Diagnostic Level of Cardiac Troponin T in Patients with Chronic Renal Dysfunction, A Pilot Study

Apichard Sukonthasarn MD*,
Sumeth Ponglopisit MD*

** Department of Medicine, Faculty of Medicine, Chiang Mai University, Chiang Mai*

Objective: *To find the diagnostic level and rising pattern of cardiac troponin T (cTnT) in patients with chronic renal dysfunction presented with acute myocardial infarction (AMI).*
Material and Method: *A pilot, cross-sectional study to compare the level of cTnT in adult patients with chronic renal dysfunction who were admitted and later confirmed to have AMI with those in the age and sex-matched controls with chronic renal dysfunction and non-coronary diagnosis.*
Results: *Twenty-three patients were enrolled into each group. The mean cTnT levels in the AMI group were significantly higher than in the control group. Magnitude and rate of post-admission rise of cTnT were not significantly different between both groups. The diagnostic level of cTnT for AMI was 0.1 nanogram per milliliter with 90.90% sensitivity and 84.50% specificity. The sensitivity and specificity of this diagnostic level were 91.30% and 100.00% respectively when patients with chronic renal replacement were excluded.*
Conclusion: *The level of cTnT of at least 0.1 nanogram per milliliter within the first 24 hours of admission was diagnostic for AMI in patients with chronic renal dysfunction. The sensitivity and specificity of the tests were better if the patients with chronic renal replacement were excluded.*

Keywords: *Cardiac troponin T, Chronic renal dysfunction*

J Med Assoc Thai 2007; 90 (9): 1749-58
Full text. e-Journal: *http://www.medassocthai.org/journal*

Acute myocardial infarction (AMI) is a medical emergency that requires early diagnosis and treatment. While a variety of treatment options such as heparin, thrombolytic therapy, and percutaneous coronary intervention are available and can reduce mortality and morbidity in patients with AMI, the effectiveness of these therapies diminishes rapidly within the first several hours after symptoms onset[1,2]. The traditional evaluation of these patients relies mostly on the history, physical examination, and the electrocardiography (ECG). This approach sometimes fails to identify patients suffering from AMI and these patients can lose their opportunity to be properly treated within the appropriate time and can be at relatively high risk of future complications or death.

Correspondence to : Ponglopisit S, Division of Cardiovascular, Department of Medicine, Faculty of Medicine, Chiang Mai University, 110 Intrawarorose Rd, Sripoom, Chiang Mai 50200, Thailand. Phone: 053-945-486, Fax: 053-214-352, E-mail: sumethponglopisit@yahoo.com

Serum cardiac biomarkers, such as creatine kinase (CK), CK-MB, cardiac-specific troponins, and myoglobin, are helpful for confirming the diagnosis of AMI. Although initiation of reperfusion therapy in ST segment elevation acute coronary syndrome (STE-ACS) should not be delayed while awaiting the results of a cardiac biomarker assay, they are useful for estimating infarct size and prognosis[3]. Even marginal elevation of troponin, an accurate marker of cardiac necrosis, correlates with worse clinical outcomes in non-ST segment elevation acute coronary syndrome (NSTE-ACS)[4].

Although cardiac troponin T (cTnT) has a nearly absolute myocardial tissue specificity and high sensitivity, its level can be falsely elevated in patients with renal dysfunction[5-8], which can partly be explained by impairment of its renal clearance[7]. Many evidences demonstrated that serum or plasma cTnT was an important predictor of long-term cardiovascular mortality in patients with end-stage renal disease (ESRD)[9-11].

The diagnostic level for acute myocardial infarction in patients with chronic renal dysfunction is still needed to guide the proper management.

The authors' objectives were to determine the diagnostic level and rising pattern of cTnT, which can be used to diagnose AMI in patients with renal dysfunction.

Material and Method

Study design

The present study was a pilot, cross-sectional study to compare the level of cTnT in adult patients with chronic renal dysfunction presented with symptoms compatible with acute coronary syndrome and later confirmed to have AMI with the level of cTnT in the age and sex-matched controls with chronic renal dysfunction who were admitted with non-coronary diagnosis. The present study was done between September 2005 and January 2006 with the approval of the institutional review board and ethic committee. All patients gave written informed consent.

