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Relation between Admission Troponin Level and TIMI Flow, Myocardial Tissue Perfusion and Clinical Outcomes after Primary Percutaneous Coronary Intervention

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ABSTRACT
Background: Cardiac troponin is a structural protein within the cardiac myocytes and is a highly sensitive and specific marker to diagnose cardiac cells injury and damage.
Objective: This study aimed to detect the effect of troponin level assessed on admission on the angiographic and clinical outcome post primary PCI.
Patients and Methods: We conducted a cohort study to assess the prognostic relation between rise of first troponin on presentation and primary PCI success and post-primary PCI clinical outcome either during the hospital stay or during the follow up period after discharge. This study was conducted on forty-four patients presented to the Emergency Department with STEMI and underwent primary PCI.
Results: The TIMI flow grade post PCI was (I) in 5 patients (11.4%), (II) in 7 patients (15.9%) and (III) in 32 patients (72.7%). For the 32 patients who had TIMI III flow, we went for further analysis of reperfusion by assessing the myocardial blush by evaluating the TMPG. It was found that 3 patients (9.4%) had TMPG (I), 4 patients (12.5%) had TMPG (II) and 25 patients (78.1%) had TMPG (III). No patients were recorded to have major flow limiting dissection that ended with complications or coronary perforation. The in-hospital outcome was assessed and the total number of patients who had complications during hospitalization was 12 patients (27.3%).
Conclusions: This study findings support that a single measurement of hs-cTnT provides significant incremental information to risk stratification after STEMI.
Keywords: Cardiac troponin, STEMI, Percutaneous coronary intervention.

INTRODUCTION
Although the incidence of ST-segment elevation myocardial infarction (STEMI) has decreased over the past decade, it remains a common and morbid diagnosis [1].

Primary PCI is a form of reperfusion therapy for STEMI, which should be done as soon as possible. This is because heart muscle starts to be lost once a coronary artery is blocked and the sooner reperfusion therapy is delivered the better the outcome for the patient. The key points for dealing with STEMI are early diagnosis and immediate reperfusion to limit myocardial ischemia and infarct size and thereby reduce the risk of post-STEMI complications including heart failure [2]. Due to the development of prevention and treatment, the 6-month mortality after acute myocardial infarction (MI) has decreased considerably for patients with STEMI and Non-ST-segment elevation myocardial infarction over the past 20 years [3]. To ensure the best outcomes for adults with STEMI, the ambulance service and hospitals delivering primary PCI should work together to minimise delays in treatment.

In the present study, we aimed to detect the effect of troponin level assessed on admission on the angiographic and clinical outcome post primary PCI.

PATIENTS AND METHODS
This study cohort was study conducted on forty-four patients admitted to the Cardiology Departments, Zagazig University and the National Heart Institute in the period from July 2019 till June 2021. The study aimed to assess the prognostic relation between rise of first troponin on presentation and primary PCI success and post-primary PCI clinical outcome either during the hospital stay or during the follow up period after discharge. They were divided into two groups: Group 1 included patients of elevated hs-cTnT with levels equal to or more than 52 ng/l and this group has included 25 cases, 56.8% of the total patients' population. Group 2 that included patients with normal hs-cTnT with levels less than 52 ng/l and this group had included a slightly less percentage of patients, 19 patients, representing 43.2% of total patients' population. Patients were informed about purpose of the study and were told that their participation was voluntary.

Inclusion criteria: Patients with clinical presentation of STEMI who presented within 12 hours of symptoms onset and were candidates for primary coronary intervention as noted by elevation of total CK or CK-MB twice above normal or high serum troponin levels [4].

Exclusion criteria: Patients who present late more than 12 hours after onset of symptoms or if time of symptoms onset was unclear. Patients who had chronic renal failure on regular dialysis. History of recent PCI, within 30 days and expected to have early stent thrombosis. Patients who had previous surgical revascularization, (CABG), as the Duke Jeopardy Scoring system for assessment of CAD severity couldn't be applied to them. Patients who developed cardiac arrest on presentation. Patients who had Prinzmetal angina or had normal coronaries free of lesions and thrombi with angiography.

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All patients were subjected to the following:

Full history taking, careful clinical assessment and routine investigations including CBC, PT, INR, serum creatinine and baseline glucose level.

