Role of Cardiac Troponin I Level in Predicting in Hospital Outcomes in Patients with
ST-segment Elevation Myocardial Infarction in Erbil-Iraq

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Abstract

Background: The correlation of cardiac troponin I with early in-hospital outcomes in acute myocardial infarction is not well established.

Objective: To assess the role of troponin I in predicting in-hospital outcomes and early left ventricular systolic dysfunction in patients with ST-segment elevation myocardial infarction.

Patients and Methods: A prospective study which consist of 116 patients (74 were males and 42 were females), with ST-segment elevation myocardial infarction who had been admitted to the Coronary Care Unit from March 2015 to September 2015 were enrolled. Patients were divided according to the level of troponin I on admission into 3 groups (low, medium and high elevation).

Results: The mean age (+ SD) of the patients was 60+11.4 years. The troponin level of 66.2% of males was high compared with 52.4% of females (p=0.002). The incidence of acute pulmonary edema (21.1%), cardiogenic shock (7%) and early left ventricular systolic dysfunction (49.3%) was significantly higher among patients with high troponin level compared with (0%, 0% and 16%, respectively) among patients with low troponin level. All deaths and cardiac arrest were of high troponin level.

Conclusion: High admission troponin I in ST-segment elevation myocardial infarction permits early identification of patients at increased risk of major cardiac complications and death.

Key words: Cardiac troponin I, Ischemic Heart Disease.

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Introduction

According to the world health rankings, data source published by World Health organization (WHO) 2014,the age-adjusted death rate of coronary heart disease in Iraq was 187.65 per 100,000 populations. Iraq's rank was 22 worldwide and the second in Arab countries for the death due to CHD [1].

The Cardiac specific Troponin I (cTn-I) is expressed only in cardiac muscle, which allows this biomarker to be highly specific for myocardial damage [2]. In ST-segment elevation myocardial infarction (STEMI), the prognostic properties of cardiac troponins are influenced by the sampling time-point. A positive cardiac troponin on admission is

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probably reflective of longer ischemic time and more extensive myocardial necrosis, and consequently predictive of clinical outcomes [3]. There are some data suggesting that a negative troponin result reflects a good prognosis due to low incidence of cardiac death or non-fatal MI at 30 days [4].

As a marker of infarct size, cTnI is related to the extent of left ventricular remodeling and risk for development of chronic left ventricular dysfunction; and it improves prediction of these end points when added to the conventional risk stratification approach by assessment of left ventricular ejection fraction (LVEF) and volumes before discharge [5].

The correlation between admission cTnI level and the development of early left ventricular systolic dysfunction was not well defined.Up to our knowledge, there was no previous study done in Iraq.

The aim of the present study is to evaluate the role of admission cardiac troponin I level in predicting in-hospital outcome and early left ventricular systolic dysfunction (LVSD) in patients presenting with ST-segment elevation myocardial infarction.

Materials and Methods

This prospective study (case series) was conducted in the Coronary Care Unit (CCU) of Rizgary and Hawler Teaching Hospital between March 2015 and September 2015. A total of 116 consecutive patients presenting with chest painof 12 hours duration or less, were enrolled in the study.

The inclusion criteria were acute myocardial infarction for the 1st time, in patients with chest pain of 12 hours duration or less, and there was no chance to do early coronary angiography, percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). The patients presented within 12 hours were given thrombolytic therapy unless there was an obvious contraindication, and after that they were treated according to the management of the physician.

The patients who presented with the following were excluded from the study: a- Previous history of myocardial infarction, PCI or CABG. b-Previous history of heart failure proved clinically or by echocardiography c-Congenital or valvular heart diseases d-Left bundle branch block (LBBB).

The diagnosis of acute STEMI was based on the following criteria: Typical rise and/or fall of troponin with at least one of the following: a- Ischemic symptoms b-Electrocardiographic changes indicative of ischemia (ST-segment elevation)[6].

In this study, demographic variables, cardiovascular risk factors and physical examination for the Killip-Kimball classification(Killip class)[7],were obtained. All patients were studied prospectively without knowledge of the admission Troponin I levels for early in-hospital outcomes including cardiac standstill, arrhythmias and mortality. Resting Electrocardiography (ECG) and transthoracic Echocardiography was done to all patients to assess the left ventricular systolic dysfunction (which was defined as ejection fraction ejection fraction <50% in the absence of significant ischemic mitral regurgitation, or ejection fraction <55% in the presence of ischemic mitral regurgitation) [8].

Venous blood was withdrawn from patients at the time of admission. The blood sample was separated for a lot of laboratory markers including Troponin I,the normal value was 0.0-0.4ng/ml (>0.4ng/ml is abnormal) according to kit reference value, assessed by Biomerieux kit with enzyme-linked fluorescent assay (ELFA).

