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ORIGINAL ARTICLE

Value of Serum Copeptin inEarly Diagnosis and Prediction of Short-Term Outcomes in STEMI

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ABSTRACT

The background: The early diagnosis and exclusion of acute coronary syndrome is still challenging, that requires monitoring of the patients and serial measurements of cardiac biomarker Hs-c-TnsI . The addition of cardiac biomarker serum Copeptin may reflect the early ischemic cardiac injury. We **aimed** at detection of the diagnostic value of serum copeptin level and its role in prediction of short-term outcomes; MACCE in STEMI patients as a new cardiac biomarker. **Subjects and methods:** We included ninety patients attended an emergency cardiac care department due to acute chest pain (within less than three hours from onset of the chest pain) and were divided into; the 1st group included forty patients (acute STEMI who successfully received primary PCI). The 2nd group included fifty Patients (other diagnosis; (non-AMI)). The study endpoint was MACCE. The patients were subjected to the full laboratory profile (CBC, kidney function tests, total CK, CK- MB, highly sensitive Cardiac Troponin I, serum Copeptin). **Results**: The ROC analysis showed that At AUC of 0.891 and cut off value of >12.4 pmol/l, copeptin had a sensitivity of 75 % ,specificity 92 %, PPV 88.2 % ,NPV 82.1 % and accuracy 84.4 % in early diagnosis of acute MI. There was statistical significance of serum Copeptin level that correlated with CK-MB and hs-cTnI. In **conclusion,** our study revealed that Copeptin can early diagnose acute myocardial infarction but not superior to highly sensitive troponin. Higher serum copeptin levels were independent prognostic predictors of MACCE in acute STEMI patients during short-term follow up
Keywords: Copeptin; acute STEMI; MACCE

INTRODUCTION

Early Diagnosis of (ACS) acute coronary syndrome may reduce morbidity, mortality, and economic costs [1]. As well, early immediate intervention is a critical decision for clinicians [2]. According to recent guidelines, troponins besides ECG are used to diagnose ACS in patients presented with acute onset of chest pain [3, 4]. However, using troponins is time-consuming that needs serial changes in troponin level, especially in NSTEMI. The release of cardiac biomarker Hs-cTnI is delayed in comparison with the start of myocardial ischemia and necrosis [5], and is a very sensitive cardiac biomarker, resulting in high false-positive

results [6, 7]. Serum Copeptin, the C-terminal pro-AVP is a biomarker that regulated via three AVP receptors, variations in blood pressure and plasma osmolality [3, 4]. Copeptin is easier to measure than AVP [5, 6]. Previous studies found that the copeptin may be elevated because of endogenous severe hemodynamic stress response within 30 min after chest pain onset in ACS patients attended the emergency department [8, 9, 10]. The serum Copeptin level has a high negative predictive value in early diagnosis and rule-out acute MI, and no need for serial Copeptin level, in contrast to cardiac troponin [11]. Nowadays, serial level of cardiac biomarkers (hs-cTnI and NT-proBNP) have been used for the prognosis assessment and predict short term clinical outcome post-acute coronary syndrome [12,13]. However, the level of the serum copeptin in prognosis assessment and detection of MACCE in ACS patients was less validated and has not been recommended yet [14,15,16]. We aimed in this study to detect the early diagnostic value of serum copeptin level and its role in prediction of short-term outcomes; the major adverse cardiac and cerebrovascular events (MACCE) in STEMI patients as a new cardiac biomarker.

SUBJECTS AND METHODS

This prospective comparative analytical study was carried out at Cardiology department, Zagazig Universityfrom May 2019 to October 2019. We included ninety Patients attended an emergency cardiac care department due to acute chest pain (within less than three hours from the chest pain onset). They were managed according to the (ESC) European Society of Cardiology recent guidelines [17] and were divided into two groups; the 1st group included forty patients (acute STEMI: acute chest pain, elevated Hs-cTnI and diagnostic ECG of acute ST-Elevation MI and who successfully received primary PCI. The 2nd group included fifty Patients (other diagnosis; (non-AMI)). We excluded Patients whose were known to have heart failure, renal failure, under corticosteroid therapy, patients with acute chest pain lasting more than four hours from presentation, Patients with previous history of AMI, PCI, CABG, Atrial fibrillation, paced rhythm, and dilated cardiomyopathy. The study endpoint was Major adverse cardiovascular and cerebral events (MACCE) as in-stent restenosis, no- reflow or slow flow post primary PCI, recurrent ACS, all-cause mortality, acute heart failure requiring hospitalization or acute stroke within three months post-acute ST-elevation MI. The adverse short- term outcomes were coded according to ESC recent guidelines.

