden of intracoronary thrombus. The small thrombus burden (STB) group consisted of 42 patients, while the large thrombus burden (LTB) group included 58 patients. Statistical analysis revealed no significant differences between the two groups regarding baseline characteristics, such as age, sex, presence of hypertension, diabetes mellitus, smoking history, hyperlipidemia, family history of coronary artery disease (CAD), or clinical markers including HbA1c and lipid profile parameters (total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides). This finding suggests that while intracoronary thrombus burden may be a determinant of clinical outcomes, other traditional cardiovascular risk factors may not differ significantly between patients with small and large thrombus burdens. Consequently, additional factors, possibly related to the thrombus's composition or other

microvascular and inflammatory processes, may play a critical role in determining the clinical prognosis in STEMI patients with varying levels of thrombus burden. Further investigations are needed to elucidate the precise mechanisms linking thrombus burden to outcomes in this population.

Similarly, when **Chu *et al.*** [20] looked into the relationship between SHR and intracoronary thrombus load in diabetic STEMI patients, they discovered that the STB and LTB groups did not significantly vary in the same characteristics. In our research, SHR and admission blood glucose were significantly higher in LTB than STB (P=0.016 and 0.002 respectively). As well, **Chu *et al***. [20] presented that SHR and admission blood glucose was related to increased thrombus burden in diabetic STEMI patients. In their study of the impact of stress hyperglycemia on thrombus load in non-diabetic STEMI patients, **Sigirci *et al.*** [21] found no differences between the two groups in terms of age, sex, the prevalence of hypertension, smoking, or dyslipidemia. However, the blood glucose levels of LTB patients were noticeably higher during admission**.** SHR remained significantly higher in LTB group in the patients with or without DM. SHR can significantly predict LTB patients (p value=0.016). ROC curve analysis showed that the ideal cut-off value of SHR as a predictor of thrombus burden was >1.37 with 67.24% sensitivity and 54.76% specificity. When **Algül *et al.*** [22] looked into the relationship between SHR and intracoronary thrombus load in ACS patients, they discovered that SHR was linked to higher thrombus burden regardless of ACS types or DM status. (P value< 0.001), with cut-off value >1.04 for SHR as a predictor of thrombus burden in ACS patients.

According to **Chu *et al.*** [20], SHR can predict LTB in STEMI diabetic patients (P value < 0.001), with a cut-off value >1.19. In univariate regression analysis, SHR and admission blood glucose were independent predictors of LTB patients. In multivariate regression analysis, SHR and admission blood glucose remained independent predictors of LTB patients. According to **Chu *et al.*** [20] univariate and multivariate logistic analyses, demonstrated that SHR was an independent predictor of LTB, which comes in the same line with our results. While admission blood glucose did not show significant difference in predicting LTB patients in either univariate or multivariate analysis, which comes in contrast to our results.

SHR had statistically insignificant relation with post primary PCI TIMI flow. In the same line, according to **Stalikas *et al***. [23], stress hyperglycemia had no effect on the incidence of poorer post-procedural TIMI flow or angiographically visible residual thrombus in STEMI patients. In contrast, our analysis found that LTB had a considerably greater rate of post-procedural TIMI flow< III than STB.

In our study, we meticulously analyzed the relationship between systolic hypertension response (SHR) and intracoronary thrombus burden with respect to the incidence of in-hospital clinical complications, including heart failure, post-infarction angina, and mortality, following primary percutaneous coronary intervention (PCI). The results indicated that there was a statistically insignificant correlation between these variables and the aforementioned clinical outcomes. This finding suggests that, within the parameters of our investigation, SHR and thrombus burden may not serve as reliable predictors of post-procedural complications in this patient cohort.

In contrast, **Lin *et al.*** [24] provided compelling evidence highlighting that hyperglycemia is significantly associated with adverse in-hospital outcomes, including congestive heart failure (HF) and mortality following emergency PCI. Their study revealed that patients exhibiting elevated glucose levels not only faced an increased risk of immediate complications but also exhibited significantly higher all-cause mortality rates at both six- and twelve-months post-procedure. Such findings underscore the critical importance of glycemic control in the acute management of patients undergoing PCI, emphasizing the potential role of metabolic parameters as key determinants of clinical outcomes.

Moreover, **Köktürk *et al.*** [25] investigated the implications of thrombus burden on clinical outcomes in individuals presenting with ST-segment elevation myocardial infarction (STEMI). Their results demonstrated that patients classified within the high thrombus burden (LTB) group experienced a higher incidence of both 30-day mortality and major adverse cardiovascular events (MACE) when compared to those with lower thrombus burden (STB). Notably, their long-term follow-up indicated that the LTB cohort also experienced an elevated risk of MACE over a ten-year horizon, reinforcing the notion that thrombus burden is a critical factor influencing both short-term and long-term clinical prognoses in STEMI patients.

The discrepancies observed between our study and those conducted by **Lin *et al.*** [24] **and Köktürk *et al.*** [25] regarding the impact of SHR and intracoronary thrombus burden on in-hospital clinical outcomes can be attributed to several methodological variations. These include differences in the operational definitions employed, sample sizes, statistical analysis techniques, and the duration of post-procedural follow-up. Such factors undoubtedly contribute to the divergent findings and highlight the complexity of assessing clinical outcomes in this patient population. Future investigations are warranted to elucidate these relationships further, ideally standardizing methodologies to improve comparability of results across studies.

CONCLUSION

Stress hyperglycemia (SHR) and admission hyperglycemia were identified as independent predictors of large thrombus burden (LTB) in patients with STEMI. A SHR value greater than 1.37 was found

to be the optimal cut-off for predicting thrombus burden in this cohort. However, our findings also indicate that SHR did not exhibit a significant correlation with post-primary PCI TIMI flow or in-hospital clinical outcomes. These results suggest that while SHR is a useful predictor for thrombus burden, its impact on immediate procedural success and short-term clinical prognosis may be limited. Given these insights, further research is warranted to explore the underlying mechanisms by which stress hyperglycemia influences both angiographic and clinical outcomes in STEMI patients, particularly in the context of thrombus burden and PCI strategies.

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