Correlation of Plasma Copeptin Levels and Early Diagnosis of Acute Myocardial Infarction Compared with Troponin-T

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Background: *Patients present with chest pain. Electrocardiography (ECG) is used and troponin-T levels slowly increases. Diagnosis of myocardial infarction requires prolonged monitoring, over six to nine hours, for serial blood sampling. It is the cause of delayed treatment and lead to a crowded emergency room. Troponin is a marker of myocardial necrosis, the gold standard in detection of acute myocardial infarction (AMI). Copeptin, the C-terminal part of the vasopressin prohormone, as a marker of acute endogenous stress, adds diagnosis information to cardiac troponin in early evaluation of patients with suspected myocardial infarction.*

Objective: *To determine the correlation between plasma copeptin level and troponin-T. It is also to determine if the copeptin level can be used as early diagnosis in patients who present with chest pain and are suspected to be acute myocardial infarction (AMI).*

Material and Method: *Patients with chest pain that presented to the emergency department of Rajavithi Hospital between October 2010 and October 2011 and were suspected to have myocardial infarction were consecutively enrolled in the present study. The level of plasma copeptin and troponin-T were measured at presentation and six hours after presentation.*

Results: *One hundred fifty patients presented to the emergency department with chest pain. Their average age was 66.71±7.78 years. The mean plasma copeptin level was 13.91±5.01 pmol/l in acute myocardial infarction. Plasma copeptin level increased compared with troponin-T. Plasma copeptin level increased and correlated with troponin-T to diagnose myocardial infarction (r = 0.317) at presentation. It further increased and correlated (r = 0.562) at six hours after presentation. Plasma copeptin levels for diagnosis of ST elevate myocardial infarction (STEMI) at presentation have an area under curve (AUC) = 0.91, p<0.001, sensitivity 90.9%, and specificity 87.8%. The non-ST elevated myocardial infarction (NSTEMI) have an area under curve (AUC) = 0.71, p<0.001, sensitivity 88.8%, specificity 69.8%, and cut-off point of 10.25 pmol/l.*

Conclusion: *Plasma copeptin can be used for early diagnosis of myocardial infarction. The additional use of copeptin to Troponin-T allows for a rapid triage of chest pain patients to an early diagnosis of non-ST elevation myocardial infarction.*

Keywords: *Copeptin, Troponin-T, Myocardial infarction*

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Myocardial infarction is a major cause of death worldwide. Its incidence is increasing. The emergency department of Rajavithi Hospital received 585 patients for myocardial infarction in 2008 and the incidence has increased every year since. Rapid assessment of these patients is critical to direct further diagnosis and therapeutic strategies. The gold standard in diagnosis of acute myocardial infarction (AMI) are by using Clinical symptoms, electrocardiography (ECG), markers of myocardial necrosis such as cardiac troponin and creatine kinase, and myocardial band (CK-MB). In particular, cardiac troponin provides excellent specificity. After cell disintegration, the delayed release of necrosis markers might explain the weakness in the diagnostic performance of conventional troponin assays early after chest pain onset[1,2].

In some patients electrocardiography (ECG) does not significantly change. Furthermore, patients come four to six hours earlier, therefore, the Troponin level does not increase. In those cases, diagnosis of myocardial infarction requires a long monitoring, over four to six hours to allow for serial blood sampling[3,4]. Copeptin is the C-terminal part of vasopressin and it is secreted stoichiometrically with arginine-vasopressin. Copeptin is Pro-hormone, a marker of acute endogenous stress and it may be used for early diagnosis of myocardial infarction[5,6,8]. In a recent study, copeptin was markedly elevated in patients after acute myocardial infarction[9-11]. However, no study is known about the diagnostic value of

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copeptin in acute myocardial infarction in Thai patients[9].

Troponin is a marker of cardiac necrosis[6], either in combination with a pathophysiologically or as a different biomarker, reflecting acute endogenous stress such as copeptin. The objective of this study is to analyze if copeptin might allowed for a rapid and accurate diagnosis of acute myocardial infarction at initial presentation without serial blood sampling.

