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Impact of the time factor on the development of in-hospital heart failure in patients presenting with anterior ST-segment elevation myocardial infarction undergoing primary PCI

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Abstract
Background: The incidence rate of newly diagnosed instances of heart failure subsequent to an acute ST-elevation myocardial infarction (STEMI) has a range of variability spanning from 10% to 45%. The aim of this study is to assess the association between the duration of delay in patients diagnosed with anterior STEMI who have primary percutaneous coronary intervention, and the occurrence of heart failure during their hospitalization.
Methods: This prospective study was carried out on 100 consecutive patients with clinical criteria of first anterior ST- elevation myocardial infarction (STEMI). Patients were subdivided into two groups: Group 1: treated by primary PCI without development of heart failure. Group 2: treated by primary PCI with development of heart failure.
Results: In univariate regression analysis, older age, HTN, DM, symptoms duration, number of diseased vessels, TIMI flow, serum creatinine and LV EF were correlated with AHF. In the multivariate regression analysis, using model adjusted for aforementioned parameters, LV EF and symptoms duration independently predicted AHF. According to the simulation results, the cut off value to discriminate AHF from Non-AHF was (>8 hours) with 100% sensitivity, 44.29% specificity, 43.5% positive predictive value and 100% negative predictive value.
Conclusions: AHF persists as a frequent complication in individuals with anterior STEMI who are treated with pPCI. Delay in primary angioplasty for patients with STEMI has significant prognostic implications, as shown by the present research. Total ischemia time must be reduced to increase survival in individuals with ST-elevation myocardial infarction (STEMI).

Keywords: Time, In-hospital heart failure, anterior STEMI, myocardial infarction, primary PCI

Introduction
Worldwide, ischemic heart disease is the leading cause of mortality. There are 1.8 million fatalities a year caused by it in Europe alone. The Centers for Disease Control and Prevention (CDC) report that it is the leading cause of death in Egypt, accounting for more than one fifth of annual fatalities (21%) [1].
During the occurrence of STEMI, the presence of an occlusive thrombus leads to ischemia and eventual necrosis in the specific area that is fed by the artery that is obstructed [2] which appears significantly in An anterior wall myocardial infarction arises from significant lesions in the left anterior descending artery, resulting in extensive myocardial necrosis and a pronounced impact on left ventricular systolic function. This kind of infarction is associated with a higher incidence of immediate and long-term mortality when compared to myocardial infarctions occurring in other places [3].
The prompt and efficient implementation of PCI, accompanied by the prompt restoration of blood flow in the coronary artery responsible for the STEMI, is widely regarded as the fundamental element of the therapeutic approach for AMI. This method has been shown to significantly reduce myocardial damage, whereas the risk of enduring myocardial dysfunction and cellular demise escalates with prolonged delays in intervention [3].

In spite of the previously mentioned criteria, new data obtained from the US National Cardiovascular Data Registry has shown that a mere 51% of patients diagnosed with STEMI who were transferred for first percutaneous coronary intervention (PCI) were able to accomplish the suggested target of a first door-to-balloon duration of less than 120 minutes [4].

Heart failure (HF) is recognized as a key prognostic factor for STEMI, and it may manifest during the acute or subacute phase of STEMI [5].

There is a wide range in the incidence of new-onset heart failure after acute STEMI, with reports from 10% to 45%. The introduction of primary percutaneous coronary intervention (pPCI) and secondary preventive medicines for coronary heart disease has improved the prognosis of HF after STEMI; nonetheless, the risk of all-cause death and re-hospitalization remains high [6, 7]. Furthermore, it has been observed that the majority of cases of HF occur in-hospital after myocardial infarction, and that this condition is a powerful independent predictor of in-hospital mortality in patients with STEMI receiving pPCI [8].

The aim of this research is to investigate the correlation between the incidence of heart failure during a hospital stay and the time delay experienced by patients with anterior STEMI after primary percutaneous coronary intervention.

Methods and Patients

This prospective research was done on 100 consecutive patients, both genders, with clinical criteria of first STEMI who had percutaneous coronary intervention (pPCI) within 12 hours of the start of symptoms, or who had pPCI because of clinical and/or ECG indications of increasing myocardial ischemia beyond 12 hours. The investigation was done out with permission from Tanta University Hospitals' Ethics Committee in Egypt. Every patient who was enrolled gave written permission after being fully informed. The time frame for this research was April 2022-April 2023.

