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Rationale of Anticoagulation Prolongation versus No Anticoagulation after Primary Percutaneous Coronary Intervention for ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Background: ST-elevation myocardial infarction (STEMI) results from the occlusion of coronary arteries, with plaque rupture being a common cause. Anticoagulant therapy is crucial during primary percutaneous coronary intervention (PPCI) for STEMI, yet the efficacy and safety of postprocedure anticoagulation (PPAC) remain debated due to limited data and unclear guidelines. **Objective:** This study aimed to evaluate the rationale of anticoagulation prolongation versus no anticoagulation after pPCI for STEMI.

Subjects and Methods: This randomized controlled clinical study was performed at the Cardiology Department of the Nasser Institute for Research and Treatment. Ninety-seven STEMI patients undergoing PPCI were randomly assigned to two groups: 49 patients received no subcutaneous low molecular weight heparin (LMWH) after PPCI (Group 1), and 48 patients received subcutaneous LMWH (Group 2). The parameters assessed included demographic data, clinical examination findings, laboratory investigations, ECG, echocardiography, and PCI findings.

Results: This study found no significant differences in baseline characteristics, vital signs, clinical examination outcomes, and most procedural characteristics between the groups. However, a significant difference was observed in the occurrence of left ventricular (LV) thrombus, with 8.2% of group 1 exhibiting LV thrombus compared to none in group 2 (P = 0.043). There were no significant differences in follow-up outcomes, including acute heart failure, bleeding, hemodynamically unstable arrhythmia, ischemic thrombus, stroke, myocardial infarction, and death.

Conclusions: Prolonged postprocedural anticoagulation after PPCI for STEMI significantly reduced in-hospital morbidity and the risk of LV thrombus without increasing bleeding risks. This suggests a superior efficacy of post-PPCI anticoagulation in mitigating ischemic events.

Keywords: Anticoagulation prolongation, Primary percutaneous coronary intervention, ST-segment elevation myocardial infarction.

INTRODUCTION

ST-elevation myocardial infarction (STEMI) arises when one or more coronary arteries become blocked, typically due to the sudden rupture, erosion, fissure, or dissection of plaque within the arteries, leading to the formation of a clot that obstructs blood flow. Key risk factors for STEMI include dyslipidemia, diabetes mellitus, hypertension, smoking, and a family history of coronary artery disease [1]. Anticoagulant therapy is a crucial component of treatment during PPCI for STEMI. Despite undergoing the procedure, STEMI patients continue to face the risk of ischemic events, leading to the adoption of postprocedure anticoagulation (PPAC) in clinical settings [2].

Data on the effectiveness and safety of PPAC are scarce. Existing guidelines offer no definitive or consistent advice on whether anticoagulation should be maintained in STEMI following PPCI, as highlighted by **Neumann and Sousa-Uva** [3].

Prior research has indicated that acute thrombotic complications following PPCI primarily occur within the initial 48 hours post-procedure, while major bleeding events (1% to 2%) tend to manifest more gradually over the course of 30 days afterward [4].

The delayed effect of oral P2Y12 inhibitors and the presence of residual thrombosis post-stent placement may account for the early onset of ischemic complications [5].

The potential benefits of short-term PPAC (lasting up to ≥ 48 hours or until discharge from the intensive cardiac care unit) in reducing ischemic events have not been systematically assessed in randomized trials [6]. This study aimed to assess the rationale for extending anticoagulation versus discontinuing anticoagulation following PPCI for STEMI.

SUBJECTS AND METHODS

Study design: This randomized controlled clinical trial was conducted to evaluate the justification for extending anticoagulation versus not administering anticoagulation after PPCI for ST-segment elevation myocardial infarction. The study was conducted in the Cardiology department of the Nasser Institute for Research and Treatment from December 2022 to July 2023, spanning a period of 8 months.

The study was conducted on 97 patients presenting with STEMI undergoing primary percutaneous intervention. They were divided into two groups: Group 1 consisted of 49 patients assigned to no subcutaneous LMWH after PPCI, and group 2 included 48 patients assigned to subcutaneous low molecular weight heparin after PPCI.