Material and Method

The present study was a cross-sectional descriptive analysis designed to determine the correlation of plasma copeptin level and early diagnosis of myocardial infarction as compared with troponin-T. The present study was approved by the ethics committee of Rajavithi Hospital. Between October 2010 and October 2011, patients with chest pain presenting to the emergency department of Rajavithi Hospital suspected acute myocardial infarction were consecutively enrolled in the present study. All patients underwent a physical examination, a 12-lead electrocardiography (ECG), a continuous ECG monitoring, standard blood test, and measured levels of plasma copeptin and troponin-T at presentation and at six hours after presentation. Results are shown in Fig. 1. Troponin-T was determined using a 1-step enzyme immunoassay, which is based on electrochemiluminescence immuno-assay technology with detection limit of 0.01 ug/ml. The reference limit was based on the ninety-ninth percentile for a healthy population, which is 0.01 ug/ml.

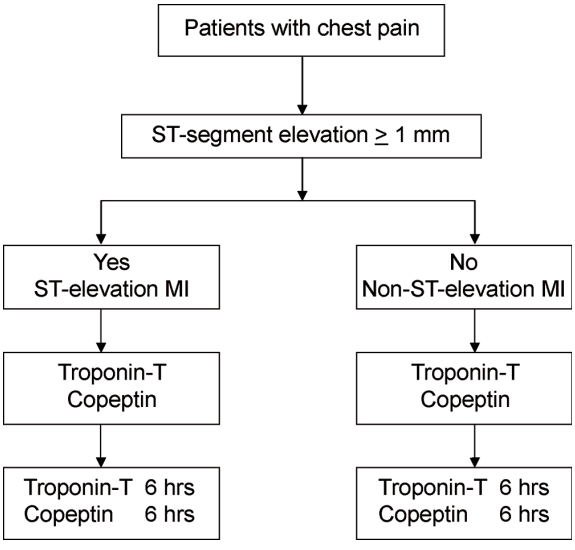


Fig. 1 Flow chart patients come with chest pain.

Blood samples for determination of copeptin were collected at presentation and at six hours after presentation. It was done by drawing blood into tubes containing potassium ethylenediaminetetra acetic acid. After centrifugation, samples were frozen at -80°C until assayed in a blinded fashion in a single batch metric assay B.R.A.H.M.S LUMI test CT-proAVP, B.R.A.H.M.S AG, Hennigsdorf Berlin, Germany. The capture antibody was replaced by a murine monoclonal antibody directed to amino acids 137-144 (GPAGAL) of proAVP. This modification improved the sensitivity of the assay. The assay has an analytical detection limit of 0.4 pmol/l and a functional assay sensitivity (lowest valve with an interassay coefficient of variation <20%) <1.0 pmol/l. It allows precise measurement of copeptin in a range of 0.4 to 1,250 pmol/l. The median copeptin level in 200 healthy persons was 3.7 pmol/l and the 97.5 percentile was 16.4 pmol/l. Acute myocardial infarction was diagnosed when there was evidence of myocardial necrosis in a clinical setting consistent with myocardial ischemia. Necrosis was diagnosed by a rising and/or falling pattern of troponin-T with at least one value at the 99th percentile, with an imprecision of <10%[12]. For the troponin-T assay, the lower limit of detection is 0.01 ug/l. Thus, to manifest a rising pattern, patients with normal initial values had to increase troponin-T levels to the cutoff level of ≥0.04 ug/l to fulfill acute myocardial infarction criteria. Unstable angina was diagnosed in patients with normal troponin-T level and typical angina pain at rest. The patients were excluded if they had clinical septicemia, surgery within three months, or did heavy exercise.

Statistical analysis

Continuous variables are presented as mean±SD or median and categorical variables are presented as numbers and percent. Continuous variables are compared with the Student t-test or Mann-Whitney U test and categorical variables are calculated by using the Pearson Chi-square test or Fisher's exact test. Receiver-operator characteristic (ROC) curves were constructed to assess the sensitivity and specificity throughout the concentrations of troponin-T level and copeptin level in diagnosing acute myocardial infarction. Statistical analysis was performed with SPSS software, version 17.0. Statistical significance was set at $p<0.05$.

Results

One hundred fifty patients were included in the present study. Their mean age was 66.71±7.78 years.

Eleven patients had ST-segment elevation myocardial infarction and 139 patients had non-ST segment elevation myocardial infarction. The mean plasma copeptin level was 13.33±4.58 pmol/l at presentation and 10.39±3.50 pmol/l at six hours after presentation. Other baseline characteristics are shown in Table 1.