Patients having a prior history of myocardial infarction, a prior coronary artery bypass graft (CABG), a history of proven left ventricular dysfunction, a history indicative of heart failure and congenital heart disease, or significant heart valve disease were excluded from consideration.

Patients were further categorized into two groups: Group 1: patients treated by primary PCI without development of heart failure. Group 2: patients treated by primary PCI with development of heart failure.

All patients were subjected to

- Complete history taking
- Complete clinical investigation

Vital signs: heart rate (HR), blood pressure (BP) and respiratory rate, adequacy of urine output over first few hours of admission, Oxygen saturation at room air via oximetry was assessed upon admission, with special attention to signs of heart failure.

The indicators of heart failure and hemodynamic instability, as outlined by the Killip classification, are as follows [9].

1. Killip class I encompass persons who do not exhibit any clinical manifestations of heart failure.
2. Killip class II encompasses patients who have rales or crackles in the lungs, an S3 heart sound, and an increased jugular venous pressure.
3. Killip class III is a classification used to designate persons who have evident acute pulmonary edema.

Killip class IV refers to persons who are experiencing cardiogenic shock or hypotension, which is characterized by a systolic blood pressure below 90mmHg, along with indications of peripheral vasoconstriction such as oliguria, cyanosis, or sweating.

1. **General examination:** With attention to patient look, decubitus, cyanosis, with special attention to signs of heart failure (e.g. orthopnoea and congested neck veins).

Local cardiac examination

Heart sounds, clinical chest examination and murmurs for evidence of congestion as the presence of wheezes or crackles.

- **Resting 12 leads ECG:** A 12-lead electrocardiogram (ECG) was acquired within a time frame of 10 minutes after the first medical contact, according to the standards set out by the European Society of Cardiology (ESC) in 2017. The ECG included the following leads: limb leads I, II, III, aVR, aVL, aVF, as well as chest leads V1 to V6. This procedure was performed for all patients upon their admission to the hospital [10].
- Laboratory testing to establish a baseline, including serum levels of creatinine and urea; cardiac enzyme levels, including troponin, CK-MB, and LDH; and hemoglobin concentration.
- The measurement of reperfusion is assessed using the TIMI blood flow grade. Successful reperfusion is defined as TIMI 3, whereas defective reperfusion is indicated by TIMI 0-1-2.

1. The term "TIMI 0 flow" is used to describe a condition in which there is a complete lack of forward blood flow past a blockage in the coronary artery, resulting in no perfusion of the affected area.
2. The TIMI 1 flow refers to a condition where there is limited antegrade coronary flow beyond the blockage, resulting in insufficient filling of the distal coronary bed. This condition is characterized by the presence of feeble coronary flow without adequate perfusion.
3. TIMI 2 flow refers to a state of partial reperfusion, characterized by delayed or slow antegrade flow, while ensuring full filling of the distal area.
4. The TIMI 3 flow refers to the presence of normal flow that adequately perfuses the distal coronary bed.
5. Echocardiography.

Statistical analysis

The statistical study was performed with SPSS v26, a software application produced by IBM Inc. in Chicago, IL, USA. The assessment of the normality of the data distribution was conducted by the use of the Shapiro-Wilks test and histograms. The statistical analysis included calculating the mean and standard deviation (SD) of the quantitative parametric variables. These values were then compared between the two groups using an unpaired Student's t-test. The research used quantitative non-parametric data, which were shown using the median and interquartile range (IQR). The data underwent analysed using the Mann Whitney-test. The qualitative variables were represented in terms of frequency and percentage (%) and

were analyzed using either the Chi-square test or Fisher's exact test, depending on appropriateness. The study population was subjected to both univariate and multivariate regression analysis to investigate possible variables linked to acute heart failure (AHF). A ROC curve analysis was performed in order to evaluate the discriminatory capacity of symptom duration in distinguishing between instances of acute heart failure (AHF) and non-AHF. A two-tailed p-value below 0.05 was considered significantly.

Results

100 patients with acute anterior STEMI (mean age 28 ± 80 years, 45% male) were carried on this study, including 70 (70%) in the non-HF group and 30 (30%) in the HF group.