The Primary PCI procedures were performed according to standard practice, as patients were prepared with ASA 325 mg given orally with clopidogrel 600 mg [5]. Anticoagulation with unfractionated heparin (UFH) and enoxaparin or bivalirudin IV. UFH initial bolus was to be 70–100 U/kg when no glycoprotein (GP) IIb/IIIa inhibitor was planned or 50–60 U/kg when the use of GP IIb/IIIa inhibitors was expected and infusion dose was adjusted according to the activated partial thromboplastin time. Enoxaparin dose was 0.5 mg/kg intra venous followed by subcutaneous treatment [6]. PCI procedures were done through femoral approach. The use of peri-procedural GP IIb/IIIa inhibitor in case of angiographic evidence of massive thrombus, slow or no-reflow as an adjunct to primary PCI was considered [7]. Successful myocardial reperfusion was diagnosed in absence of microvascular obstruction (MVO). MVO after primary PCI is diagnosed when post-procedural angiographic TIMI flow was < 3, or in the case of a TIMI flow of 3 when myocardial blush grade was 0 or 1, or when ST segment resolution within 60–90 min of the procedure was < 70% [2].

Time assessment for each patient, the time of the maximum pain intensity, time of blood sample collection for troponin level assessment and time of wire crossing were determined. Angiographic data were collected and included.

Angiographic assessment of reperfusion by TIMI flow and myocardial blush were graded on the angiograms immediately after the primary coronary angioplasty procedure to assess the myocardial region of the infarct-related coronary artery. Two-dimensional echocardiography was used for assessment of the left ventricle (LV) function by measuring ejection fraction.

Ethical consent:

An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. 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.

Statistical analysis

The collected data were coded, processed and analyzed using the statistical package for social sciences version 22 for Windows® (IBM SPSS Inc., Chicago, IL, USA). Data were tested for normal distribution using Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test (χ^2) was used to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD (Standard deviation). Independent samples t-test was used to compare between two independent groups of normally distributed variables (parametric data). P value ≤ 0.05 was considered significant.

RESULTS

Distribution of patients according to their troponin on admission and peak troponin levels:

In the study, troponin level was considered as high when hs-cTnT rises above 52 ng/l and patients in the study were divided according to hs-cTnT levels into 2 groups: **Group I** included patients who had no significant rise of hs-cTnT levels and it included 19 patients (43.2%). **Group II** included patients who had significant rise of hs-cTnT levels and it included 25 patients (56.8%). The mean value of baseline troponin levels on admission of all patients was 1121 ± 4333 ng/l (Table 1).

Table (1): Distribution of patients according to their troponin on admission and peak troponin levels. (n=44)

hs-cTnT	**Total (n=44)**
Baseline hs-cTnT levels on admission	
Range	3-27694
Mean ± SD (ng/l)	1121.09 ± 223.54
Median (IQR)	54.5 (244.7)
hs-cTnT (Low level <52 ng/L)	*19 (43.2%)*
hs-cTnT (High level >=52 ng/L)	*25 (56.8%)*
hs-cTnT peak levels	
Range	256-27694
Mean ± SD	6244.16±1325.76
Median (IQR)	3475 (5702)

Comparison between both groups according to their baseline characteristics and for prevalence of risk factors of CAD:

Regarding patients' baseline characteristics and type of MI, in the low troponin level group, there were 1 female patient (5.3%) and 18 male patients (94.7%) with mean age of 48.37 ± 7.02 years. 8 patients had anterior type MI (42.1%), 10 patients had inferior type MI (52.6%) and 1 patient had lateral type MI (5.3%). On the other side, in the troponin high level group, there were 2 female patients (8.0%) and 23 male patients (92.0%) with mean age of 49.84 ± 9.93 years. 15 patients had anterior type MI (60.0%), 9 patients had inferior type MI (36.0%) and 1 patient had lateral type MI (4.0%). This comparison as shown in table (2) showed no statistically significant difference between both groups according to their baseline characteristics regarding gender, age and the type of MI. Regarding the presence of cardiac risk factors, in the low troponin level group, 8 patients were hypertensive (42.1%), 8 patients were diabetic (42.1%), 8 patients had hyperlipidemia (42.1%), 10 patients (52.6%) were smokers, 5 patients had family history of CAD (26.3%), no patients had history of CAD and only 1 patient had history of PVD (5.3%). While, in the high troponin level group, 14 patients were hypertensive (56.0%), 16 patients were diabetic (64.0%), 15 patients

had hyperlipidemia (60.0%), 13 patients were smokers (52.0%), 5 patients had family history of CAD (20.0%), 6 had history of CAD (24.0%) and only 1 patient had history of PVD (4.0%). The difference in presence of CAD history between both groups was statistically significant but the other risk factors didn't show statically significant differences between both groups.