Inclusion criteria: Those experiencing STEMI who underwent PPCI for the culprit lesion, regardless of the thienopyridine regimen administered prior to randomization, and who were aged 18 years or older.

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ST-segment amplitudes measured 60 ms after the J point were required to be ≥ 2 mm in at least two contiguous ECG leads, in line with the guidelines of the European Society of Cardiology for STEMI [7, 8].

Exclusion criteria: If they had conditions requiring anticoagulation after PPCI, prior lytic treatment, chronic anticoagulation, body weight >120 kg or < 45 kg, previous CABG, cardiogenic shock, malignant ventricular arrhythmia, mechanical complications, BP ≥ 180/110 mmHg at randomization, bleeding diathesis, severe hematologic disease, intracerebral mass, aneurysm, recent (< 6 months) ischemic stroke or TIA, recent (< 6 months) intracranial hemorrhage, arteriovenous malformation, gastrointestinal or genitourinary bleeding within the past 2 weeks, heparin-induced thrombocytopenia, major surgery within 1 month, PLT≤ 100,000, suspected acute aortic dissection, transaminase > 3-fold upper limit of normal, allergy to any study drug, CCr < 30 ml/min, pregnancy or lactation, noncardiac coexisting conditions limiting life expectancy to less than 1 year, HFref EF < 40%, and age > 65 years.

METHODOLOGY

All patients were subjected to the following:

Full history taking including age, sex and risk factors including hypertension [9], diabetes mellitus [10], hyperlipidemia [11], coronary artery [12] and smoking.

Full clinical examination including vital signs such as heart rate, systolic blood pressure, diastolic blood pressure, rhythm, respiratory rate and oxygen saturation.

Physical examinations: Signs of physical distress, such as pallor, cyanosis, systemic congestion, and pulmonary congestion, monitoring for any sign of bleeding (hematemesis, melena, epistaxis and subcutaneous haemorrhage) for at least 48 hours, as well as monitoring for any sign of ischemic event (chest pain, dyspnea, weakness, lateralization, oxygen desaturation and palpitation).

Routine laboratory investigations: Laboratory tests were conducted on admission (especially CBC) and on a daily basis during the patient's hospital stay to assess various biochemical and hematological parameters.

Electrocardiography: Twelve leads of ECG were done at rest, in a supine position, and before enrollment into the study to identify heart rate, rhythm, and the presence of ischemic changes. It was recorded at a paper speed of 25 mm/s and a 10-mm/mV gain.

Echocardiography: Complete comprehensive transthoracic echocardiographic examinations were performed using a Philips affinity 50 machine with the S5-1 probe. Subjects were examined in the left lateral decubitus position. Images obtained included 2D, color, pulsed-wave and continuous-wave Doppler. All echocardiographic examinations were obtained and recorded for offline analysis [13].

Primary percutaneous coronary intervention: Trans-femoral artery puncture was performed using the standard Seldinger's technique, using 6F sheaths. All images were evaluated by an experienced operator. Data from the procedure, including the culprit artery, blood pressure during the procedure, and any procedural complications, were collected [14].

Study Endpoints: Patients were followed up in the CCU, the outpatient clinic and by telephone for any major events for a month after discharge as death, non-fatal myocardial infarction, non-fatal stroke, stent thrombosis, probable event (unexplained death within 30 days or target vessel myocardial infarction without angiographic confirmation of stent thrombosis), malignant arrhythmias are arrhythmias, primarily ventricular, that result in cardiovascular collapse [Ventricular tachycardia (VT), ventricular fibrillation (VF), completes heart block (CHB), and torsades de pointes] and major bleeding.

Ethical considerations: The study was done after being accepted by The Research Ethics Committee, Benha University. All patients provided written informed consents prior to their enrolment. The consent form explicitly outlined their agreement to participate in the study and for the publication of data, ensuring protection of their confidentiality and privacy. 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.