Patients were categorized into 3 groups according to the level of Troponin I at admission. Group-I patients with mildly elevated level (one fold elevation), Group-II

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patients with moderately elevated level (2-3 fold elevation), and Group-III patients with highly elevated level (> 3 fold elevation)[9]. All groups were evaluated and compared according to the baseline characteristics of the study population (demographics and risk factors), Global Registry of Acute Coronary Events (GRACE) risk score, in-hospital outcomes or complications and echocardiographic findings. GRACE risk score was also categorized into 3 groups, group A (low risk <126), group B (intermediate risk 126-154), and group C (high risk >154) [10].

Verbal informed consent was obtained from all patients, and the protocol was approved by theEthics Committee of the College of Medicine of Hawler Medical University.

Statistical analysis

The statistical package for social sciences (SPSS, version 19) was used for data analysis. Chi square test of association was used to compare proportions. Fisher's exact test was used when the expected count of more than 20% of the cells of the table was less than 5. P-value of ≤ 0.05 was considered as statistically significant.

Result

A total of 116 patients with first time acute ST-segment elevation myocardial infarction were included in the present study. The mean age ($\pm$SD)was 60$\pm$11.4 years ranging from 33-99 years. Most patients were males (63.7%). The troponin level of 66.2% of males was high compared with 52.4% of females (P=0.002). Family history of IHD shows statistically significant association with high troponin level (P=0.049). Although other socio-demographic and clinical variables did not show any statistically significant association with troponin level, it is worth to mention that a relatively high proportion of diabetics, hyperlipidemics, and smokers had high troponin levels (p > 0.05).as shown in Table 1.

Table (1): Troponin-I levels by socio-demographic and clinical variables.

	N	Troponin-I						p
		Low		Medium		High		
		No.	%	No.	%	No.	%	
Age (years)								
< 50	16	4	25	1	6.3	11	68.8	0.102*
50-59	42	14	33.3	9	21.4	19	45.2	
60-69	31	3	9.7	6	19.4	22	71	
≥ 70	27	6	22.2	2	7.4	19	70.4	
Gender								
Male	74	10	13.5	15	20.3	49	66.2	0.002
Female	42	17	40.5	3	7.1	22	52.4	
BMI categories								
< 25	63	20	31.7	11	17.5	32	50.8	0.277*
25-29	33	4	12.1	5	15.2	24	72.7	
30-34	17	3	17.6	2	11.8	12	70.6	
≥ 35	3	0	011	0	0	3	100	
Diabetes								
No	84	21	25	15	17.9	48	57.1	0.319
Yes	32	6	18.8	3	9.4	23	71.9	
Hypertension								
No	53	9	17	11	20.8	33	62.3	0.182
Yes	63	18	28.6	7	11.1	38	60.3	
Hyperlipidemia								
No	35	9	25.7	7	20	19	54.3	0.557
Yes	81	18	22.2	11	13.6	52	64.2	
Smoking								
No	64	19	29.7	8	12.5	37	57.8	0.163

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Yes	52	8	15.4	10	19.2	34	65.4	
Alcohol drinking								
No	111	26	23.4	16	14.4	69	62.2	0.250*
Yes	5	1	20	2	40	2	40	
Family history of IHD**								
No	87	23	26.4	16	18.4	48	55.2	0.049
Yes	27	3	11.1	2	7.4	22	81.5	
Thrombolytic therapy								
No	63	20	31.7	12	19	31	49.2	0.014
Yes	53	7	13.2	6	11.3	40	75.5	
***By Fisher's exact test. **There is missing data**								

There was a statistically significant association between troponin levels and risk according to GRACE score. Patients with high troponin levels had high risk GRACE score (P=0.026), as shown in Table 2.

Table (2): Association between troponin levels and GRACE+score.

Troponin-I categories	N	Risk according to GRACE[+] score						p
		Low		Intermediate		High		
		No.	%	No.	%	No.	%	
Mild	27	4	14.8	10	37	13	48.1	0.026*
Moderate	18	1	5.6	5	27.8	12	66.7	
Severe	71	1	1.4	16	22.5	54	76.1	
Total	116	6	5.2	31	26.7	79	68.1	
***By Fisher's exact test. Agreement = 54.3%**								

* GRACE:GlobalRegistryofAcuteCoronaryEvents

Table 3 shows no significant association between in- hospital death and cardiac arrest at admission with troponin level. It is worth to mention that all deaths (four cases), seriousarrhythmias(4 cases) were of high troponin level. The same table shows that 7% of those with high troponin level developed cardiogenic shock (KILLIP class IV) compared with 0% among those with low and medium troponin level. In addition to that, 21.1% of those with high troponin developed acute pulmonary oedema(KILLIP class III) heart failure compared with 11.1% among those with medium troponin and 0% among those with low troponin, while the majority of those with low troponin level developed KILLIP class 1 and II (p = 0.011).