Regarding the regular use of medications related to CAD or CAD risk factors, table (2) showed that 4 patients in the low troponin level group were on medications (21.1%) but it was more in troponin high level group as 14 patients were using medication (56.0%). This difference was statistically significant.

Table (2): Comparing both groups according to baseline characteristics and for prevalence of risk factors of CAD

Baseline characteristics	Low level group [hs-cTnT <52 ng/L] *(n=19)*	High level group [hs-cTnT ≥52 ng/L] *(n=25)*	Test	p-value
Gender				
Female	1 (5.3%)	2 (8.0%)	x^2=0.127	0.721 NS
Male	18 (94.7%)	23 (92.0%)		
Age (years):				
Mean ± SD	48.37 ± 7.02	49.84 ± 9.93	*t*=0.302	0.586 NS
Range	34-59	32-67		
Type of MI:				
Anterior	8 (42.1%)	15 (60.0%)	x^2=2.868	0.153 NS
Inferior	10 (52.6%)	9 (36.0%)		
Lateral	1 (5.3%)	1 (4.0%)		
Risk factors				
HTN	8 (42.1%)	14 (56.0%)	0.834	0.361 NS
DM	8 (42.1%)	16 (64.0%)	2.087	0.149 NS
Hyperlipidemia	8 (42.1%)	15 (60.0%)	1.386	0.239 NS
Smoking	10 (52.6%)	13 (52.0%)	x2=0.002	0.967 NS
FH	5 (26.3%)	5 (20.0%)	0.245	0.620 NS
CAD	0 (0.0%)	6 (24.0%)	4.735	**0.029 S**
PVD	1 (5.3%)	1 (4.0%)	0.040	0.842 NS
Medications use	4 (21.1%)	14 (56.0%)	4.946	**0.038 S**

Using: Chi-square test; Independent Sample t-test p-value >0.05 NS

Comparison between both troponin groups according to their clinical conditions on admission:

Table (3) showed the differences between both troponin groups related to clinical condition of the patients on admission to the hospital. In the low troponin level group, only 2 cases showed signs of HF (10.5%) and only 1 patient was hypotensive (5.3%). But, in the high troponin level group, 7 patients had HF symptoms (28.0%) and 7 patients were hypotensive (28.0%). There were statistically significant differences between both groups regarding HF and Low BP.

Table (3): Comparing both troponin groups according to their clinical conditions on admission

Clinical condition	Low level group [hs-cTnT <52 ng/L] *(n=19)*	High level group [hs-cTnT ≥52 ng/L] *(n=25)*	x^2	p-value
HF	2 (10.5%)	7 (28.0%)	4.178	**0.041 S**
Low BP	1 (5.3%)	7 (28.0%)	4.054	**0.044 S**

Comparison between both troponin groups according to time to presentation and time to PCI:

Comparison between both troponin groups according to the duration from the time of the typical symptoms of MI to the time of presentation to ER and another comparison included the time from presentation to ER to time of wire crossing during coronary intervention were done. Table (4) showed that the mean time to presentation was 1.97 ± 0.79 hours for the normal troponin group versus 2.61 ± 1.41 hours for the high level troponin group and time to wire during primary was 1.51 ± 0.68 hour for the normal troponin group versus 1.66 ± 1.15 hours for the troponin high level group with no statistically significant differences between both troponin groups.

Table (4): Comparing both groups according to time to presentation and time to PCI (hrs.)

Duration (hrs)	Low level group [hs-cTnT < 52 ng/l] *(n=19)*	High level group [hs-cTnT ≥ 52 ng/l] *(n=25)*	t-test	p-value
Time to presentation (hrs):				
Mean ± SD	1.97 ± 0.79	2.61 ± 0.51	1.785	0.082 NS
Range	0.16-3	0.8-6.4		
Time to catheterization (hrs):				
Mean ± SD	1.51 ± 0.68	1.66 ± 0.35	0.756	0.454 NS
Range	0.90-4.3	0.90-4.08		

Using: Independent Sample t-test; p-value >0.05 NS

Comparison between both troponin groups according to Duke Jeopardy score, culprit lesion location and TIMI flow before intervention:

Concerning the angiographic findings before intervention and regarding the Duke Jeopardy score, culprit lesion, and coronary TIMI flow before intervention, table (5) showed no significant difference regarding Duke Jeopardy score and TIMI flow. But, there was a statistically significant difference between both groups regarding the Culprit lesion as for 6 patients of the normal troponin group the culprit vessel was LAD (31.6%), while in the high level troponin group the culprit vessel for 18 patients was LAD (72.0%). IIb/IIIa inhibitor medication was used in 4 patients of the group of low level of troponin and in 2 patients of the group of elevated troponin with no statistical significance.