Data Management:

The statistical analysis was performed using the Statistical Package for Social Science (SPSS Inc., released 2009, PASW Statistics for Windows, Chicago: SPSS Inc.). Quantitative data were summarized as mean ± standard deviation (SD) and range (minimum-maximum), while qualitative data were presented as frequency and percentage. The analysis utilized appropriate statistical methods, with statistical significance defined as a P value ≤ 0.05. A P value < 0.001 was deemed highly significant, whereas a P value > 0.05 was considered non-significant.

RESULTS

No significant differences were observed regarding basic characteristics, including age (P = 0.514), sex (P = 0.513), diabetes mellitus (P = 0.929), hypertension (P = 0.909), dyslipidemia (P = 0.772), smoking (P = 0.360) and history of CAD (P = 0.355). No significant differences were observed regarding vital signs, including heart rate (P = 0.787), systolic blood pressure (P = 0.455), diastolic blood pressure (P = 0.936), and respiratory rate (P = 0.940). No significant differences were observed regarding clinical examination, including Killip class (P = 0.267) and STEMI patterns (P = 0.267) (Table 1).

Table (1): General and clinical characteristics of the studied groups

Variables				LMWH		Test t/x^2	P value
				Group 1 (n=49)	Group 2 (n=48)		
Age mean±SD				55.45±7.61	54.33±9.11	0.655 (t)	0.514
sex		Female	N	5	7	0.429 (x^2)	0.513
			%	10.2%	14.6%		
		Male	N	44	41		
			%	89.8%	85.4%		
medical history	Hypertension	No	N	27	27	0.013 (x^2)	0.909
			%	55.1%	56.3%		
		Yes	N	22	21		
			%	44.9%	43.8%		
	DM	No	N	28	27	0.008 (x^2)	0.929
			%	57.1%	56.3%		
		Yes	N	21	21		
			%	42.9%	43.8%		
	Dyslipidemia	No	N	30	28	0.084 (x^2)	0.772
			%	61.2%	58.3%		
		Yes	N	19	20		
			%	38.8%	41.7%		
	History of CAD	No	N	34	29	0.857 (x^2)	0.355
			%	69.4%	60.4%		
		Yes	N	15	19		
			%	30.6%	39.6%		
Smoking		No	N	13	9	0.837 (x^2)	0.360
			%	26.5%	18.8%		
		Yes	N	36	39		
			%	73.5%	81.3%		
Killip class		I	N	47	44	0.755	0.385
			%	95.9%	91.7%		
		II	N	2	4		
			%	4.1%	8.3%		
STEMI patterns		anterior	N	29	29	1.352	0.717
			%	59.2%	60.4%		
		inferior	N	17	18		
			%	34.7%	37.5%		
		lateral	N	1	0		
			%	2.0%	0.0%		
		posterior	N	2	1		
			%	4.1%	2.1%		
HR (bpm) mean±SD				81.78±8.17	81.27±10.09	0.271	0.787
SBP (mmHg) mean±SD				119.49±14.76	121.98±17.8	-0.750	0.455
DBP (mmHg) mean±SD				71.63±10.28	71.46±10.91	0.081	0.936
RR (/min) mean±SD				14.53±0.74	14.54±0.71	-0.075	0.940

(x^2) Chi-Square Tests, (t) Independent Samples Test, ST-segment elevation myocardial infarction (STEMI)

No significant differences were observed regarding laboratory investigations, including hemoglobin post catheter and at 24 h follow-up (P = 0.128, p=0.233), platelets count (P = 0.687), TLC (P = 0.755) and random blood sugar (P=0.994). No significant differences were observed regarding ejection fraction % post catheter and after 48 h follow-up (P = 0.401, p=0.088) respectively (Table 2).