Table 1. Baseline characteristics

Characteristics	MI		Total (n = 150)	p-value
	NSTEMI (n = 139)	STEMI (n = 11)		
Age				0.027*
Mean±SD	67.10±7.72	61.73±6.96	66.71±7.78	
Min-max	53-89	53-74	53-89	
Gender				1.000
Male	112 (80.6%)	9 (81.8%)	121 (80.7%)	
Female	27 (19.4%)	2 (18.2%)	29 (19.3%)	
Nutrition status				0.371
Over weight	70 (50.4%)	4 (36.4%)	74 (49.3%)	
Obesity	69 (49.6%)	7 (63.6%)	76 (50.7%)	
Underlying disease	110 (79.7%)	4 (36.4%)	114 (76.5%)	0.004*
Coronary disease	32 (23.0%)	1 (9.1%)	33 (22.0%)	0.457
Hypertension	87 (62.6%)	3 (27.3%)	90 (60.0%)	0.027*
Diabetes mellitus	84 (60.4%)	2 (18.2%)	86 (57.7%)	0.009*
Dyslipidemia	87 (62.6%)	1 (9.1%)	88 (58.7%)	0.001*
Chronic kidney disease	25 (18.0%)	2 (18.2%)	27 (18.0%)	1.000
Smoking	47 (33.8%)	9 (81.8%)	56 (37.3%)	0.002*
Drinking	33 (23.7%)	7 (63.6%)	40 (26.7%)	0.008*
ST elevation ECG	1 (0.7%)	10 (90.9%)	11 (7.3%)	<0.001*
ST depression ECG	92 (66.2%)	0 (0.0%)	92 (61.3%)	<0.001*
T-inversion ECG	16 (11.5%)	0 (0.0%)	16 (10.7%)	0.608
Left bundle branch block ECG	0 (0.0%)	3 (27.3%)	3 (2.0%)	<0.001*
Non-specific ECG	29 (20.9%)	0 (0.0%)	29 (19.3%)	0.124
Angiotensin converting enzyme inhibitor (ACEI)	73 (52.5%)	2 (18.2%)	75 (50.0%)	0.028*
Angiotensin II receptor blockers (ARB)	12 (8.6%)	0 (0.0%)	12 (8.0%)	0.601
Betablocker	48 (34.5%)	0 (0.0%)	48 (32.0%)	0.017*
Calcium channel blocker	65 (46.8%)	3 (27.3%)	68 (45.3%)	0.346
Diuretic	77 (55.4%)	2 (18.2%)	79 (52.7%)	0.017*
Oral hypoglycemic	80 (57.6%)	2 (18.2%)	82 (54.7%)	0.023*
Aspirin	37 (26.6%)	1 (9.1%)	38 (25.3%)	0.291
Nitrate	34 (24.5%)	1 (9.1%)	35 (23.3%)	0.459
Statin	78 (56.1%)	1 (9.1%)	79 (52.7%)	0.003*
Systolic blood pressure	139.72±12.04	135.00±15.65	139.37±12.34	0.223
Diastolic blood pressure	79.62±8.91	76.18±10.03	79.37±9.01	0.224
Heart rate	66.47±4.22	65.91±5.80	66.43±4.34	0.679
Troponin-T at presentation	0.00±0.02	0.41±0.20	0.03±0.12	<0.001*
Copeptin at 6 hr	13.33±4.58	21.25±4.45	13.91±5.01	<0.001*
Troponin-T at presentation	0.28±0.19	3.85±3.26	14.34±6.82	<0.001*
Copeptin at 6 hr	10.39±3.50	17.80±3.90	10.93±4.01	<0.001*
Blood sugar	143.45±39.10	139.09±56.36	143.13±40.38	0.731
Cholesterol	241.31±26.14	275.27±20.38	243.80±27.20	<0.001*
Low-density lipoprotein (LDL)	146.91±12.75	165.82±11.60	148.30±13.56	<0.001*
Triglyceride (TG)	211.40±24.91	229.73±23.02	212.74±25.17	0.020*
High density lipoprotein(HDL)	52.76±6.58	46.36±10.40	52.29±7.09	0.004*