Table (5): Comparing both groups for Duke Jeopardy score, culprit lesion location and TIMI flow before intervention

	Low level group [hs-cTnT <52 ng/L] *(n=19)*	High level group [hs-cTnT ≥52 ng/L] *(n=25)*	Test	p-value
Duke Jeopardy score				
Mean ± SD	4.21 ± 1.07	4.56 ± 1.12	*t*=0.176	0.677 NS
Range	2-10	0-10		
Culprit lesion				
Diagonal	0 (0.0%)	1 (4.0%)	x^2= 9.835	**0.043 S**
LAD	6 (31.6%)	18 (72.0%)		
LCX	1 (5.3%)	0 (0.0%)		
OM	1 (5.3%)	0 (0.0%)		
RCA	11 (57.9%)	6 (24.0%)		
TIMI flow before intervention				
0 (Total occlusion)	16 (84.2%)	18 (72.0%)	x^2= 3.362	0.339
>0 (Partial flow)	3 (15.9%)	7 (28.0%)		
IIb/IIIa inhibitor	4 (12.5%)	2 (16.7%)	x^2= 0.129	0.720 NS

Using: Chi-square test; Independent Sample t-test; p-value >0.05 NS; p-value <0.05 S.

Comparison between both troponin groups according to in-hospital outcome:

In the low troponin level group, a total of 3 patients had complications, the 3 patients developed H.F and cardiogenic shock symptoms (15.8% for all).

No patients had major bleeding (0.0%), 1 patient needed in-hospital redo of coronary angiography (5.3%), 0 patients had re-infarction (0.0%), no patients died during this hospitalization (0.0%) and 4 patients had EF less than 40% (21.1%).

While, in the high troponin level group, 9 patients had complications (36.0%), 6 patients developed H.F (24.0%), no patients had major bleeding (0.0%), 1 patients developed cardiogenic shock (4.0%), 1 patient (4.0%) needed in-hospital redo of coronary angiography but didn't need further revascularization, 1 patient (4%) had re-infarction and died during this hospitalization and 11 patients had EF less than 40% (44.0%).

There was statistically significant differences between the low level group and the high level group concerning the development of in-hospital complications and regression of the LV function to EF < 40% (Table 6).

Table (6): Comparing both troponin groups for in-hospital outcome

In-hospital outcome	Low level group [hs-cTnT <52 ng/L] *(n=19)*	High level group [hs-cTnT ≥52 ng/L] *(n=25)*	x^2	p-value
In-hospital complications	3 (15.8%)	9 (36.0%)	3.962	**0.048 S**
Heart failure	3 (15.8%)	6 (24.0%)	0.447	0.504 NS
Bleeding	0	0		
C shock	3 (15.8%)	1 (4.0%)	1.816	0.178 NS
Redo angiography	1 (5.3%)	1 (4.0%)	0.040	0.842 NS
Repeat Revascularization of the IRA	0	0		
Re-infarction	0 (0.0%)	1 (4.0%)	0.778	0.378 NS
Death in hospital	0 (0.0%)	1 (4.0%)	0.778	0.378 NS
EF < 40%	4 (21.1%)	11 (44.0%)	4.027	**0.045 S**
Mean ± SD of EF	45.53 ± 11.29	38.60 ± 8.32	*t*=2.769	**0.026 S**
Range of EF	20-65	10-60		

Using: Chi-square test; Independent Sample t-test; p-value >0.05 NS; p-value <0.05 S

DISCUSSION

Level of hs-cTnT on admission and baseline characteristics and CAD risk factors:

In the present study, there were no significant differences in the baseline characteristics between the patients of both troponin groups as both groups were age- and sex-matched with homogenous distribution of risk factors of CAD that included smoking, hypertension, DM, hyperlipidemia, history of peripheral vascular disease, personal history of CAD and family history of CAD with no statistical differences. Also, there was no significant difference between both groups regarding the type of MI. As for the patients' **mean age,** in the group of normal troponin it was less than the mean age in the group of elevated troponin, 48 ± 7 years versus 49 ± 9 years, but with no statistical significance. This is concordant with **Matetzky and his colleagues [8]** study, which included 110 acute STEMI patients managed by primary angioplasty.