In age, dyslipidemia, diabetes mellitus (DM) and hypertension (HTN) there were significantly difference between two groups (P value <0.05) but in sex, smoking, and family history of ischemic heart diseases there were no statistically significant difference between two groups. Table 1.

Table 1: Comparison between the two studied groups according to risk factors and demographic data

		Total (n = 100)	Non- AHF (n = 70)	AHF (n = 30)	p
Age (years)		54.91±11.86	50.83 ± 11.39	64.43 ± 6.16	<0.001*
Gender	Male	54 (54.0%)	34 (48.6%)	20 (66.7%)	0.096
	Female	46 (46.0%)	36 (51.4%)	10 (33.3%)	
Risk factors	HTN	50 (50.5%)	24 (34.3%)	26 (86.7%)	<0.001*
	DM	51 (51.0%)	31 (44.3%)	20 (66.7%)	0.040*
	Smoking	49 (49.0%)	31 (44.3%)	18 (60.0%)	0.150
	Dyslipidemia	78 (78.0%)	55 (78.6%)	23 (76.7%)	0.833
	Family history	59 (59.0%)	44 (62.9%)	15 (50.0%)	0.231

P: p value for comparing between the two studied groups.

Shortness of breath, symptoms duration, orthopnea and Killip class, pulse, urine output during first two hours of admission, crackles on auscultation, oxygen saturation room air via oximetry, number diseased vessel, final TIMI flow and LV EF were significantly differences between two groups. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) door to balloon (hours) and IV tirofiban were no significantly difference between two groups. Table 2.

Table 2: Comparison between the two studied groups according patients characteristics, and LV EF (%)

Symptoms		Total (n = 100)	Non- AHF (n = 70)	AHF (n = 30)	p
Shortness of breath	NYHA Class I	70 (70.0%)	70 (100.0%)	0 (0.0%)	^{MC}p <0.001*
	NYHA Class II	12 (12.0%)	0 (0.0%)	12 (40.0%)	
	NYHA Class III	7 (7.0%)	0 (0.0%)	7 (23.3%)	
	NYHA Class IV	11 (11.0%)	0 (0.0%)	11 (36.7%)	
Symptoms duration (hours)		16.51 ± 13.48	9.09 ± 4.64	33.83 ± 11.20	<0.001*
Orthopnea		22 (22.0%)	0 (0.0%)	22 (73.3%)	<0.001*
Killip class	I	70 (70.0%)	70 (100.0%)	0 (0.0%)	^{MC}p <0.001*
	II	12 (12.0%)	0 (0.0%)	12 (40.0%)	
	III	8 (8.0%)	0 (0.0%)	8 (26.7%)	
	IV	10 (10.0%)	0 (0.0%)	10 (33.3%)	
Hemodynamic parameters					
Systolic blood pressure (mmHg)		134.05 ± 25.42	139.50 ± 19.38	121.33 ± 32.77	0.007*
Diastolic blood pressure (mmHg)		84.55 ± 11.26	85.21 ± 10.23	83.0 ± 13.43	0.370
Pulse (bpm)		82.85 ± 12.86	75.86 ± 5.38	99.17 ± 10.09	<0.001*
Urine output during first two hours of admission	Adequate	82 (82.0%)	70 (100.0%)	12 (40.0%)	^{MC}p <0.001*
	Oliguric	8 (8.0%)	0 (0.0%)	8 (26.7%)	
	Anuric	10 (10.0%)	0 (0.0%)	10 (33.3%)	
Crackles on auscultation		30 (30.0%)	0 (0.0%)	30 (100.0%)	<0.001*
Oxygen saturation room air via oximetry (%)		93.58 ± 5.48	96.59 ± 1.65	86.57 ± 4.85	<0.001*
Angiographic and procedural characteristics					
Number diseased vessel	Single vessel	49 (49.0%)	42 (60.0%)	7 (23.3%)	0.001*
	Multi vessel	51 (51.0%)	28 (40.0%)	23 (76.7%)	
Door to balloon (hours)		1.34 ± 0.78	1.26 ± 0.68	1.52 ± 0.95	0.137
Final TIMI flow	<3	23 (23.0%)	7 (10.0%)	16 (53.3%)	<0.001*
	3	77 (77.0%)	63 (90.0%)	14 (46.7%)	
IV Tirofiban		53 (53.0%)	34 (48.6%)	19 (63.3%)	0.175
LV EF (%)		41.61 ± 7.38	45.19 ± 5.32	33.27 ± 3.98	<0.001*

MC: Monte Carlo.