Our patients were subjected to the following:

A. History taking including: age, gender, the time between the chest pain onset to the first medical contact and rapid complete chest pain analysis; (onset, course, duration, precipitating factor, relieving factors, and associated symptoms). Cardiac risk factors such as hypertension, diabetes mellitus (DM), smoking, addiction, MI, PCI, CABG, history of other symptoms suggestive of cardiac problems, history of current medications, history of systemic disease and family history of premature ischemic heart disease.

B. Full clinical examination: Blood pressure measurement, heart rate, clinical examination: For diagnosis & exclusion of patient with exclusion criteria.

C. The twelve lead resting surface electrocardiography was recorded at the emergency cardiac care unit for all ninety patients. The Patients were divided according to the presenting ECG into the 1st group: forty patients who were presented with acute STEMI for primary PCI. 2nd group: fifty patients who were presented with other diagnosis (non-AMI).

D. The laboratory profile: CBC, kidney function tests, total CK, CK- MB, highly sensitive Cardiac Troponin I, serum Copeptin were done for all patients presenting to E.R. with chest pain symptoms suggestive of acute MI. The blood samples were withdrawn on time of admission. Hs-cTnI had been measured automatically. Serum copeptin measurement; venous blood samples were collected that were immediately centrifuged for ten minutes and processed according to a standardized operating procedure [13].The usage of the enzyme-linked immunosorbent assays for measuring the Serum copeptin level (Changjin, Ltd., China).The measuring level was 1.9–2,000 pmol/L.

E. The two- Dimensional Echocardiography was performed as early as possible prior to or immediately after admission to CCU on the first day.The estimation of LV EF % by modified Simpson's method. The calculation of (WMSI) wall motion score index was done for all patients.

F. Coronary angiography and primary PCI: Group I underwent coronary intervention according to the recent ESC guidelines [17]. Multiple angiographic views were interpreted by two interventional cardiologists. The Classifications of coronary lesions was based on the (ACC/AHA) American College of Cardiology/American Heart Association recent guidelines [18,19]. The ST-segment elevation MI management was based on recent guidelines and evaluating the infarct-related culprit artery by coronary angiography [20]. Initial TIMI flow grading and after primary PCI were assessed.

Ethical consideration: We obtained informed signed consent from all patients and his /or her first-degree relatives. The IRB approval from ethical committee was obtained.

Statistical analysis:

The data of our study were analyzed using (SPSS) (USA) Statistical Program for Social Science version twenty-five for windows and MedCalc 14.8 for windows (MedCalc Software, Belgium). The calculation of (mean ± (SD)) for parametric numerical quantitative data. The calculation of Median for non-parametric numerical quantitative data. The calculation of Qualitative data as frequency and percentage. The Independent-T-test was used when the comparison between two means of normally distributed data. The Mann Whitney U test was used when the comparison between two independent groups without normally distributed data. Chi-square test was used for comparison between two categorical variables. The Spearman's correlation coefficients were calculated, (+) indicate direct correlation & (-) indicate inverse correlation, also values near to one indicate strong correlation & values near zero indicate weak correlation. The (ROC) curve was used to find the optimal cut-off values. Probability (P value): the level of significance. P value > 0.05: Non significance (NS), P value < 0.05: Significant (S) and P value < 0.01: Highly significant (HS)