Subjects

All admitted patients with an established diagnosis of chronic renal dysfunction for at least 3 months, defined as creatinine clearance (CrCl) less than 60 milliliters per minute per body surface area of 1.73 square meters as determined by Cockcroft and Gault equation[12] were considered for recruitment. Stages of chronic renal dysfunction were classified by K/DOQI guideline[13]. Diagnosis of AMI was based on the consensus document of the Joint European Society of Cardiology and American College of Cardiology committee for the Redefinition of Myocardial Infarction published in the year 2000[14]. In this consensus the clinical criteria, which satisfies the diagnosis for an acute, evolving, or recent myocardial infarction is typical rise and gradual fall (troponins) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following: a) Ischemic symptoms b) Development of pathological Q waves on ECG readings c) ECG changes indicative of ischemia (ST-segment elevation or depression) d) Coronary artery intervention (e.g., coronary angioplasty). Subjects who presented with symptoms compatible with ACS and who met these diagnostic criteria were placed in the acute myocardial infarction group, AMI group. Age and sex matched subjects who were admitted with non-coronary causes were placed in the control group. Patients with ACS symptoms who did not meet the diagnostic criteria for AMI were excluded.

The authors also excluded patients with pulmonary embolism, muscle diseases, acute stroke, renal dysfunction of duration less than 3 months, recent ACS other than this admission, history of recent heavy exercise, muscle trauma, or those being treated by electrical cardioversion.

The reasons for admission in the control group were anemia from chronic renal dysfunction (3 cases), renal transplantation preparation (1 case), gastrointestinal bleeding (2 cases), fever (2 cases), drug allergy (1 case), pneumonia (2 cases), volume overload (7 cases), hyponatremia (1 case), hematological malignancy (1 case), syncope (2 cases), and seizure (1 case).

All patients were examined by investigators and were optimally investigated and treated for their cardiac and non-cardiac illnesses. The cTnT levels were determined by the third generation Elecsys Troponin T STAT test of Roche Diagnostics at the time of admission, after informed consents were given, and at 6, 12, and 24 hours later.

Statistical analysis

Descriptive statistic with mean, median, range, and standard deviation (SD) are provided. The authors compared cTnT levels at each specified time in the AMI group with the corresponding levels in control group, the authors also compared the magnitude of post-admission rise (differences between cTnT level at admission and at any specific time) and the rate of post-admission rise (nanogram per milliliter per hour) between the two groups by unpaired t-test and Chi-square test. All statistics were calculated by using SPSS Version 12.0. A p value of ≤ 0.05 was considered statistically significant.

Results

Fifty-three patients were enrolled in the present study. Three patients with history of recent myocardial infarction from previous admissions and four patients who did not fulfill the diagnostic criteria for AMI were excluded from the AMI group. There were twenty-three patients in each group as in Fig. 1. Table 1 describes the baseline characteristics, underlying diseases, and renal function. Both groups were similar in all clinical parameters except mean creatinine levels, which were higher in the AMI group (2.23 ± 1.26 milligram per deciliter vs. 1.51 ± 0.68 milligram per deciliter, $p = 0.047$). The number of patients in each stage of renal dysfunction was not different between the AMI and the control group. There was no difference in underlying diseases and prevalence of coronary risk

Table 1. Characteristics of patients

Characteristics	Group of patients		p-value
	AMI group (n = 23)	Control group (n = 23)	
Age			
Mean ± SD, years	71.74 ± 8.67	65.65 ± 15.70	0.111
Range, years	47-83	33-95	
Median, years	72	70	
Sex			
Male	8 (34.8%)	8 (34.8%)	1.000
Female	15 (65.2%)	15 (65.2%)	1.000
Diagnosis and clinical parameters			
NSTEMI	15 (65.2%)	0 (0%)	-
STEMI	8 (34.8%)	0 (0%)	-
DM	6 (26.1%)	3 (13.0%)	0.459
HT	11 (47.8%)	10 (43.5%)	1.000
Dyslipidemia	4 (17.4%)	4 (17.4%)	1.000
Previous stroke	1 (4.3%)	1 (4.3%)	1.000
CAD	5 (21.7%)	5 (21.7%)	1.000
PVD	0 (0%)	1 (4.3%)	1.000
COPD	4 (17.4%)	1 (4.3%)	0.346
Liver disease	0 (0%)	2 (8.7%)	0.489
Chronic smoking	8 (34.8%)	8 (34.8%)	1.000
Stages of renal dysfunction			
Stage 3 (CrCl 30-59)	5 (21.7%)	8 (34.8%)	0.514
Stage 4 (CrCl 15-29)	11 (47.8%)	8 (34.8%)	0.550
Stage 5 (CrCl <15)	7 (30.4%)	7 (30.4%)	1.000
Creatinine level*			
Mean ± SD, mg/dL	2.23 ± 1.26	1.51 ± 0.68	0.047
Range, mg/dL	0.7-4.4	0.8-3.2	
Median, mg/dL	1.80	1.30	
Chronic hemodialysis	2 (8.7%)	7 (30.4%)	0.135