ECG = electrocardiography

Patients with an underlying disease may be included in the ST-segment elevation myocardial infarction (p = 0.004). Underlying disease are hypertension, diabetes mellitus, and hyperlipidemia (LDL) p<0.001. Furthermore, it include patients who smoke (p = 0.007) or drink (p = 0.009). All increase incident of ST elevated myocardial infarction (STEMI) are shown in Table 2. At presentation, the correlation of copeptin level and troponin-T level have less correlation r = 0.317. This is because copeptin level increases more rapidly than troponin-T level in myocardial infarction. troponin-T release is delayed until myocardial necrosis, which is about four to six hours. After six hours of presentation, copeptin level and troponin-T level continuously increase. Therefore, there is more correlation between copeptin and troponin-T (r = 0.562) as shown in Fig. 2 and 3. The ROC curve was analyzed to identify the best cut-off point of copeptin levels to be used as a predictor of ST elevation myocardial infarction (STEMI). The copeptin level of 8.65 pmol/l gives the best sensitivity (90.9%) and specificity (87.8%). The troponin-T, at a cut-off point of 0.16 ng/ml, gives sensitivity of 90.9% and specificity of 100% as shown in Fig. 4 and Table 3. Copeptin levels in non-ST elevation myocardial infarction (NSTEMI) at presentation in emergency room has an area under curve (AUC) 0.71, p<0.001, cut-off point of 10.25 pmol/l, sensitivity 88.8%, and specificity 69.8%. In comparison, troponin-T at presentation has area under curve (AUC) 0.394, cut-off point 0.05 ug/ml, sensitivity 2.8%, and specificity 76.7%.

The comparison detail show the group of non-ST elevation myocardial infarction (NSTEMI) troponin-T level increased faster than copeptin level as shown in Fig. 5 and Table 4. Copeptin level was significantly higher than troponin-T level in both non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI) at presentation in the emergency room.

Table 2. Multivariate analysis of risk factors of STEMI

Characteristics	OR (95%CI)	p-value
Age	0.89 (0.81-0.99)	0.032*
Underlying disease	0.15 (0.04-0.53)	0.004*
Smoking	8.81 (1.83-42.42)	0.007*
Drinking	5.62 (1.55-20.40)	0.009*
Troponin-T	453.33 (43.13-4,765.44)	<0.001*
Copeptin	2.09 (1.46-3.01)	<0.001*
LDL	1.11 (1.05-1.17)	<0.001*

LDL = low-density lipoprotein; STEMI = ST elevation myocardial infarction; OR = odds ratio

Discussion

This is the first study to assess acute myocardial infarction by using copeptin level with Thai patients. The present study used a cross-sectional descriptive study by selecting patients presenting in the emergency department of Rajavithi Hospital with chest pain symptom that were suggestive of acute myocardial infarction.

The gold standard diagnosis of myocardial infarction is troponin-T, a marker of cardiac necrosis. However, the diagnostic has a low sensitivity of 43% in detecting acute myocardial in patient presenting within four hours. It compared with new biomarker

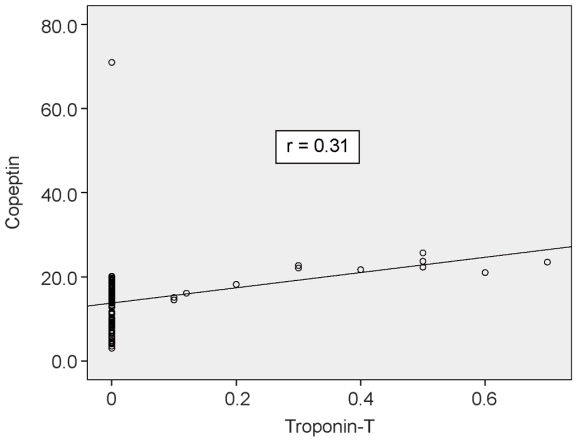


Fig. 2 Correlation of copeptin levels and troponin-T levels at presentation.