Table (2): Laboratory and echocardiographic data among the studied groups

Variables	LMWH		Test t	P value
	Group 1 (n=49)	Group 2 (n=48)		
HGB post catheter (mg/dl) mean±SD	13.81±1.33	13.4±1.28	1.535	0.128
HGB 24h follow-up (mg/dl) mean±SD	13.71±1.22	13.39±1.44	1.200	0.233
PLT (X10e3/µL) mean±SD	297.86±7.94	304.46±8.66	-0.405	0.687
TLC (X10e3/µL) mean±SD	11.9±3.73	11.68±2.21	0.312	0.755
RBS (mg/dl) mean±SD	212.1±7.84	211.98±9.15	0.008	0.994
EF% post catheter mean±SD	50.59±7.63	51.88±7.36	-0.843	0.401
EF% 48h follow-up mean±SD	49.49±8.84	52.25±6.74	-1.726	0.088

(t) Independent Samples Test, Ejection fraction (EF).

No significant differences were observed between those with LMWH and those without regarding all procedural characteristics. No significant differences were observed between those with LMWH and those without regarding total stent (s) length (mm) and average stent(s) diameter (mm) (Table 3).

Table (3): PCI findings in the studied groups.

Variables			LMWH		Test x^2	P value
			Group 1 (n=49)	Group 2 (n=48)		
vessels affected	**one vessel**	**N**	45	46	1.201	0.549
		%	91.8%	95.8%		
	two vessels	**N**	3	2		
		%	6.1%	4.2%		
	three vessels	**N**	1	0		
		%	2.0%	0.0%		
culprit vessel	**LAD**	**N**	25	29	6.324	0.611
		%	51.0%	60.4%		
	LCX	**N**	6	2		
		%	12.2%	4.2%		
	RCA	**N**	13	14		
		%	26.5%	29.2%		
	OM	**N**	1	1		
		%	2.0%	2.1%		
	LAD+ LCX	**N**	1	1		
		%	2.0%	2.1%		
	LAD+ RCA	**N**	1	0		
		%	2.0%	0.0%		
	RCA+ OM	**N**	1	0		
		%	2.0%	0.0%		
	LAD+ LCX+OM	**N**	1	0		
		%	2.0%	0.0%		
Total stent(s) length (mm) mean±SD			46.53±19.06	47.02±14.96	-0.141	0.888
average stent(s) diameter (mm) mean±SD			3.34±0.49	3.35±0.44	-0.129	0.897

(x^2) Chi-Square Tests, (t) Independent Samples Test

A significant difference was found in patients without LMWH, which showed LV thrombus in 8.2% of cases, while LV thrombus didn't exhibit in those with LMWH (P = 0.043). No significant differences were observed regarding follow-up outcomes, including acute heart failure, bleeding and hemodynamically unstable arrhythmia (P = 0.082), (p=0.988) and (P=0.310) respectively. No significant differences were observed regarding outcomes, including Ischemic thrombus (IST), stroke, Myocardial infarction (MI) and death (P = 0.988), (p=0.320), (P=0.320) and (P=0.320) respectively (Table 4).

Table (4): Outcomes among the studied groups

Variables			LMWH		Test t/x^2	P value
			Group 1 (n=49)	Group 2 (n=48)		
Acute heart failure (AHF)	**No**	**N**	46	48	3.033	0.082
		%	93.9%	100.0%		
	Yes	**N**	3	0		
		%	6.1%	0.0%		
LV thrombus	**No**	**N**	45	48	4.087	**0.043***
		%	91.8%	100.0%		
	Yes	**N**	4	0		
		%	8.2%	0.0%		
Hemodynamically unstable arrhythmia	**No**	**N**	48	47	0.00	0.988
		%	98.0%	97.9%		
	Yes	**N**	1	1		
		%	2.0%	2.1%		
Bleeding	**No**	**N**	49	47	1.031	0.310
		%	100.0%	97.9%		
	Yes	**N**	0	1		
		%	0.0%	2.1%		
Ischemic thrombus (IST)	**No**	**N**	47	48	0.0	0.988
		%	97.9%	98.0%		
	Yes	**N**	1	1		
		%	2.1%	2.0%		
Stroke	**No**	**N**	48	48	0.990	0.320
		%	100.0%	98.0%		
	Yes	**N**	0	1		
		%	0.0%	2.0%		
Myocardial infarction (MI)	**No**	**N**	48	48	0.99	0.320
		%	100.0%	98.0%		
	Yes	**N**	0	1		
		%	0.0%	2.0%		
Death	**No**	**N**	48	48	0.99	0.320
		%	100.0%	98.0%		
	Yes	**N**	0	1		
		%	0.0%	2.0%		