* Patients with chronic renal replacement were excluded

AMI: acute myocardial infarction, SD: standard deviation, NSTEMI: non-ST segment elevation myocardial infarction, STEMI: ST segment elevation myocardial infarction, DM: diabetes mellitus, HT: hypertension, CAD: coronary artery disease, PVD: peripheral vascular disease, COPD: chronic obstructive pulmonary disease, CrCl: creatinine clearance (millilitre per minute per body surface area 1.73 square metre), mg/dL: milligram per decilitre

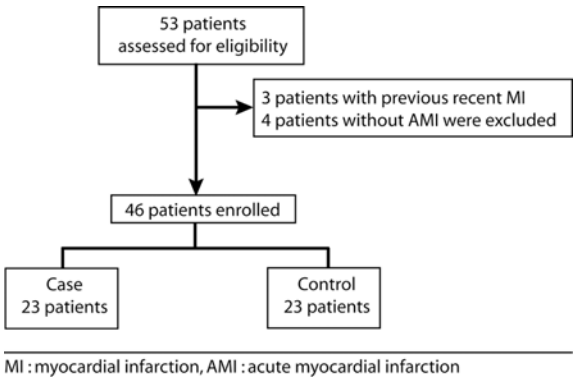

Fig. 1 Enrollment of patients

factors between the study groups. Mean time after onset of chest pain to admission in the AMI group was 5.79 ± 5.15 hours (range 10 minutes to 20 hours, median 4 hours).

Table 2 and Fig. 2 show the levels of cTnT in both groups. The mean levels of cTnT in AMI group were higher than control in every studied time. Magnitude and rate of change of cTnT were not different between both groups except the rate of change at time between 0 hour and 24 hours since admission (0.18778 ± 0.29717 vs. -0.00008 ± 0.00113 nanogram per milliliter per hour).

The diagnostic level of cTnT for AMI in patients with chronic renal dysfunction was calculated

Table 2. Levels and changes of cardiac troponin T in AMI and control group at a specific time after admission

Cardiac Troponin T	Group of patients		p-value
	AMI group	Control group	
0 hour (ng/mL)			
Mean ± SD	2.51420 ± 5.08073	0.06700 ± 0.10540	0.026
Median	0.5160	0.0230	
6 hour (ng/mL)			
Mean ± SD	5.43630 ± 7.58874	0.06680 ± 0.11073	0.002
Median	2.5600	0.0120	
12 hour (ng/mL)			
Mean ± SD	5.45170 ± 6.72712	0.06752 ± 0.10737	0.001
Median	3.0800	0.0100	
24 hour (ng/mL)			
Mean ± SD	5.45930 ± 6.88577	0.07050 ± 0.11162	0.002
Median	2.9500	0.0130	
Magnitude of change at 0-6 hour (ng/mL)			
Mean ± SD	2.90100 ± 7.09911	-0.02640 ± 0.13082	0.066
Median	0.4375	0.0000	
Magnitude of change at 0-12 hour (ng/mL)			
Mean ± SD	2.91640 ± 7.49130	-0.00050 ± 0.02083	0.082
Median	0.7070	0.0000	
Magnitude of change at 0-24 hour (ng/mL)			
Mean ± SD	2.93150 ± 7.94769	-0.00010 ± 0.02695	0.117
Median	0.5840	0.0000	
Rate of change 0-6 hour (ng/mL/hr)			
Mean ± SD	0.49355 ± 1.18020	-0.00020 ± 0.00414	0.062
Median	0.1042	0.0000	
Rate of change 0-12 hour (ng/mL/hr)			
Mean ± SD	0.22968 ± 0.62850	0.00340 ± 0.01561	0.107
Median	0.0465	0.0000	
Rate of change 0-24 hour (ng/mL/hr)			
Mean ± SD	0.18778 ± 0.29717	-0.00008 ± 0.00113	0.009
Median	0.0512	0.0000	