RESULTS

Our patients were divided into two main groups. The Mean age of group I was 54.4 ± 7.5years Vs 51.3 ± 10.7 years in group II. The male patients represented 67.5%. in group I Vs 66 % in group II. The risk factors distribution in group I was 60 % for smoking, 27.5% for diabetes, 12.5% for dyslipidemia, 42.5% for hypertension and 17.5% of the family history of premature CAD. the Risk factors distribution in group II was 44 % for smoking, 26% for diabetes, 36% for hypertension, 16% for dyslipidemia and 14 % of family history of premature CAD. As regards the comparison between these demographics and the clinical data, were statistically non-significant (table 1). There was a statistically high significance difference between the mean EF % in group I (53.3± 8.6) Vs group II (64.8 ± 8.0) (P value <0.001).The comparative study of the median WMSI between group I 1.25 (1.08 – 1.38) and group II 1.0 (1.0 – 1.0)was a statistically high significance difference (P value <0.001).The comparative study of , the median range of CK-T level between group I (166.5 IU/L) and group II (107.5 IU/L) was a statistically high significance difference (P value <0.001).The comparative study of , the median range of CK-MB level between group I (27 IU/L) and group II (19 IU/L) was a statistically high significance difference (P value <0.001).The comparative study of , the median range of Hs-cTn I level between group I (0.31 ng/L) and group II (0.006 ng/L) was a statistically high significance difference (P value <0.001).The comparative study of , the median range of copeptin level between group I (16 pmol/L) and group II (6.9 pmol/L) was a statistically high significance difference (P value <0.001) (Table [1]). The ROC curve analysis in group I for laboratory data; At an area under the curve of 0.758 and cut off value of total CK =147 iu/l, with 70% sensitivity , 72 % specificity, 66.7% positive predictive value , 75% negative predictive value and 71.1%accuracy . At an area under the curve of 0.817 and cut off value of CK MB 24 iu/l, with 70% sensitivity, 76% specificity, 70%positive predictive value, 76%negative predictive value and 73.3%accuracy. At an area under the curve of 0.97 and cut off value of hs-cTnI >0.6 ng/l, with92.5%sensitivity ,96%specificity, 94.9%positive predictive value, 94.1%negative predictive value and 94.4% accuracy .At an area under the curve of 0.891 and cut off value of copeptin >12.4 pmol/l, with 75% sensitivity,92%specificity, 88.2%positive predictive value, 82.1%negative predictive value and 84.4%accuracy (Table2,figure1) According to Pairwise comparison of ROC curve in group I there was a a statistically significant correlation between CK MB and Copeptin (P value = 0.0342), there was a a statistically significant correlation between Hs-cTn I and Copeptin (P value = 0.0212). (Table 3). According to Spearman's correlation between copeptin (pmol/L) and other variables in group I there was a significant correlation between Copeptin and age, EF%, WMSI, CK-Total, CK-MB and hs-cTnI (Table 4). MACCE occurred in eleven patients during a follow-up period of three months. MACCE was occurred in patients with a higher copeptin values. The Prognostic performance characteristics of the serum copeptin level following Receiver Operating Characteristics (ROC) curve analysis has proved statistically

significant performance in prediction of MACCE in AMI patients. The ROC curve analysis revealed that the serum copeptin level value of ≥21.1 pmol/L could predict MACCE in AMI group with AUC 0.88 suggesting strong accuracy (P< 0.001) with sensitivity 74.5% and specificity 94.24%. the ROC curve analysis of the combined serum copeptin level value of ≥21.1 pmol/L and serum Hs-cTI level value ≥1.1ng/L could predict MACCE in AMI group with AUC 0.915 suggesting strong accuracy (P< 0.001) with sensitivity 90.5% and specificity 82% (table 6)(figure4). The adverse events during the three months follow up period post primary PCI in group I AMI was the following: the stent thrombosis and re-infarction incidences were 5% of patients in group I , while acute heart failure incidence was 10 % of the patients and the death rate was 5 % of the patients in group I post AMI intervention (table s1).The Infarct related artery was LAD (41.66%) of patients with MACCE Vs (43.13%) without MACCE, while it was RCA (16.6%) of patients with MACCE Vs (23.52%) without MACCE and it was LCX (29.6%)of patients with MACCE Vs (27.45%)without MACCE. According to the initial TIMI flow grading vs final TIMI flow 3 there was significant difference between patients with MACCE Vs without MACCE (P<0.03) (Table [s2]).