Fifty-four patients (49%) in this study had elevated troponin on presentation and 51% had normal levels. Their study showed no significant difference between both groups regarding the mean patients' age as the mean age was 67 ± 14 in the group of elevated admission troponin versus 68 ± 15 years in the group of normal admission troponin (P = 0.66).

Regarding **smoking,** in our study, active smokers were 10 patients (52.6%) in the group of normal admission troponin and 13 patients (52.0%) in the high troponin level group with no statistically significant difference (P = 0.967). This finding is in agreement with the results of the study conducted by **Matetzky and his colleagues** [8] who found that the active smokers were 13 smokers (23%) in the group of normal troponin vs 11 smokers (21%) in the elevated troponin group with no statistically significant difference (P = 0.72).

For **hypertension**, in the present study, 8 patients (42.1%) were hypertensive in the low troponin level group versus 14 hypertensive patients (56.0%) in the group of high admission troponin level but this was statistically non-significant (P = 0.361). This goes with **Matetzky and his colleagues** [8] where the incidence of rise of troponin on admission in the hypertensive and normotensive patients had no statistically significant difference (52% versus 59%; P = 0.71).

Regarding **DM**, 16 patients were diabetic representing 64.0% of the high-troponin level group and a less number of diabetic patients, 8 patients (42.1%), were in the normal troponin group with no statistically significant difference (P = 0.149). This also goes with **Matetzky and his colleagues** [8] who showed a non-statistically significant increase in the number of diabetic patients in the group of high admission troponin than the normal troponin group (P = 0.15).

Concerning **Hyperlipidemia,** our study showed that 15 patients (60%) in the high-troponin level group had hyperlipidemia versus 8 patients (42.1%) in the normal troponin group but this difference was statistically non-significant (p = 0.239). This is in concordance with the findings of **Matetzky and his colleagues** [8] study as they found a statistically non-significant difference between both troponin groups in number of patients who had hyperlipidemia (39% in the elevated troponin group versus 54% in the normal troponin group; P = 0.13).

Regarding the history of **PVD** in our study, it was positive for 1 patient (5.3%) in the group of normal admission troponin and also for 1 patient (4%) in the group of high-troponin with no statistically significant difference (P = 0.842).

As regards **STEMI**, the differences in distribution between both troponin groups had no statistically significant value. In the high-troponin level group, anterior type STEMI was the most common as 15 patients (60.0%) had anterior STEMI, then 9 patients (36.0%) had inferior STEMI and 1 patient (4.0%) had lateral STEMI. However, in the normal troponin group at admission, inferior STEMI was the most common, 10 patients (52.6%) then anterior STEMI, 8 patients representing 42.1%, and lastly lateral type STEMI, 1 patient (5.3%) (P value = 0.153). In **Matetzky and his colleagues** [8] study, almost half of patients in both groups had anterior STEMI with no statistically significant difference between them in STEMI type distribution (56% of the elevated troponin group versus 46% of the normal troponin group, P = 0.34).

In the present study, **regular and compliant use of medications** for cardiac related risk factors (DM, HTN, dyslipidemia, or PVD), or CAD was statistically more significant in the group of elevated troponin at admission than the group of normal troponin at admission, 14 patients (56.0%) versus only 4 patients (21.1%, P = 0.038). Every one of the 25 patients in the group of elevated troponin had at least one CAD risk factor and 14 of them (56%) were using risk factors controlling medications with compliance. On the other side, 16 patients in the group of normal troponin had at least one risk factor and out of them, 4 patients were using medications with compliance (25%). Also, the presence of **history of CAD** was more common in the group of elevated admission troponin, 6 patients (24.0%) with significant difference compared to normal troponin group, which had no patients with history of CAD. But, this was against **Matetzky and his colleagues** [8] study as it showed no significant difference between both admission troponin groups regarding the number of patients who had history of previous infarction, 11 patients (20%) in the elevated troponin group vs 12 patients (21%) in the normal troponin group (P = 0.89).