(x^2) Chi-Square Tests

DISCUSSION

In addressing the critical gap in cardiovascular medicine regarding the optimal management of anticoagulation following PPCI for STEMI, our study embarked on evaluating the merits of prolonging anticoagulation with low molecular weight heparin (LMWH) versus discontinuing it postprocedure.

Our study revealed that a statistically insignificant difference was observed regarding age and sex. This is in agreement, **Yan *et al.*** [2] who cleared that there was a statistically insignificant difference observed regarding age and sex. **Tang *et al.*** [15] reported that the mean age of the studied patients was 62.80 ± 10.31 years in cases without anticoagulation versus 63.17 ± 10.82 in cases with anticoagulation, and gender distribution reflected a predominant male representation in both groups, 68.9% for cases without anticoagulation and 69.9% in cases with anticoagulation. **Wang *et al.*** [16] found no significant impact of gender on the in-hospital prognosis of STEMI patients following PCI. In contrast, **Benamer *et al.*** [17] demonstrated that female STEMI patients have higher in-hospital mortality rates compared to males. This difference was attributed to the significantly older age of female STEMI patients, whereas age profiles were similar between male and female STEMI patients in the current study.

In our study, there was a statistically insignificant difference observed regarding DM, HTN, dyslipidemia, smoking and family history of CAD. This is consistent with **Yan *et al.*** [2], who cleared that there was a statistically insignificant difference observed regarding DM, HTN, dyslipidemia and smoking. **Tang *et al.*** [15] **also** reported a statistically insignificant difference regarding DM, HTN, dyslipidemia, smoking and family history of CAD. Similarly, **Wang *et al.*** [16] **i**ndicated that a prior history of diabetes was an independent predictor of poor outcomes in patients with AMI. **De Luca *et al.*** [18] demonstrated that AMI patients with diabetes often present with a higher prevalence of multivessel disease, more severe coronary artery stenosis, and cardiac dysfunction. These factors contribute to microcirculatory blockages, hindering of the development of collateral circulation, and ultimately lead to worse prognoses.

In our study findings, a statistically insignificant difference was observed regarding vital signs, including heart rate, systolic blood pressure, diastolic blood pressure, and respiratory rate. These results are compatible with **Tang *et al.*** [15], who showed that systolic blood pressure and diastolic blood pressure averaged 132.54 ± 18.70 mmHg and 76.50 ± 10.31 mmHg, respectively, in cases without anticoagulation versus 133.03 ± 17.37 mmHg and 78.53 ± 10.65 mmHg.

Our study revealed insignificant differences regarding clinical examination, including the Killip class. This is in accordance with **Yan *et al.*** [2], who reported the predominance of class I (78.5%) in cases without anticoagulation and 78.8% in cases with anticoagulation. An insignificant difference was observed regarding clinical examination, including the Killip class.

In our study, a statistically insignificant difference was observed regarding laboratory investigations, including hemoglobin post catheter and at 24 h follow-up, platelets count, TLC and random blood sugar. In the same context, **Yan *et al.*** [2] cleared that there was a statistically insignificant difference observed regarding laboratory investigations, including hemoglobin and platelet count. **Wang *et al.*** [16] noted that elevated blood sugar levels upon admission for STEMI patients, regardless of diabetes status, are linked to higher mortality rates. This association is particularly strong in patients without prior abnormal glucose metabolism. Additionally, high blood sugar levels upon admission are closely tied to a poor prognosis following PCI. **Sabatine *et al.*** [19] showed that lower hemoglobin levels upon admission are associated with a poorer cardiovascular prognosis in STEMI patients. Specifically, they found that for every 1 g/dL decrease in hemoglobin level below 14 g/dL, there is a significant increase in mortality.