Table (1): The Comparison between the studied groups regarding the demographic, clinical, echocardiographic and Laboratory data

Demographic data	**Group I (AMI)**	**Group II (Other diagnoses)**	**Test**	**P-value (Sig.)**
Count	40	50		
Age (years)				
Mean ± SD	54.4 ± 7.5	51.3 ± 10.7	1.552 *	0.124(NS)
Gender				
Male	27 (67.5%)	33 (66%)	0.023 ‡	0.881(NS)
Female	13 (32.5%)	17 (34%)		
Risk factors				
HTN	17 (42.5%)	18 (36%)	0.395 ‡	0.530(NS)
DM	11 (27.5%)	13 (26%)	0.026 ‡	0.873(NS)
Smoking	24 (60%)	22 (44%)	2.277 ‡	0.131(NS)
Dyslipidemia	5 (12.5%)	8 (16%)	0.220 ‡	0.639(NS)
Family History of CAD	7 (17.5%)	7 (14%)	0.207 ‡	0.649(NS)
Duration of chest pain (min)				
Median (IQR)	180 (120 – 240)	180 (120 – 195)	-0.178 •	0.859(NS)
HR (beat/min)				
Median (IQR)	80 (75.25 – 90)	79 (75 – 85)	1.413 •	0.158(NS)
SBP (mmHg)				

Demographic data	Group I (AMI)	Group II (Other diagnoses)	Test	P-value (Sig.)
Count	40	50		
Median (IQR)	120 (110 – 130)	120 (100 – 140)	0.016 •	0.987(NS)
DBP (mmHg)				
Median (IQR)	72.5 (70 – 80)	77.5 (70 – 90)	-1.080 •	0.280(NS)
EF%				
Mean ± SD	53.3 ± 8.6	64.8 ± 8.0	-6.582 *	<0.001(HS)
WMSI				
Median (IQR)	1.25 (1.08 – 1.38)	1.0 (1.0 – 1.0)	6.407 •	<0.001(HS)
CK-total (IU/L)				
Median (IQR)	166.5 (134.25 – 203.25)	107.5 (85.75 – 151.5)	4.195 •	<0.001(HS)
CK-MB (IU/L)				
Median (IQR)	27 (23 – 55.75)	19 (14.75 – 23.25)	5.166 •	<0.001(HS)
Hs-cTn I(ng/L)				
Median (IQR)	0.31 (0.163 – 0.56)	0.006 (0.003 – 0.007)	7.681 •	<0.001(HS)
Copeptin (pmol/L)				
Median (IQR)	16 (12 – 31.6)	6.9 (4.6 – 9.7)	6.351 •	<0.001(HS)

* Independent samples of the Student's t-test. ‡ Chi-square test.• Mann Whitney U test.

p< 0.05 is significant. Sig.: significance.

Table (2): ROC curve analysis; the diagnostic performance of cardiac biomarkers for AMI

Cut-off value	SN % (95% CI)	SP % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy % (95% CI)	AUROC (95% CI)	P-value (Sig.)
CK-total ≥ 147 IU/L	70% (53.5 – 83.4)	72% (57.5 – 83.8)	66.7% (50.5 – 80.4)	75% (60.4 – 86.4)	71.1% (60.6 – 80.2)	0.758 (0.639 – 0.842)	**<0.001 (HS)**
CK-MB ≥ 24 IU/L	70% (53.5 – 83.4)	76% (61.8 – 86.9)	70% (53.5 – 83.4)	76% (61.8 – 86.9)	73.3% (63.0 – 82.1)	0.817 (0.712 – 0.887)	**<0.001 (HS)**
Hs-cTn I ≥ 0.6 ng/L	92.5% (79.6 – 98.4)	96% (86.3 – 99.5)	94.9% (82.7 – 99.4)	94.1% (83.8 – 98.8)	94.4% (87.5 – 98.2)	0.970 (0.901 – 0.991)	**<0.001 (HS)**