A recent study in 2019 done by **Brett and his colleagues** [9] on 14061 patients aiming to examine the association between admission troponin levels and in-hospital mortality for patients who had primary PCI for STEMI. They found that 47.2% of them had the initial troponin levels undetectable or within the reference range**.** Also in this trial, no significant differences were detected between troponin groups regarding baseline characteristics (age, gender and race) or risk factors (smoking, HTN, DM, Hyperlipidemia, CAD, PVD and HF).

Time to presentation and time to primary PCI relation to levels of hs-cTnT:

Where the troponin peak levels are driven by the success of revascularization and the territory of myocardium affected, troponin release kinetics indicate that the levels observed in the early acute phase are highly dependent on the time of ischemia [10]**.** In our study we checked for every patient **the time to presentation,** which represent the time interval between the onset of STEMI symptoms, as described by patients, till the time of evaluation in the ER. We found that, the determination of ischemia time based on history can be problematic as for groups more likely to present with atypical symptoms such as diabetic patients. The symptoms onset didn't correlate with the rise of troponin on admission as the relation between the symptoms to presentation time and the rise of troponin on presentation wasn't statistically significant. The mean time to presentation was non significantly longer for the high troponin level group, 2.61 ± 1.41, vs 1.97 ± 0.79 hours for the normal troponin group (P = 0.082). Also in a further analysis, the onset of symptoms and time to presentation did not provide incremental prognostic information over admission troponin levels as no

correlation was found between them and the rate of in-hospital complications or the rate of complications during the 4 months post-discharge follow up.

We found the mean time to presentation in the group of patients who didn't have in-hospital complications was 2.40 ± 1.33 vs 2.17 ± 0.87 hours for the group that had in-hospital complications with no significant difference (P = 0.357). The mean time to presentation in the group of patients who had no complications during the post-discharge follow up was 2.43 ± 1.27 versus 2.03 ± 0.99 hours for the group that had complications during the follow up with no significant difference (P = 0.342). These findings may be due to the relatively small patient population that was enrolled in our study and/or to the early attendance to the ER, as the mean value of time to presentation as shown above was between 2-3 hours for both troponin groups. However, **Cannon *et al.*** [11] also found the mortality when primary PCI is done was related to door-to-balloon time but not to symptom onset-to-balloon time due to wrong estimation of time of symptoms onset and peak by many patients**.**

Our study findings suggested that troponin levels on presentation may provide an important objective insight into the degree and duration of pre-hospital ischemia than the history of symptoms' onset and duration given by the patients, and that the increased ischemic time, when determined by troponin elevation, may be a significant driver of worse outcome for those in high troponin groups. These findings are in agreement with **Matetzky and his colleagues** [8] as they reported that the time to presentation did not provide more prognostic information over admission troponin levels. There was also no correlation between the duration of the pain and the cTnI levels on admission as in this trial although the mean time to presentation was longer for the group of high admission troponin than the group of normal troponin, 212 ± 186 vs 179 ± 171 minutes with no significant difference (P = 0.38).

Regarding delay of the primary PCI and reperfusion, it is known to be a reason of worse short- and long-term outcomes after STEMI [12]**.** In our study, we compared both troponin groups regarding the **Time to catheterization,** which represented the interval between the time of presentation and the time of coronary wire crossing the Culprit lesion. We found this time factor, which affects the outcome after primary PCI wasn't statistically significantly different between both groups. The median value for the normal troponin group was 1.51 ± 0.68 hours and for the elevated troponin groups was 1.66 ± 1.15 hours (P = 0.454).

Clinical condition on admission and level of hs-cTnT:

Acute myocardial infarction with LV dysfunction effects ranging from low BP or heart failure up to cardiogenic shock is associated with worse clinical outcome psot-STEMI [13]. All patients presented to the ER with chest discomfort or other symptoms suggestive of ACS were considered as high-priority triage cases. Routinely, the patients were generally assessed and examined for symptoms of low BP, heart failure and cardiogenic shock (Killip class II-IV). **The clinical condition on admission** was found significantly worse in the group of elevated admission troponin suggesting a more extensive myocardial damage on presentation. **Hypotension on admission** was recorded in 8 patients representing 18.2% of the whole study population and this was recorded statistically more significantly in the group of elevated admission troponin than the group of normal admission troponin, 7 patients (28.0%) versus 1 patient (5.3%) **(**P = 0.044). **Heart failure on admission,** (Killip class II-IV), was diagnosed in 9 patients out of the whole study population, representing 20.5%, and it was also significantly more common in the group of elevated admission troponin than the group of normal admission troponin, 7 patients (28.0%) versus 2 patients (10.5%) (P = 0.041). However, this was against **Matetzky and his colleagues** [8] who found the numbers of patients who had heart failure on admission, Killip class IV, were almost equal in both groups of admission troponin, 2 patients (3.6%) in the group of normal troponin and 2 patients (3.7%) in the elevated troponin group (P = 1.0).