AMI: acute myocardial infarction, SD: standard deviation, ng/ml: nanogram per millilitre, ng/ml/hr: nanogram per millitre per hour

with ROC curve as shown in Table 3. Patients with chronic renal dysfunction can be diagnosed to have AMI if cTnT level is at least 0.1 nanogram per milliliter with sensitivity 90.90%, specificity 84.50% and if patients with chronic renal replacement were excluded, the sensitivity and specificity of this diagnostic cTnT level will increase to 91.30% and 100.00% respectively. The authors reanalyzed our data by using the time from the onset of chest pain to blood sampling time for cTnT to separated patients with AMI into 4 groups i.e., 0-6, 6-12, 12-18 and > 18 hours after chest pain. The authors compared cTnT of AMI group collected within these specific time periods with cTnT collected at 0-6, 6-12, 12-18 and > 18 hours from admission in control group in Table 4. Mean cTnT levels were significantly higher in the AMI group than in control in every time periods from this reanalyzed data. The authors also found that the magnitude of change of cTnT from the first samples at 0-6 hours to the second samples at 6-12 hours and also to the third samples at 12-18 hours after chest pain in the AMI group were significantly higher than the corresponding levels of cTnT in the control group, $p = 0.019$ and 0.020 respectively. The changing rate of cTnT from the first sample to the second sample and to the third sample after chest pain in the AMI group were also significantly higher than the corresponding levels in the control group, $p = 0.017$ and 0.034 respectively. However, the magnitude of change and rate of change of cTnT from the first sample to the fourth sample at > 18 hours after chest pain in the AMI

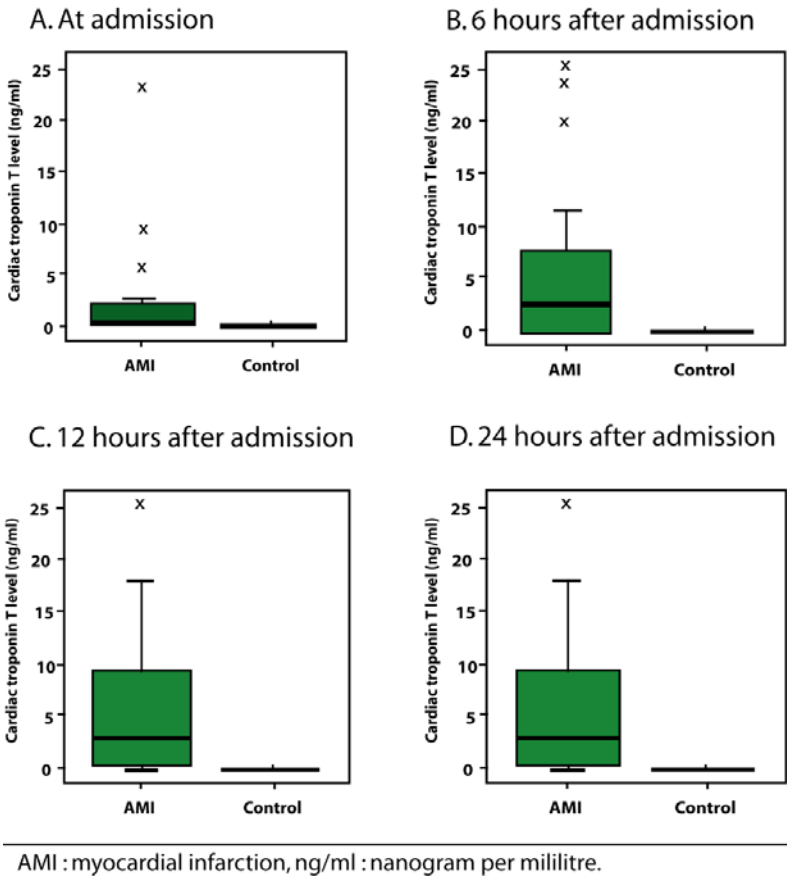

Fig. 2 Cardiac troponin T level at admission and 6, 12 and 24 hours later

group were not significantly higher than the corresponding levels of cTnT in the control group (Table 4).