Cut-off value	SN % (95% CI)	SP % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy % (95% CI)	AUROC (95% CI)	P-value (Sig.)
Copeptin ≥ 12.4 pmol/L	**75% (58.8 – 87.3)**	**92% (80.8 – 97.8)**	**88.2% (72.6 – 96.7)**	**82.1% (69.6 – 91.1)**	**84.4% (75.3 – 91.2)**	**0.891 (0.804 – 0.941)**	**<0.001 (HS)**

ROC curve: Receiver Operating Characteristic curve.SN: Sensitivity.SP: Specificity.PPV: Positive Predictive Value.NPV: Negative Predictive Value.AUROC: Area Under Receiver Operating Characteristic curve.95%CI: 95% Confidence Interval.p< 0.05 is significant. Sig.: significance.

Table (3): Pairwise comparison of ROC curves

CK-MB ~ Copeptin	
Difference between areas	0.133
Standard Error	0.0627
95% Confidence Interval	0.00987 to 0.256
z statistic	2.117
Significance level	P = 0.0342
Hs-cTn ~ Copeptin	
Difference between areas	0.0793
Standard Error	0.0344
95% Confidence Interval	0.0119 to 0.147
z statistic	2.305
Significance level	P = 0.0212

Table (4): The Spearman'scorrelation between copeptin (pmol/L) and variables in AMI group

Variable	Copeptin (pmol/L)	
	r	p
Age (years)	0.249	0.018
Duration of chest pain (min)	0.182	0.085
HR (beat/min)	-0.031	0.771
SBP (mmHg)	0.007	0.950
DBP (mmHg)	-0.115	0.279
EF (%)	-0.401	<0.001
WMSI	0.444	<0.001
CK-total (IU/L)	0.317	0.002
CK-MB (IU/L)	0.402	<0.001
Hs-cTn (ng/L)	0.643	<0.001

Table (5): the serum copeptin level and the prediction of MACCE in AMI group

Prediction	Cut-off (>) pmol/L	AUC	Sensitivity	Specificity	95% CI	p value
MACCE	**21.1**	**0.885**	**74.50**	**94.24**	**0.739 to 0.968**	**< 0.001****