There were no significant differences seen between the two groups in terms of hemoglobin, LDH, troponin, and CK-MB. However, a significant difference was found between the two groups in relation to creatinine, with a p-value of less than 0.001. There was a substantial difference seen in the in-hospital death rates. Table 3

Table 3: Comparison between the two studied groups according to Laboratory data, and in-hospital mortality rate

Laboratory data		Total (n = 100)		Non- AHF (n = 70)		AHF (n = 30)		p
		No.	%	No.	%	No.	%	
Hemoglobin (gm/dl)		12.28 ± 2.68		12.35 ± 2.75		12.12 ± 2.55		0.691
Creatinine (mg/dl)		1.21 ± 0.38		1.12 ± 0.40		1.40 ± 0.23		<0.001*
Troponin	Negative	10 (10.0%)		10 (14.3%)		0 (0.0%)		FEp= 0.030*
	Positive	90 (90.0%)		60 (85.7%)		30 (100.0%)		
CK-MB (U//L)		76.43 ± 39.83		54.76 ± 15.82		127.0 ± 32.29		<0.001*
LDH (mg/dl)		765.8 ± 596.9		700.4 ± 547.3		918.67 ± 684.6		0.166
Death		21(21.0%)		5 (7.1%)		16 (53.3%)		<0.001*

FE: Fisher Exact.

In univariate regression analysis, older age, HTN, DM, symptoms duration, number of diseased vessels, TIMI flow, serum creatinine and LV EF were correlated with AHF. In the multivariate regression analysis, using model adjusted for aforementioned parameters, LV EF and symptoms duration independently predicted AHF. Table 4.

Table 4: Multivariate and univariate Logistic regression analysis for the parameters affecting AHF

	Univariate		#Multivariate	
	p	OR (LL – UL 95%C.I)	p	OR (LL – UL 95%C.I)
Age (years)	<0.001*	1.171(1.092 – 1.256)	0.286	1.421(0.745 – 2.712)
HTN	<0.001*	12.458(3.895 – 39.845)	0.190	0.002(0.0 – 20.338)
DM	0.043*	2.516(1.029 – 6.150)	0.518	0.170(0.001 – 36.497)
Symptoms duration	0.001*	2.080(1.328 – 3.257)	0.022*	2.505(1.142 – 5.496)
Systolic blood pressure	0.002*	0.970(0.952 – 0.989)	0.900	1.011(0.848 – 1.207)
Number diseased vessel (Multi vessel)	0.001*	4.929(1.865 – 13.025)	0.998	0.363(0.003 – 38.385)
Final TIMI flow (3)	<0.001*	0.097(0.034 – 0.281)	0.791	3027.048(0 – 1.55642191)
LV EF (%)	0.006*	0.046(0.005 – 0.407)	0.014*	0.010(0.0 – 0.388)
Creatinine (mg/dl)	0.001*	7.390(2.154 – 25.354)	1.000	0.962(0.730 – 1.268)

OR: Odd`s ratio, C.I: Confidence interval, LL: Lower limit, UL: Upper Limit, #: All variables with $p<0.05$ was included in the multivariate.

ROC curve for symptoms duration (Time from the onset of symptoms to FMC) in hours to discriminate AHF

According to the simulation results, the cut off value to discriminate AHF from Non-AHF was (>8 hours) with 100% sensitivity, 44.29% specificity, 43.5% positive predictive value (PPV) and 100% PPV. Figure 1