Comparison of Coronary findings between both hs-cTnT groups:

Califf *et al.* [14] (Duke Jeopardy score) is a simple score that is used to assess the coronary artery disease severity and extension with 97% five-year survival in patients with a jeopardy score of 2 and 56% for patients with a score of 12 [14]**.** We applied Jeopardy score on our cases and found its mean value was higher in the group of elevated admission troponin (4.56 ± 2.86) than in the normal troponin group (4.21 ± 2.57) but this difference wasn't statistically significant.

Regarding the **culprit lesion location**, there was a statistically significant different distribution between both groups of troponin as in the normal group, more than half of the patients, 11 patients (57.9%), had the culprit lesion located in the RCA but most of the patients in the group of elevated troponin on admission, 18 patients (72.0%), had the culprit lesion located in the LAD (P = 0.043). Looking at the **culprit vessel flow** during the diagnostic angiography, the majority of cases in the study, 34 patients, had the culprit vessel totally occluded (TIMI 0 flow) but this finding was non significantly more in the normal troponin group than the group of elevated troponin, 84.2% (16 patients) versus 72.0% (18 patients) (P = 0.339).

The use of **IIb/IIIa inhibitor medications** was more for the patients who had normal troponin levels. It was used for 4 patients (12.5%) in the group of low level of troponin versus 2 patients (16.7%) in the group of elevated troponin but this difference was not statistically significant.

Admission hs-cTnT level and In-hospital follow up:

During the hospitalization time post-angioplasty, patients were evaluated for development of **post-infarction complication** including heart failure, major

bleeding, cardiogenic shock, in-hospital redo of coronary angiography, re-infarction and in-hospital death. We found that elevated troponin on presentation was an objective measure of the pre-hospital ischemic time and burden that affected the total rate of complications after PCI as it was significantly higher among the patients of elevated troponin on presentation. Nine patients (36.0%) of this group developed in-hospital complications against 3 patients (15.8%) in the normal troponin group (P = 0.048). Those 9 patients of the group of elevated troponin were distributed as following: 6 patients (24.0%) developed H.F, 1 patient (4.0%) developed cardiogenic shock, 1 patient (4.0%) needed in-hospital redo of coronary angiography but the stent was patent and didn't need further angioplasty and 1 patient had reinfarction and died during this hospitalization (4.0%). No patients had major bleeding (P = 0.048). On the other hand, only 3 patients in the group of normal troponin developed in-hospital complications; the 3 cases had symptoms of heart failure associated with cardiogenic shock and one of them also needed in-hospital redo of coronary angiography but also without further revascularization. This is similar to the results of **Matetzky and his colleagues** [8] study. They found that patients with elevated troponin were more likely to develop congestive heart failure (23% versus 9%, P 0.05) and shock or death (30% versus 9%, P = 0.006). They also found that elevated troponin remained a significant predictor of the composite end point after controlling for other clinical data that were available early in the course, including time to presentation and angiographic results. Also, this is in concordance with the **Brett and his colleagues** [9] study who found a statistically significant correlation between troponin rise on presentation and the in-hospital outcome after primary PCI as patients who had normal troponin on presentation had a relatively better outcome but mortality increased with elevated troponin levels on admission, regardless of baseline clinical risk. In this study, in-hospital mortality affected 1.8% of normal troponin group versus 5.1% mortality in the high troponin group.

CONCLUSION

This study findings supported that a single measurement of hs-cTnT provides significant incremental information to risk stratification after STEMI. It is an inexpensive, and easy available tool of value for assessment of prognosis, and our results suggest that the routine use of hs-cTnT values provide complementary information to the well-established risk factors. These data also confirmed a reminder that low biomarker levels should never be used to delay a decision to proceed to coronary angiography in a patient who had suspected STEMI.

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