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Prognostic value of high-sensitive cardiac troponin-T in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention in Erbil cardiac center

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Abstract

Background and objectives: Patients with ST-segment elevation myocardial infarction have higher rates of major adverse cardiovascular events including deaths, and it is recommended that these patients should undergo primary percutaneous coronary interventions. The objective of this study was to determine correlation of high-sensitivity cardiac troponin-T measured on admission with the mortality and major adverse cardiovascular event. **Methods:** In this study, 167 patients with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary interventions were enrolled. The cut off value of high-sensitivity cardiac troponin-T measured according to the highest Youden's index was 528 ng/L. According to this cut off value, we divided the patients in to two groups: those with high-sensitivity cardiac troponin-T $\geq$ 528 ng/L (33 patients), and those with high-sensitivity cardiac troponin-T < 528 ng/L (134 patient). **Results:** The mean age + SD were 57.56 + 11.11 years; the rate of death was 10.8% (n=18) within follow-up period. Incidence of death among patients with higher cardiac troponin-T values ($\geq$ 528 ng/L) was 27.3%, which was significantly higher than the incidence (6.7%) among those with lower cardiac troponin-T values. After adjusting for the other factors, higher cardiac troponin-T values were associated with higher death rate. Old age and diabetes was found to be significant risk factors for death. While other outcomes and complications were statistically insignificant. Those who suffered cardiovascular death tend to be older, female gender, diabetics, hypertensive, and had diffused coronary arterial disease. **Conclusions:** Initial cardiac troponin-T on admission is independently associated with the higher risks for 2-year all-cause mortality in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary interventions.

Key words: High-sensitivity cardiac troponin-T, Myocardial infarction, ST-segment elevation.

Introduction

Cardiovascular disease (CVD) is the leading cause of death in both men and women in the United States, it is estimated that ischemic heart disease will be the number one cause of disability and death worldwide by the year 2020[1]. Acute coronary syndromes (ACS) almost all results from coronary atherosclerosis, with superimposed coronary thrombosis caused by erosion or rupture of an atherosclerotic lesion[2]. As the coronary blood flow is occluded, the ST segment elevation occurs on electrocardiography {ST segment elevation acute myocardial infarction(STEMI)} and impaired myocardial perfusion cause ischemia that result in myocardial cell death or injury, cardiac arrhythmias, and ventricular dysfunction. Within 15 minutes of occlusion, myocardial cell death begins and rapidly proceeds from endocardium to epicardium in a wave front. Salvaging myocardium partially can be achieved by restoration of blood flow within 3 to 6 hours; the degree of salvage and the duration of ischemia are inversely proportional[3,4]. After the damage of myocardial cells, proteins are released into the blood. The availability of plasma and serum cardiac markers, with more enhanced sensitivity for myocardial injury, makes clinicians detect much lower levels of injury, but does not provide information on the cause of the damage[5]. Cardiac troponin (cTn) is the preferred biomarker to identify myocardial injury, which consists of three subunits that regulate the calcium-mediated contractile function of striated muscle. These are Troponin C, Troponin T and

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Troponin I[6]. Following myocyte injury, the initial release of cardiac-specific cTnT and cTnI is from the cytosolic pool, (6 - 8% of cTnT dissolved in the cytosol; and 2 - 3% of cTnI is in the cytosolic pool) followed by release of the myofilament bounded protein[7]. The cornerstone of the diagnostic criteria for myocardial infarction (MI) is detection of a rise and fall in cTnT or cTnI in the appropriate clinical setting[8], which can be detected approximately 3 hours after onset of chest pain by conventional assays (non–high-sensitivity)[9]. As the cardiac troponin is continuously released from degeneration of contractile apparatus in necrotic myocytes, causing persistent elevations up to 10-14 days in cTnT and 4-7 days in cTnI after MI[10], which is helpful for late diagnosis of MI. Successful recanalization of the infarct-related artery has a rapid release of cardiac troponins, which may indicate reperfusion. With high-sensitivity assays there is more precise measurement of very low concentrations of cardiac troponin, and this term high-sensitivity cardiac troponin (hs.cTn) is used for assays which are capable for detection of cTn in more than 50% of healthy population[7,8,11]. The objective of this study was to determine correlation between levels of high-sensitive cardiac troponin-T measured on admission with the mortality and major adverse cardiovascular events.