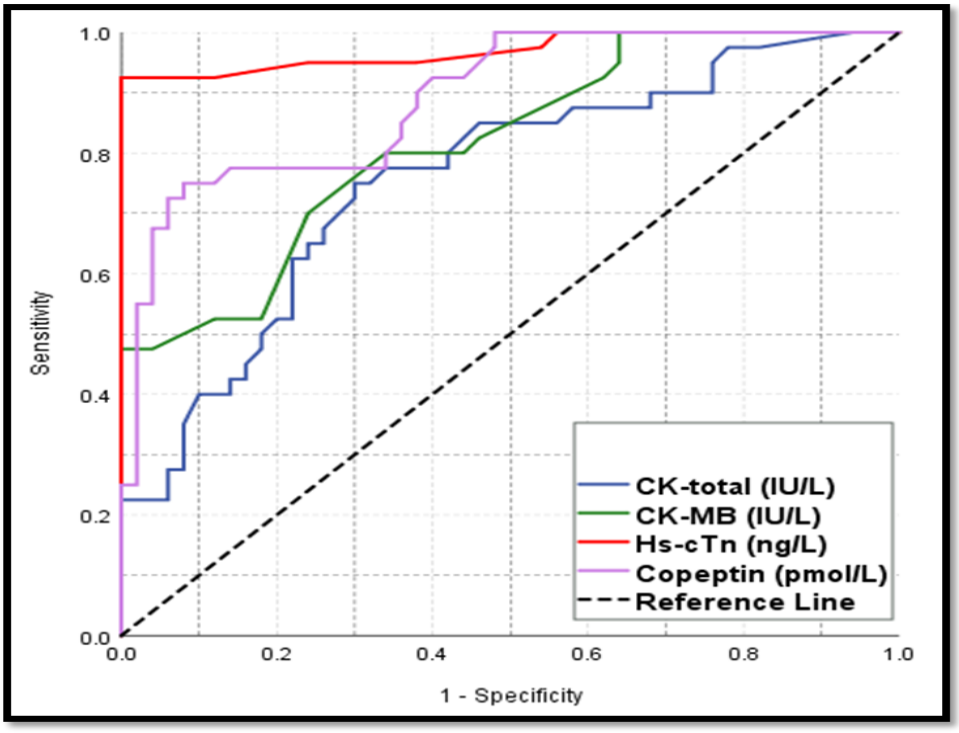
**p<0.01 is highly significant

Table (6): combined copeptin& Hs cTI and the prediction of MACCE in AMI group

Prediction	Copeptin>21.1 pmol/l and Hs cTI >1.1ng/l	AUC	Sensitivity	Specificity	95% CI	p value
MACCE		0.915	90.5	82	0.840 to 0.973	0.001**

**p<0.01 is highly significant

Figure 1: ROC curve analysis for diagnosis of AMI



Figure(2): The Spearman's correlation between copeptin (pmol/L) and Hs -cTn I

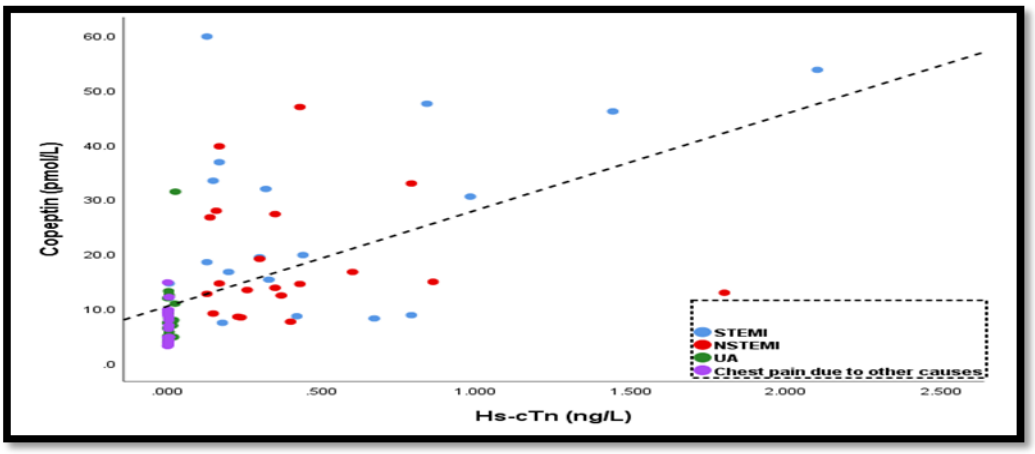


Figure (3): the ROC curve of serum copeptin and the prediction of MACCE in AMI group.

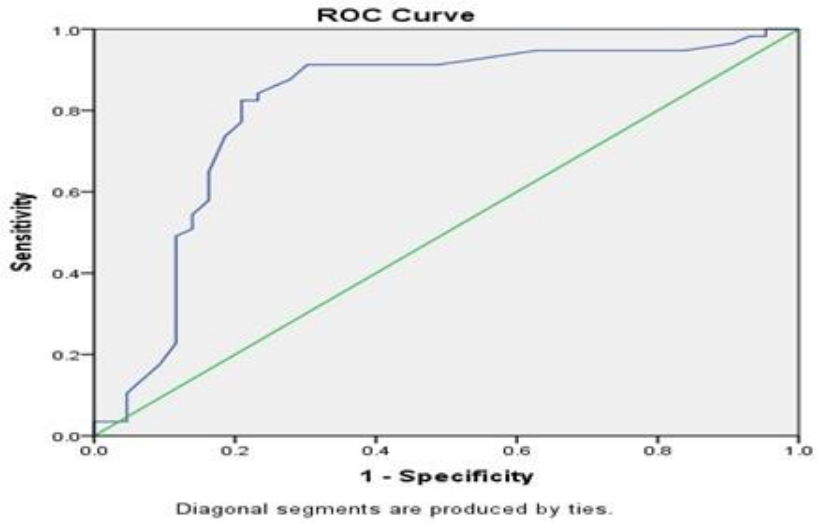
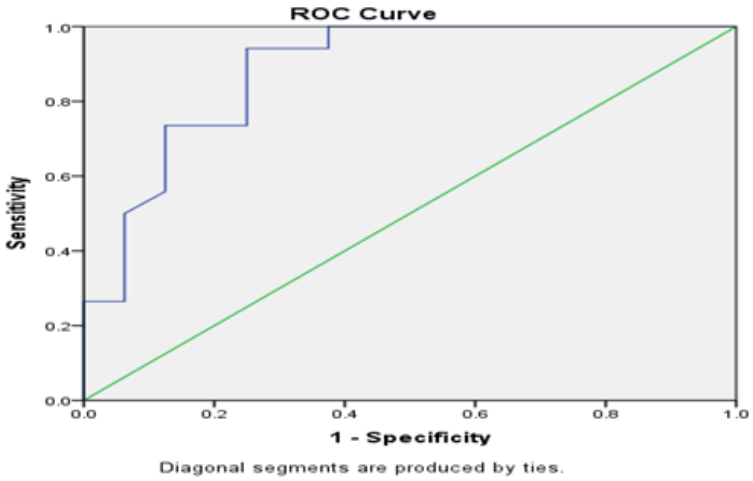