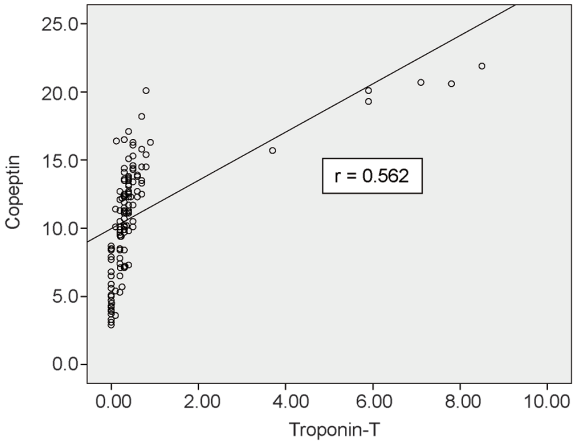


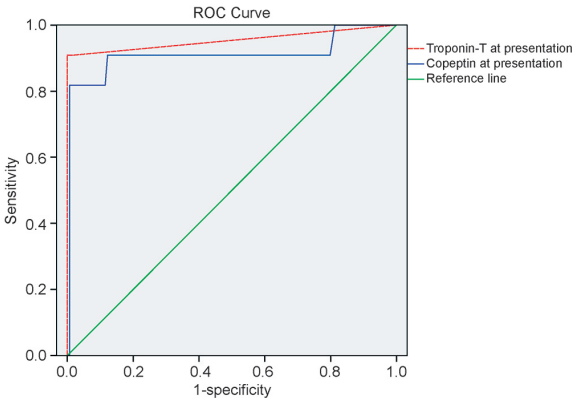
Fig. 3 Correlation of copeptin levels and troponin-T levels at 6 hours after presentation.

Table 3. STEMI at presentation

	AUC (SE)	p-value	Cut off point	Sensitivity	Specificity
Troponin-T	0.955 (0.052)	<0.001*	0.16	90.9	100.0
Copeptin	0.910 (0.069)	<0.001*	8.65	90.9	87.8

Table 4. NSTEMI at presentation

	AUC (SE)	p-value	Cut off point	Sensitivity	Specificity
Troponin-T	0.394 (0.055)	<0.044*	0.05	2.8	76.7
Copeptin	0.710 (0.066)	<0.001*	10.25	88.8	69.8



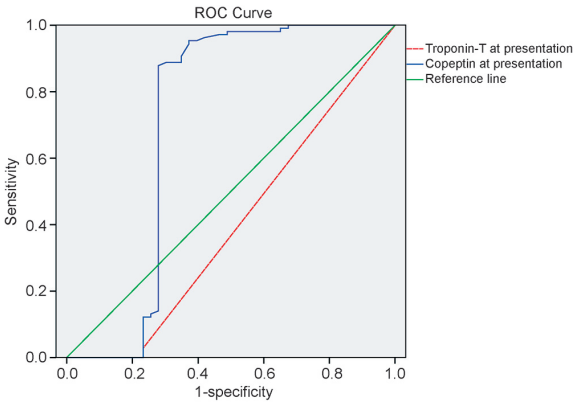


Fig. 4 ROC curve of copeptin levels and troponin-T levels at presentation.

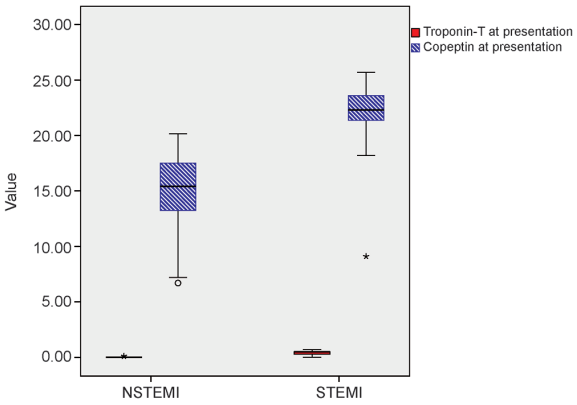


Fig. 5 Coparison between copeptin levels and troponin-T at presentation.

(a part of vasopressin secreted stoichiometrically with arginine vasopressin). Copeptin is a marker of endogenous stress that increase rapidly. It can be the fast rule out of acute myocardial infarction.

The present study reported three major findings. First, copeptin level was significantly higher and increased earlier in patients with chest pain with acute myocardial infarction than troponin-T.