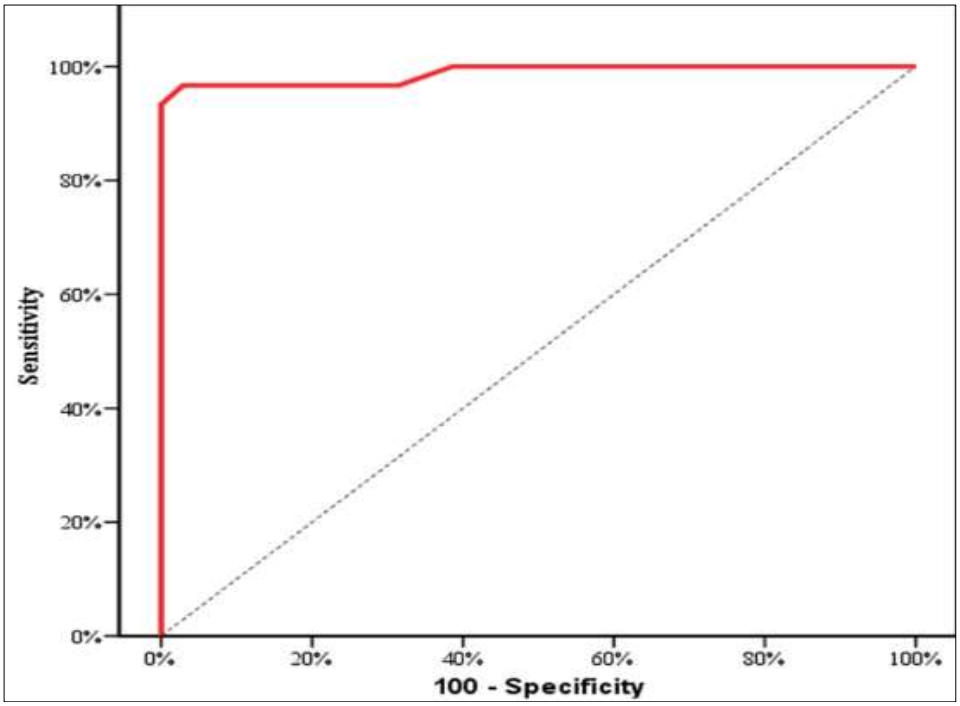

Fig 1: ROC curve for Symptoms duration (hours) to discriminate AHF (n = 30) from non AHF (n = 70)

Discussion

The expeditious reestablishment of blood circulation in the coronary artery impacted by an infarction by PCI is essential for optimizing the treatment of ST-STEMI. This methodology aims to decrease the size of the infarction, minimize damage to the myocardium, sustain ventricular function, and eventually decrease both mortality and morbidity rates [11].

In this research, there was significantly variance among the two groups, therefore symptoms to FMC time, should be the real goal for developing mortality in STEMI patients the

user did not provide any text. Numerous clinical experiments primarily concentrate on reducing the overall duration of ischemia and its consequential effects on the occurrence of shock and other severe cardiovascular events, similar to the research. Conducted by Khalid *et al.* [12]. There was an increase in mortality of 7.5% for every 30-minute delay in reperfusion in a research of 1,791 patients with STEMI who had primary angioplasty for STEMI. When compared to this research, the one by Koul *et al.* [13] Delay from FMC to PCI, measured in hours, was strongly linked with higher mortality in a total of 13790 patients.

In door to balloon there was no significantly variance among the two groups that is this came in contrast to Koul *et al.* [13], and Giuseppe DeLuca MD *et al.* [14] studies including 1,791 patients with STEMI treated with primary angioplasty to determine the impact of symptom start to balloon time and door-to-balloon time on mortality.

In killip class there was significantly difference between the two groups that is similar to the study conducted by Lourdes Vicent *et al.* that was performed in 991 patients underwent pPCI confirming that In-hospital mortality rate increased with high Killip class. In addition to the study conducted by Berger *et al.* [15] declaring The death rates in patients with acute myocardial infarction are higher if reperfusion by coronary angioplasty is delayed.

There was significantly variance among 2 groups in all hemodynamic parameters except SBP that is similar to the research conducted by Jingkang Liang *et al.* [16] and Berger *et al.* research [15]. In number diseased vessel: there was significantly variance among 2 groups that is similar to the research conducted by Liang *et al.* [16] containing data from 2823 out of 3204 patients demonstrate association among all components of time to treatment and in-hospital MACCEs among patients with STEMI who underwent pPCI. In contrast to Younes Nozari *et al.* study that show no significantly variance among 2 groups [17]. In final TIMI flow there was significantly variance among 2 group that is similar to the research conducted by Liang *et al.* [16] and De Luca MD, *et al.* [14], and In contrast to Younes Nozari *et al.* research that demonstrated no significantly variance among 2 groups [17].