Figure (4): The ROC curve of combined of serum copeptin and serum Hs cTI and the prediction of MACCE in AMI group.



DISCUSSION

The cardiac biomarker hs cTns I assay is less sensitive in the first hour from starting the myocardial ischemia and injury a phenomenon known as 'troponins-blind time'. [21]. There were many comorbid medical cases that may affect the sensitivity and specificity of elevated hs cTns I [22,23]. The other cardiac biomarker like, serum copeptin may be elevated early in myocardial ischemia and necrosis [24], due to the stress and hemodynamic response with increased secretion of vasopressin/copeptin from the posterior pituitary gland [25]. Our results agree with Reichlin et al., as they reported significantly higher levels of serum copeptin in acute myocardial infarction patients than other diagnoses [26]. According to Eggers et al., the combination of highly sensitive cardiac troponin I with serum copeptin can add accurate diagnostic information within three hours after onset of chest pain [33]. According to Maisel et al., the availability of serum copeptin allowed 43% reduction in time for early diagnosis of acute myocardial infarction with average time 3hrs to 1.8 hrs.[27], and a falling of serum copeptin level till ten hrs. after symptoms onset. [28,29,30]. Our results are supported by findings reported by Mahmoud et al., showed that, the cutoff value of serum copeptin more than 12.6 pg/mL, revealed 92%sensitivity, 67%specificity, 74.2% positive PV and 77.8%negative PV[31]. Regarding Aborehab et al., hs cTn-I cut-off value more than 1.017 ng/l can be diagnosed 90.9% of acute myocardial infarction patients correctly but with false positive results 35.3% of normal individuals with 90.9%&64.7% sensitivity and specificity respectively .The serum copeptin cut-off values more than 30.7 pmol /l with hs cTn-I cut-off value more than 0.58ng/l can be diagnosed 100% of acute myocardial infarction patients correctly but without false positive results with 100% sensitivity and specificity[32]. Our results were in agreement with findings reported by Lotze et al., as they reported a highly significant positive correlation between serum copeptin and Hs cTnI at the admission time (P value < 0.001) [34,35,36 ,37].Also , Reinstadler et al., stated that at the time of admission the combined serum copeptin and Hs cTnI can exclude acute myocardial infarction with (98.8%& 99.7%) sensitivity and negative predictive value respectively. But serum Copeptin value alone cannot replace hs-cTnI [38].At the time of admission, despite the adding value of combined Hs cTn-I and CKMB; it was less valuable than the adding value of combined serum copeptin and Hs cTnI while at three hrs. from admission ; the cut-off value of hs cTnI > 1.017 ng/l revealed area under the curve 0.93, with (90.9%, 64.7%, 78.6% and 83.3%)sensitivity, specificity, NPV and PPV respectively , while serum copeptin cut-off value > 51.7 pmol/l revealed area under the curve 0.97, with (97%, 88.2%,93.8%&94.1%)sensitivity, specificity, NPV and PPV respectively [39].Recently, an interest has been demonstrated for copeptin in acute coronary syndrome patients. [40,41]. High copeptin levels may predict adverse cardiovascular outcomes. The serum copeptin level was increased in acute heart failure after acute STEMI [42,43]. We found that higher serum copeptin levels were in acute STEMI patients group I and was associated with increased risk of MACCE during the three months follow up. Slagman A el al., found that higher serum copeptin levels were higher in acute ST elevation MI patients who was presented to the cardiac emergency unit [44,45]. Furthermore, primary coronary intervention