Second, copeptin level was significantly higher in patients with non-ST elevation myocardial infarction (NSTEMI) presenting early to the emergency department and still negative for troponin-T. Sensitivity of troponin-T at presentation of chest pain in emergency room was very low at 2.8% compared with copeptin at 88.8%. Some studies added copeptin to troponin to provide a stable diagnostic performance to provide a better result regardless of time of presentation after chest pain. This is because of the early peak of copeptin with the following decrease, and the slow increase of troponin-T levels[12].

Third, the combination of troponin-T and copeptin resulted in a better diagnosis accuracy of acute myocardial infarction at presentation. While troponin-T is low sensitivity, delayed increase, it has high specificity and stayed for a long time after increase as compare with copeptin. On the other hand, copeptin has a high sensitivity and a rapid increase. When both biomarkers are used, they can increase accuracy and early diagnosis of acute myocardial infarction[13].

As previously reported, copeptin can help rule-out acute myocardial infarction early.

This finding shows the incremental value of copeptin for rapid rule-out of acute myocardial

infarction. Furthermore, copeptin can improve early diagnosis of acute myocardial infarction.

These findings have important clinical implications to exclude acute myocardial infarction in patients presenting with chest pain. Without this, physicians have to do serial blood sampling and wait for the increase in troponin-T, which can take up to six hours. Furthermore, the normal or non-specific ECG finding requires longer monitoring[4]. Therefore, this finding can help in providing treatment earlier, reduce patients waiting in the emergency room, and reduce crowding in Emergency room. Some data suggested that acute myocardial infarction induces a higher level of endogenous stress than unstable angina does. This is related, at least in part, to the more prolonged course of chest pain in patients with acute myocardial infarction[15]. The combination of copeptin and troponin-T can be useful in rapid evaluation of chest pain of patients in the emergency room.

Study limitations

The small sample size and need for a long-time follow-up.

Copeptin is not known to be influenced by delayed measurement, prolonged storage, or repeated freeze-thaw cycles.

Conclusion

Plasma copeptin can be used for early diagnosis myocardial infarction. The additional use of copeptin to troponin-T allows a rapid triage of chest pain patients to early diagnosis non-ST elevation myocardial infarction.

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Potential conflicts of interest

None.

References

1. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. J Am Coll Cardiol 2007; 50: 2173-95.
2. Apple FS, Wu AH, Jaffe AS. European Society of Cardiology and American College of Cardiology guidelines for redefinition of myocardial infarction: how to use existing assays clinically and for clinical trials. Am Heart J 2002; 144: 981-6.
3. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined—a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000; 36: 959-69.
4. Pope JH, Aufderheide TP, Ruthazer R, Woolard RH, Feldman JA, Beshansky JR, et al. Missed diagnoses of acute cardiac ischemia in the emergency department. N Engl J Med 2000; 342: 1163-70.
5. Reichlin T, Hochholzer W, Bassetti S, Steuer S, Stelzig C, Hartwiger S, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. N Engl J Med 2009; 361: 858-67.
6. Melanson SE, Morrow DA, Jarolim P. Earlier detection of myocardial injury in a preliminary evaluation using a new troponin I assay with improved sensitivity. Am J Clin Pathol 2007; 128: 282-6.
7. Itoi K, Jiang YQ, Iwasaki Y, Watson SJ. Regulatory mechanisms of corticotropin-releasing hormone and vasopressin gene expression in the hypothalamus. J Neuroendocrinol 2004; 16: 348-55.
8. Robertson GL, Mahr EA, Athar S, Sinha T. Development and clinical application of a new method for the radioimmunoassay of arginine vasopressin in human plasma. J Clin Invest 1973; 52: 2340-52.
9. Preibisz JJ, Sealey JE, Laragh JH, Cody RJ, Weksler BB. Plasma and platelet vasopressin in essential hypertension and congestive heart failure. Hypertension 1983; 5: I129-38.
10. Morgenthaler NG, Struck J, Alonso C, Bergmann A. Assay for the measurement of copeptin, a stable peptide derived from the precursor of vasopressin. Clin Chem 2006; 52: 112-9.
11. Khan SQ, Dhillon OS, O'Brien RJ, Struck J, Quinn PA, Morgenthaler NG, et al. C-terminal provasopressin (copeptin) as a novel and prognostic marker in acute myocardial infarction: Leicester Acute Myocardial Infarction Peptide (LAMP) study. Circulation 2007; 115: 2103-10.
12. Apple FS, Jesse RL, Newby LK, Wu AH, Christenson RH. National Academy of Clinical Biochemistry and IFCC Committee for Standardization of Markers of Cardiac Damage Laboratory Medicine Practice Guidelines: Analytical issues for biochemical markers of acute coronary syndromes. Circulation 2007; 115:

e352-5.
13. Reichlin T, Hochholzer W, Stelzig C, Laule K, Freidank H, Morgenthaler NG, et al. Incremental value of copeptin for rapid rule out of acute myocardial infarction. J Am Coll Cardiol 2009; 54: 60-8.
14. Keller T, Tzikas S, Zeller T, Czyz E, Lillpopp L, Ojeda FM, et al. Copeptin improves early diagnosis of acute myocardial infarction. J Am Coll Cardiol 2010; 55: 2096-106.
15. Keller T, Zeller T, Peetz D, Tzikas S, Roth A, Czyz E, et al. Sensitive troponin I assay in early diagnosis of acute myocardial infarction. N Engl J Med 2009; 361: 868-77.

ศึกษาความสัมพันธ์ของระดับพลาสมาโคเป็ปตินกับความรวดเร็วในการวินิจฉัยโรคหัวใจขาดเลือดเทียบกับโทโปนินที

สุมิตษ์ตรา ปียะณัตดิ์พูล

ภูมิหลัง: *ผู้ป่วยมาด้วยอาการแน่นหน้าอกบางครั้งคลื่นไฟฟ้าหัวใจแสดงไม่ชัดเจนและค่าโทโปนินทียังไม่ขึ้น ทำให้ต้องเฝ้าติดตามอาการผู้ป่วยนาน 6 ถึง 9 ชั่วโมง เพื่อติดตามการเจาะเลือดทำให้การรักษาล่าช้า และผู้ป่วยรอคอยในห้องฉุกเฉินมาก*

วัตถุประสงค์: *เพื่อศึกษาความสัมพันธ์ระหว่างระดับพลาสมาโคเป็ปตินเทียบกับโทโปนินทีในการวินิจฉัยผู้ป่วยโรคหัวใจขาดเลือดที่มาด้วยอาการแน่นหน้าอกได้อย่างรวดเร็ว*

วัสดุและวิธีการ: *ศึกษาผู้ป่วยที่มาห้องฉุกเฉินโรงพยาบาลราชวิถีด้วยอาการแน่นหน้าอกตั้งแต่เดือนตุลาคม พ.ศ. 2553 ถึง ตุลาคม พ.ศ. 2554 ที่สงสัยว่าเป็นโรคหัวใจขาดเลือด และทำการเจาะเลือดเพื่อดูระดับของพลาสมาโคเป็ปตินเทียบกับระดับโทโปนินที่เวลาผู้ป่วยมาถึงโรงพยาบาล และอีก 6 ชั่วโมงต่อมา*

ผลการศึกษา: *ผู้ป่วยทั้งหมด 150 ราย อายุเฉลี่ย 66.7±7.78 ปี ระดับพลาสมาโคเป็ปตินที่เป็นโรคหลอดเลือดหัวใจขาดเลือดอยู่ที่ 13.91±5.01 พิโคโมลต่อลิตร ระดับพลาสมาโคเป็ปตินมีความสัมพันธ์เชิงบวกกับคาโทโปนินที $r = 0.317$ และมีความสัมพันธ์มากขึ้นที่ 6 ชั่วโมง $r = 0.562$ พลาสมาโคเป็ปตินสามารถใช้ในการช่วยวินิจฉัยโรคกล้ามเนื้อหัวใจขาดเลือดได้ $AUC = 0.71$ และ $p<0.001$ ด้วยค่าความน่าเชื่อถือที่ 88.8 เปอร์เซ็นต์*

สรุป: *พลาสมาโคเป็ปตินสามารถใช้ในการช่วยวินิจฉัยโรคกล้ามเนื้อหัวใจขาดเลือดได้อย่างรวดเร็ว และสามารถใช้เสริมร่วมกับค่าโทโปนินทีในการวินิจฉัยโรคหัวใจขาดเลือดได้รวดเร็วยิ่งขึ้น*