Patients and methods

This study was a prospective review of cases of STEMI admitted to surgical specialty hospital/cardiac center in Erbil city/Iraq from January 2017 to Jun 2017, and who underwent successful PPCI. Fourth universal definition of myocardial infarction criteria was used for acute MI and ST elevation[12]. After obtaining informed consent from patients or their relatives, according to the above criteria, 225 patients enrolled in this study, 58 patients lost contact during follow-up either because they stopped responding, or because they closed or changed their given phone numbers. Patients with the diagnosis of any grades of renal failure (RF), presence of cardiogenic shock or pulmonary oedema, those who underwent emergency coronary artery bypass graft (CABG) and patient with severe comorbidity such as chronic obstructive pulmonary disease, liver or neurological diseases were excluded from the study. The plasma concentration of hs-cTnT was measured with the high sensitivity assay by a Cobas e411 immunoanalyzer (Roche Diagnostics), according to the instructions of the manufacturer (detection limit of 5 ng/L, 99th percentile in the general population of 14 ng/L and 10% coefficient of variation level of 13 ng/L). All patients underwent PPCI according to global and local guidelines. The patients have been followed up for up to 20 to 24 months (median 22 months) after STEMI. The primary endpoint of interest in this study was all-cause mortality. The secondary endpoint was other major adverse cardiac event (MACE) including heart failure, reinfarction, readmission, and in need for further revascularization. Information on deaths was obtained from hospital records or telephone contact with relatives of the patient. Arterial hypertension was diagnosed in the presence of active treatment with antihypertensive agents or otherwise as a systolic blood pressure of $\geq$140 mm Hg and/or diastolic blood pressure of $\geq$90 mm Hg on at least 2 separate occasions[13]. Diabetes mellitus was diagnosed in the presence of active treatment with antidiabetic agents or based on current guidelines[14]. Heart failure was diagnosed according to Modified Framingham clinical criteria[15], or on echocardiography in case of LVSD and EF less than 50%. The infarct related artery is a coronary artery that contains the culprit lesion which is defined as a lesion presenting as an acute occlusion[16]. Successful PCI was defined as TIMI III flow across culprit lesion and less than 30% residual stenosis[17]. Data were analyzed using the Statistical Package for Social Sciences (SPSS, version 22). 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. ROC curve analysis was used in order to determine the cut off value of hs-cTnT that predict death. Youden's index was calculated, and the highest value of it was considered to determine the cut off value of hs-cTnT that gives the highest sensitivity and specificity[18]. Factors found (By the Chi square test) to be associated with death were entered into a binary logistic regression model in order to show the independent effect of each factor. Number of vessels was removed from the model because of insufficient sample size. A p-value of $\leq$ 0.05 was considered statistically significant. The study was approved by the local ethical committee of the Kurdistan Higher Council of Medical Specialties (KHCMS).

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Results

In this study, a total of 167 patients were studied. Their mean age + SD were 57.56 + 11.11 years, ranging from 29 to 87 years; the median was 58 years. The majority (77.25%) of the sample were males. The male: female ratio was 3.4: 1. The baseline patient characteristics and risk factors for CVD are shown in Table (1), which shows that the incidence of death was highest (75%) among patients aged ≥ 80 years, while it was much less among younger patients (p-value < 0.001). The incidence of death among females (21.1%) was significantly higher than the incidence (7.8%) among males (p-value= 0.033). It is evident in the table that the more the number of affected vessels, the higher the mortality (p-value = 0.005). No significant association was detected between death with smoking (p-value= 0.218) and infarct related artery (p-value= 0.379). On the other hand, significant association was detected with hypertension (p-value= 0.035) and diabetes (p-value <0.001).