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Table (3): Rates of complications in different categories of troponin.

	N	Troponin-I						p
		Low		Medium		High		
		No.	%	No.	%	No.	%	
Serious Arrhythmias (VF/VT)								
No	103	24	100	17	100	62	93.9	0.467*
Yes	4	0	0	0	0	4	6.1	
Heart block								
No	111	25	92.6	17	94.4	69	97.2	0.429*
Yes	5	2	7.4	1	5.6	2	2.8	
In hospital death								
No	109	27	100	17	100	65	94.2	0.475*
Yes	4	0	0	0	0	4	5.8	
Total	113	27	100	17	100	69	100	
Cardiac arrest at admission								
No	112	27	100	18	100	67	94.4	0.472*
Yes	4	0	0	0	0	4	5.6	
Total	116	27	100	18	100	71	100	
KILLIP class								
I	66	19	70.4	8	44.4	39	54.9	0.011*
II	28	8	29.6	8	44.4	12	16.9	
III	17	0	0	2	11.1	15	21.1	
IV	5	0	0	0	0	5	7	
Total	116	27	100	18	100	71	100	
***By Fisher's exact test.**								

The incidence of early LVSD (49.3%) was significantly higher among patients with high troponin levels (P=0.001). It is worth to mention that 3 of 4 patients with severe ischemic mitral regurgitation (IMR) had high troponin levels compared to (0) patient with low troponin level as shown in Table 4.

Table (4): Ventricular systolic dysfunction and ischemic mitral regurgitation diagnosed by transthoracic echocardiography versus troponin I level.

	N	Troponin-I						p
		Low		Medium		High		
		No.	%	No.	%	No.	%	
LVSD**								
No	70	21	84	15	88.2	34	50.7	0.001
Yes	39	4	16	2	11.8	33	49.3	
Total	109	25	100	17	100	67	100	
Ischemic mitral regurgitation (IMR)								
No MR	72	23	85.2	13	72.2	36	50.7	0.055*
Mild	26	3	11.1	2	11.1	21	29.6	
Moderate	14	1	3.7	2	11.1	11	15.5	
Severe	4	0	0	1	5.6	3	4.2	
Total	116	27	100	18	100	71	100	
***By Fisher's exact test. **There is missing data**								

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Discussion

This study was done as an attempt to evaluate the role of troponin I in predicting the in-hospital outcomes in patients with first STEMI.

Results showed that admission cTn-I was very high among males (66.2%) compared with (52.4%) of females (p=0.002); these results goes in parallel with Shah et al (2015); that study found that women were less likely to have biomarker evidence of myocyte necrosis, this can lead to under diagnosis of myocardial infarction in women and contribute to sex inequalities in treatments and outcomes[11].

The agreement rate between troponin levels and GRACE score in our study was 54.3%. Some of the cases categorized as 'mild troponin' were categorized as 'high grace score'. These findings are consistent with Bonaca et al study (2010), which found low-level increment in cTnI using a sensitive assay identify patients at higher risk of death or MI[12].

In contrary to other studies, no significant association was found between in hospital death and cardiac arrest at admission with troponin level, but it is worth to mention that all deaths (four cases) were of high troponin level; this may be explained by small number of patients in this study. Entman et al found that the detection of the highly specific marker cardiac troponin I in blood is an independent risk factor that identifies patients presenting with acute coronary syndrome who are at increased risk of death[13].

This study found that 7% of those with high troponin level developed cardiogenic shock (KILLIP class IV) compared with 0% among those with low and medium troponin level. In addition to that, 21.1% of those with high troponin developed KILLIP class III heart failure compared with 11.1% among those with medium troponin and 0% among those

with low troponin, while the majority of those with low troponin level developed KILLIP class 1 and II (p = 0.011), these findings were consistent with Peacock et al study (2011) which found that patients with admission troponin I positive patients had lower systolic blood pressure and lower ejection fractions[14].

The incidence of early LVSD (49.3%) was significantly higher among patients with high troponin levels (P=0.001). These results are consistent with other previous studies where high levels of admission cTnIwas associated with more severe heart failure, including worse left ventricular function, also troponins were associated with more severe symptoms, more need for aggressive supportive measures, including inotropic therapy, and worse outcomes [14,15].

In conclusions: High admission cardiac troponin I in first ST-segment elevation myocardial infarction, permits the early identification of patients at increased risk of cardiac death, cardiogenic shock, acute pulmonary edema and early left ventricular systolic dysfunction.

Conflicts of interest : The authors report no conflicts of interest..

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