can cause acute onset heart failure despite successful revascularization of infract related coronary artery and patients with a higher serum copeptin levels were associated with MACCE [46]. Our study found that higher levels of serum copeptin were an independent predictor for unfavorable outcome including stent thrombosis, re-infarction, acute heart failure and mortality. The findings of this study were in concordance with previous studies that were intended to determine the association of admission serum copeptin levels with the outcome of primary PCI treated patient. Several studies demonstrated that serum copeptin level is a useful predictor for unfavorable outcomes in patients with acute coronary syndrome [47]. The combination of serum copeptin and Hs cTnI was significantly able to predict MACCE than hs cTnI alone. However, other results from many studies have found that there was a statistically non-significant ability of serum copeptin level alone or with hs cTnI for prediction of MACCE post-acute coronary syndrome [48, 49,50]

CONCLUSION

Copeptin can early diagnose acute myocardial infarction but not superior to highly sensitive troponin. Higher serum copeptin levels were independent prognostic predictors of MACCE in acute STEMI patients treated with primary PCI during short-term follow up.

RECOMMENDATIONS

We need larger randomized interventional studies on a larger scale of the patients to find more accurate cut-off levels of serum copeptins for diagnostic and prognostic clinical impact. The use of the newest generation and the recommended techniques of (high sensitivity cTnI , CK MB, and copeptin) assays for more accurate results. Health education programs are recommended to raise awareness of acute chest pain management and early intervention of AMI. Copeptin is recommended to be routinely assessed, if applicable, for early diagnostic and prognostic cardiac biomarker in acute coronary syndrome patients.

Limitations:

The serum copeptin levels were not assessed serially in a larger number of patients from time of admission till hospital discharge and during follow up time, as serum copeptin was decreased early after symptom onset. This study was only observational, and no clinical or interventional decision was based on the serum copeptin values. The follow up period was relatively short time, so we will need a longer follow-up time to improve the power of our study finding.

No conflict of interest was reported,

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(Table s1): Adverse events for 3 months follow up

		No.	%
Stent thrombosis	**No**	**36**	**90**
	Yes	**2**	**5**
Re-infarction	**No**	**38**	**95**
	Yes	**2**	**5**
Acute HF	**No**	**36**	**90**
	Yes	**4**	**10**
Death	**No**	**38**	**95**
	Yes	**2**	**5**
Outcome	**Favorable**	**29**	**72.5**
	Unfavorable	**11**	**27.5**

Table s2:angiographic data & incidence of MACCE in AMI group post PCI

data	**Group I (AMI) with MACCE**	**Group I (AMI) without MACCE**	**Test**	**P-value (Sig.)**
Count	11	29		
Infarct related artery:				
LM	3(12.52%)	3 (5.88%)	1.2791‡	p < .05. (NS)
LAD	10(41.66%)	22(43.13%)		
LCX	7(29.16%)	14 (27.45%)		
RCA	4 (16.66%)	12 (23.52%)		
Initial TIMI flow				
0	10 (90.90.%)	15 (51.72%)	6.4433‡	.039888 (S)
≥1	1(9.1%)	14 (48.27%)		
Final TIMI 3	7 (63.63%)	29 (12.5%)		
Residual thrombi	1 (9.09%)	1 (3.44%)	0.5346‡	.464694 (NS)
Slow flow/no reflow	3 (27.27%)	3 (10.34%)	1.7924‡	.180638 (NS)
Satisfactory PCI outcome	9 (81.81%)	25 (86.20%)	0.1205‡	.72852 (NS)

‡ Chi-square test.p< 0.05 is significant. Sig.: significance.

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