Table (1): Clinical characteristics of patients according to survival status.

	Yes		No		Total			
	No.	(%)	No.	(%)	No.	(%)	p-value	RR (95% CI)
Age (years)								
20-39	1	(8.3)	11	(91.7)	12	(100.0)		
40-59	1	(1.3)	74	(98.7)	75	(100.0)		
60-79	13	(17.1)	63	(82.9)	76	(100.0)		
≥ 80	3	(75.0)	1	(25.0)	4	(100.0)	< 0.001	NA
Gender								
Female	8	(21.1)	30	(78.9)	38	(100.0)		
Male	10	(7.8)	119	(92.2)	129	(100.0)	0.033*	2.716 (1.153-6.395)
No. of affected vessels								
One	0	(0.0)	55	(100)	55	(100.0)		
Two	8	(14.0)	49	(86)	57	(100.0)		
Three	10	(18.2)	45	(81.8)	55	(100.0)	0.005	NA
Current smoking								
Yes	4	(6.8)	55	(93.2)	59	(100.0)		
No	14	(13.0)	94	(87.0)	108	(100.0)	0.218	0.523 (0.180-1.517)
Hypertension								
Yes	11	(17.2)	53	(82.8)	64	(100.0)		
No	7	(6.8)	96	(93.2)	103	(100.0)	0.035	2.529 (1.0234-6.188)
Diabetes								
Yes	13	(24.5)	40	(75.5)	53	(100.0)		
No	5	(4.4)	109	(95.6)	114	(100.0)	< 0.001	5.592 (2.102-14.880)
Infarct related artery								
LAD	9	(9.8)	83	(90.2)	92	(100.0)		
RCA	9	(13.8)	56	(86.2)	65	(100.0)		
LCX	0	(0.0)	10	(100)	10	(100.0)	0.379	NA
Total	18	(10.8)	149	(89.2)	167	(100.0)		

*By Fisher's exact test. RR = Relative Risk. CI = Confidence interval. NA= Not Applicable

ROC curve analysis showed that the area under the curve was 0.643 (95% CI was 0.482-0.804). The cut off value of hs-cTnT measured according to the highest Youden's index was 528 ng/L, giving sensitivity (for hs-cTnT values ≤528 ng/L) of 50% and a specificity of 83.9%, as presented in Figure (1) and Table (2).

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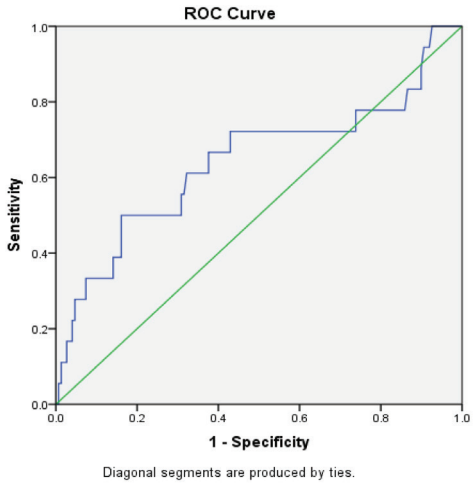

Figure (1): ROC curve analysis where the hs-cTnT level is the predictor of death

Table (2): Area under the curve

			Asymptotic 95% Confidence Interval	
Area	Standard error	p-value	Lower Bound	Upper Bound
0.643	0.082	0.048	0.482	0.804

Note: The hs-cTnT cut off value = 528 giving a sensitivity of 50% and a specificity of 83.9%.

According to this cut off value (528ng/L), the patient had been divided in to two groups: first group (33 patients), those with hs.cTnT ≥ 528 ng/L (ranging from 528 to 1890 ng/L), and second group (134 patient), those with hs.cTnT < 528 ng/L (ranging from 14 to 522 ng/L).

It is evident in Table (3) that the incidence of death among patients with higher hs-cTnT values (≤528 ng/L) was 27.3% which was significantly higher than the incidence (6.7%) among those with lower hs-cTnT values (p-value = 0.002).