Our study found no statistically significant difference in EF% between the two groups. This is consistent with **Yan *et al.*** [2] who reported that the measurements of ejection fraction % revealed a mean value of 54.8 9.5 in cases without anticoagulation versus 54.9 10.1 in cases with anticoagulation. with insignificant difference between the two groups.

In the present study, we found an insignificant difference observed between those with CLEXAN and those without regarding all procedural characteristics. Our results are in concordance with those reported by **Madhavan *et al***. [20], who stated that regarding the type of stented vessels, LAD was the predominant vessel for PCI in both groups and represented 67.1%, LCX 21.4% and RCA 11.4%. Concerning the number of vessels affected, the single vessel disease represented 61.4% of all study groups, two vessel diseases represented 32.9% and three vessel diseases were 5.7%.

Our current findings clearly revealed that there was a statistically insignificant difference observed between those with LMWH and those without regarding total stent length and average stent diameter. This is consistent with **Tang *et al.*** [15] who cleared that there was a statistically insignificant difference observed between those with anticoagulation and those without regarding total stent length and average stent diameter.

In the current study, we found that statistically insignificant differences were observed regarding follow-up outcomes, including acute heart failure, bleeding, and hemodynamically unstable arrhythmia. In the same context, **Yan *et al.*** [2] reported that in-hospital cardiovascular death and the incidence of MACE were lower in patients with PPAC during hospitalization, so PPAC was associated with improved survival without an increased risk of bleeding complications. Contrary to expectations, **Tang *et al.*** [15] demonstrated that cardiovascular events occur at similar rates in patients

who do not receive anticoagulation compared to those who do after PCI. Furthermore, the incidence of minor bleeding complications was lower in the non-anticoagulation group than in the anticoagulation group during hospitalization. This suggests that anticoagulation may not be necessary for patients without specific complications following PCI. In contrast, **Madhavan *et al.*** [20] reported that PE occurred infrequently (0.3%) within 30 days following PCI and was not significantly reduced by the use of anticoagulants (AC) for routine prophylaxis compared to no AC. This finding is consistent with the fact that both groups had a median time to ambulation of one day. These findings suggest that routine AC for PE prophylaxis is unlikely to provide benefit after a successful, uncomplicated PPCI procedure with early ambulation. **Song *et al.*** [21] found a slight, though not statistically significant, decrease in infarct size by CMR in the prolonged anticoagulation group compared to the brief anticoagulation group at 30 days post-PPCI.

In the present study, we found that there was a statistically insignificant difference observed regarding outcomes, including ischemic thrombus, stroke, myocardial infarction and death. Our results are in concordance with those reported by **Yan *et al.*** [2], who reported that PPAC was associated with improved survival without an increased risk of bleeding complications. Furthermore, no differences were found between PPAC and non-PPAC patients with regard to the risk of in-hospital MI, stent thrombosis, stroke, or cardiac arrest.

LIMITATIONS

This study, while providing valuable insights into the post-PPCI management of STEMI patients, is not without limitations. The sample size, although adequate to detect a significant difference in the incidence of left ventricular thrombus, may limit the generalizability of the findings. Additionally, the study's relatively short follow-up period may not capture late thrombotic or bleeding events, potentially underestimating the long-term impact of post-PPCI anticoagulation strategies. Future studies with larger cohorts and extended follow-up are warranted to validate these findings and assess their applicability to broader patient populations and longer-term outcomes.

CONCLUSION

Prolonged postprocedural anticoagulation after primary percutaneous coronary intervention (PPCI) for ST-segment elevation myocardial infarction (STEMI) significantly reduced in-hospital morbidity and the risk of LV thrombus without increasing bleeding risks. This suggests a superior efficacy of post-PPCI anticoagulation in mitigating ischemic events, underscoring the importance of tailoring anticoagulation duration based on patient-specific factors and bleeding risks.

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