In GP IIa/IIIb Inhibitor there was no significantly variance among 2 groups that is similar to the research conducted by Kurmi *et al.* (Kurmi *et al.*, 2022). All 113 consecutive STEMI patients undergoing primary PCI were prospectively included. According to TIT to improve survival in STEMI patients. In LV EF (%) there was significantly variance between the two groups that is similar to the research conducted by Liang *et al.* [16] demonstrating that, the high peak of NT-pro-BNP and low LVEF were independently associated with in-hospital HF in patients with anterior STEMI and De Luca MD *et al.* [14], while In contrast to Pradeep Kurmi, *et al.* [18] research that show significantly variance among 2 groups (P = 0.26). In in-hospital mortality there was significantly variance among 2 groups. that is similar to the research conducted by Koul *et al.* [13] that to examine the effects of FMC-to-PCI delay was significantly associated with increased mortality when exceeding one hour, Younes Nozari, *et al.* research [17] and De Luca MD *et al.* [14], in contrast to Kurmi, *et al.* [18] study that had no significantly p value =0.15. In laboratory data there was statistically significantly variance among both groups similar to the research conducted simi N Khullar *et al.* [19] demonstrate that mortality had significantly higher peak troponin levels. Peak troponin is also inversely proportional to LVEF and Ohlmann *et al.* [20] study that involve 87 patients with STEMI undergoing primary PCI At the acute phase of MI in patients undergoing pPCI, cTnI can be considered as a clinical marker for the estimation of infarct size predicting the extent of myocardial damage and necrosis & LDH There was significantly variance among both groups in contrast to De Luca MD *et al.* [14] study that was no significant in relation to symptoms-onset to balloon time & serum creatinine level which was statistically significant (p<0.001) similar to the research conducted by Liang *et al.*, [16].

In our research Univariate and multivariate regression analysis were performed to investigate the possible prameters of AHF in the study population. In univariate regression analysis, older age, HTN, DM, symptoms duration, number of diseased vessels, TIMI flow and LV EF were correlated with AHF. That was similar to the research conducted by Bruce R Brodie MD *et al.* [21], Acute EF <40%, number of diseased vessels and TIMI flow are a very strong univariate predictors of mortality.

In the multivariate regression analysis, using model adjusted for previous parameters, LV EF and symptoms duration independently predicted AHF That was similar to the research conducted by De Luca MD *et al.* [14] demonstrating that a symptom-onset-to-balloon time >4 h (p< 0.05) was an independent predictor of one-year mortality in addition to the research conducted by to Kurmi, *et al.* [18], the present research strongly support that TIT can be a good quality measurement together with other clinical determinants in order to improve survival in STEMI patients. Hence, attempts should be made to shorten TIT in order to enhance patients' survival.

Prognostic performance for Symptoms duration (hours) to FMC that defines the optimal cut-point value whose specificity and sensitivity are the closest to the value of the area under the curve. According to the simulation findings, the cut off value to discriminate AHF from Non-AHF was (>8 hours). That comes in agreement with the research conducted by De Luca MD *et al.* [14] that strongly support the prognostic implication of time delay in patients with STEMI undergoing primary angioplasty but with a variant cut off value of a symptom-onset-to-balloon time >4 h was identified as an independent predictor of one-year mortality.

Limitations

The study's limited sample size is a constraint, since it was conducted over a short time. Additionally, there is a possibility of selection bias arising from the prospective design. The research conducted did not include a comprehensive set of indicators, since several crucial variables, such as NPs tests, were not incorporated into our study. Consequently, the diagnosis of AHF mostly relied on clinical examination and TTE. The determination of time to reperfusion is prone to inaccuracies due to the challenges associated with assessing the exact beginning of symptoms in cases of acute myocardial infarction. The determination of the first medical contact was based on the timing of the first ECG. The operator evaluated the TIMI flow in the infarct artery after the operation, and the measures of left ventricular ejection fraction were conducted on-site instead of being analyzed by a core laboratory.

Conclusions

AHF persists as a frequent complication in individuals with anterior STEMI who are treated with pPCI. Delay in primary angioplasty for patients with STEMI has significant prognostic implications, as shown by the present research. Total ischemia time must be reduced to increase survival in individuals with ST-elevation myocardial infarction (STEMI).

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