Table (3): Incidence of death by hs.cTn level.

	Death							
	Yes		No		Total			
hs-cTnT	No.	(%)	No.	(%)	No.	(%)	p-value	RR (95% CI)
High (≥ 528 ng/l)	9	(27.3)	24	(72.7)	33	(100.0)		
Low (< 528 ng/l)	9	(6.7)	125	(93.3)	134	(100.0)	0.002*	4.061 (1.750-9.423)
Total	18	(10.8)	149	(89.2)	167	(100.0)		

*By Fisher's exact test.

Binary logistic regression analysis in Table (4) shows that high hs-cTnT values were associated with death (OR = 4.035, 95% CI = 1.265-12.872) after adjusting for the other factors. Old age (OR = 6.310, 95% CI = 1.265-31.489) and diabetes (OR = 5.092, 95% CI= 1.259-20.599) found to be significant risk factors for death.

Table (4): Binary logistic regression analysis between death as a dependent variable and several covariates.

			95% CI for OR		
	B	p-value	OR	Lower	Upper
High hs-cTnT (≥ 528 ng/l)	1.395	**0.018**	4.035	1.265	12.872
Age (≥ 60 years)	1.842	**0.025**	6.310	1.265	31.489
Female gender	0.804	0.177	2.235	0.695	7.185
Hypertension	-0.372	0.599	0.690	0.173	2.752
Diabetes	1.628	**0.022**	5.092	1.259	20.599
Constant	-4.708	0.000	0.009		

B: standardized regression coefficient. Age < 60 years (reference)

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No significant association was detected between hs-cTnT level with the incidence of outcomes of STEMI such as heart failure (p-value = 0.125), further PCI (p-value = 0.288), readmission (p-value = 0.552), recurrent chest pain (p-value = 0.726), and other complications such as RF, reinfarction, stent thrombosis, stroke and upper GIT bleeding (p-value = 0.205), as presented in Table (5).

Table (5): Incidence of outcomes and other complications after STEMI by hs-cTnT level.

	Hs-cTnT						p-value	RR (95% CI)
	High		Low		Total			
Outcomes	No.	(%)	No.	(%)	No.	(%)		
Heart failure								
Yes	8	(24.2)	18	(13.4)	26	(15.6)		
No	25	(75.8)	116	(86.6)	141	(84.4)	0.125	1.805 (0.861-3.785)
Further PCI								
Yes	4	(12.1)	27	(20.1)	31	(18.6)		
No	29	(87.9)	107	(79.9)	136	(81.4)	0.288	0.602 (0.226-1.601)
Readmission								
Yes	5	(15.2)	15	(11.2)	20	(12.0)		
No	28	(84.8)	119	(88.8)	147	(88.0)	0.552*	1.354 (0.550-3.457)
Recurrent chest pain								
Yes	6	(18.2)	21	(15.7)	27	(16.2)		
No	27	(81.8)	113	(84.3)	140	(83.8)	0.726	1.160 (0.509-2.643)
Others								
Yes	6	(18.2)	12	(9.0)	18	(10.8)		
No	27	(81.8)	122	(91.0)	149	(89.2)	0.205*	2.030 (0.823-5.008)
Total	**33**	**(100)**	**134**	**(100)**	**167**	**(100)**		

*By Fisher's exact test.

Discussion

This study demonstrates that patients with STEMI who underwent primary PCI and had higher admission hs.cTnT levels were associated with higher risk of 2-year all-cause mortality. Possibly, the more hs.cTn, the more time lapsed from symptom, the more necrosis occurs, the more extensive and established ischemic outcome.

This study also observed that the non-survived group have higher levels of hs.cTnT than the survived group, and they were mostly older in age. This was also shown by Ndrepepa G et al[19], who studied 818 patients with STEMI treated by PPCI, and proved the non-survivors were older in age, more likely to have diabetes, higher hs-cTnT levels, lower estimated GFR and left ventricular function, and concluded that admission and peak hs-cTnT is correlate independently with higher 3 year mortality.

In current study, female gender have more risks for development of complications of MI, which was similar to Wijnbergen I et al[20] and De Luca G[21]. As explained by van der Meer et el[22], for their unfavorable risk profile and longer symptom to balloon time. We observed that diabetic patients have higher levels of hs-cTnT and higher mortality on follow-up. Piccolo et el[23], and Van der Schaaf at el[24], concludes that diabetes confers a worse prognosis. In this study, higher mortality rate was observed in non-smoker than current smoker patients, although statistically insignificant, it is possibly due to higher mean age, and more risk factors like DM and HTN in non-smoker. Same showed by Ndrepepa G et al.[19]

Around 5385 of the STEMI patients treated with PPCI, studied by Velders et al[25], showed that hs-cTnT measured on admission predict occurrence of subsequent cardiovascular death or MI. Wang et al[26] studied 173 STEMI patients who underwent PPCI, the incidences of MACE at 30 days and 1 year were 10% and 18%, respectively, and they concluded that hs-cTnT measured on admission was

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strongly associated with 30 days and 1 year rates of MACE after PPCI.

Other studies show that hs.cTn can predict higher cardiovascular mortality and poor outcomes (as Ndrepepa G et al[19], divided circulating hs.cTn to 4 fractions) according to the time of measurement, each of them having multiple possible sources. The first fraction is caused by physiological cardiomyocyte turnover[27], de Lemos JA et al[28] showed the association between hs.cTnT with structural heart disease and subsequent risk for mortality in general population. The second fraction is due to enhanced cardiomyocyte stress by many abnormal stimuli (like comorbidities and cardiovascular risk factors) which cause leakage of troponin through multiple mechanisms[29]. The third fraction is from acute myocardial ischemia/necrosis in patients presenting with an ACS, as in the early phase of STEMI detection of troponin I correlating closely with infarct size[30] and the mortality[31]. The fourth fraction related to PPCI complications, and correlate to poor outcome[32].

Although there were higher rates of other outcomes of STEMI (such as HF) and other complications (such as reinfarction) in correlation to the levels of hs-cTnT, it was statistically insignificant.

On the other hand, there are some conflict and controversy regarding levels of cTn and outcomes of STEMI, and no clear published data present on the value of initial cTn level for predicting clinical outcomes. Giannitsis et al33 showed that higher levels of cTnT associated with poorer epicardial and tissue perfusion and all-cause mortality after PPCI; but the rates of the combined end point and nonfatal reinfarction were statistically not different at 1 and 9 months. Khullar et al[34] showed that peak hs-cTnT measurements did not predict all-cause mortality or repeat revascularization at 1year. Regarding the use of cardiac biomarkers on admission including cTn I, a study done by Jeong et al[35] showed that a study of 207 patients with STEMI who were treated by PPCI didn't show any predictive value added on the validated TIMI risk score in assessing MACE (death, reinfarction, and new or worsening congestive heart failure) through 1 year of follow-up. These controversies are even present in stable CAD undergoing PCI[36] and in the size of infarction. While Boden et al[37], show cTn significantly correlate with infarct size, no significant correlation was found by Ferraro et al[38].

Although the exact reasons for such a high degree of controversy between these studies in the prognostic value of cTn levels are unclear, the differences might be related to patient characteristics, time (peak or initial) and type of cTn assays (conventional or high sensitive); time of treatment, disease severity, sample sizes and exclusion criteria (in this study we exclude renal failure, cardiogenic shock, pulmonary odema) may also affect. Furthermore, in most patients treated by PCI, the variable technical procedure might have great effect on longer patency of stents and outcomes later on. By time, according to the most data and researches, there is a close association between hs.cTn with baseline cardiovascular risk and the extent of myocardial damage in patients with STEMI and subsequent mortality.

Conclusions

Initial hs-cTnT on admission is independently associated with the higher risks for 2-years all-cause mortality in patients with STEMI undergoing PPCI, possibly due to higher risk profile like age, DM and multi-vesssel coronary artery lesions in patients with higher levels of hs.cTnT.

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