There were three patterns of cTnT change in the present study. The first pattern was rapid rise and sustained, the second pattern was rapid rise and fall, and the third pattern was raised level without change. The first two patterns were found only in the AMI group while the third pattern was found in the control group (Fig. 3).

There were four (17.39%) and fourteen (60.87%) patients from the control group with cTnT level $\geq$ 0.1 and $\geq$ 0.01 nanogram per milliliter respectively. All of those with cTnT level $\geq$ 0.1 nanogram per milliliter had stage 5 of chronic renal dysfunction as shown in Table 5. There were five patients with chronic renal replacement who had cTnT level $\geq$ 0.01 nanogram per milliliter, among which two had cTnT level $\geq$ 0.1 nanogram per milliliter. Mean cTnT level of stage 5 chronic renal dysfunction in the control group was significantly higher than stage 3 and stage 4 in the same group ($p <$ 0.05). Two patients with stage 5 chronic renal dysfunction in the control group had diabetic nephropathy and had a trend toward a higher mean cTnT level (0.343 nanogram per milliliter) than the rest without diabetic nephropathy (0.085 nanogram per milliliter) in the same stage ($p = 0.32$).

Discussion

In the present study, the authors found that in patients with an established diagnosis of chronic renal dysfunction, those with AMI had significantly higher mean cTnT level within the first 24 hours of admission than those without AMI. The authors also found that the cTnT level at least 0.1 nanogram per milliliter in patients with chronic renal dysfunction had the sensitivity and specificity for AMI diagnosis of 90.90 and 84.50 percent respectively. If patients with chronic renal replacement were excluded the sensitivity and specificity of this cTnT level for AMI diagnosis will increase to 91.30 and 100.00 percent respectively.

Table 3. Diagnostic levels of cardiac troponin T (n = 23)

Patient group by renal function	cTnT (ng/ml)	AUC	Sensitivity	Specificity
CrCl < 60 ml/min/1.73 m^2	0.1	0.940	90.90%	84.50%
	0.2		77.30%	85.70%
	0.3		71.60%	98.60%
	0.4		70.50%	100.00%
	0.5		68.20%	100.00%
CrCl < 60 ml/min/1.73 m^2 Except hemodialysis	0.1	0.976	91.30%	100.00%
	0.2		80.00%	100.00%
	0.3		73.80%	100.00%
	0.4		72.50%	100.00%
	0.5		70.00%	100.00%
CrCl 15 - 59 ml/min/1.73 m^2	0.1	0.983	100.00%	96.60%
	0.2		93.30%	100.00%
	0.3		91.70%	100.00%
	0.4		90.00%	100.00%
	0.5		88.30%	100.00%
CrCl 30 - 59 ml/min/1.73 m^2	0.1	0.987	90.00%	96.80%
	0.2		90.00%	100.00%
	0.3		85.00%	100.00%
	0.4		80.00%	100.00%
	0.5		80.00%	100.00%
CrCl 15 – 29 ml/min/1.73 m^2	0.1	0.987	97.50%	92.90%
	0.2		97.50%	100.00%
	0.3		95.00%	100.00%
	0.4		92.50%	100.00%
	0.5		92.50%	100.00%
CrCl < 15 ml/min/1.73 m^2	0.1	0.645	85.71%	48.00%
	0.2		57.14%	52.00%
	0.3		32.14%	76.00%
	0.4		28.57%	100.00%
	0.5		25.00%	100.00%

cTnT: cardiac troponin T, AUC: area under the curve, CrCl: creatinine clearance, ng/ml: nanogram per millitre, ml/min/1.73 m^2: millitre per minute per body surface area 1.73 square metre

However, this diagnostic cTnT level needs to be interpreted with caution in stage 5 chronic renal dysfunction especially in those who also had diabetic nephropathy; since these patients could have higher rate of false positive. Van Lente et al reported that the level of cTnT above 0.5 nanogram per milliliter could accurately predict adverse cardiovascular outcomes in every stages of chronic renal dysfunction but this level could not be used to diagnose AMI[15]. There were reports that patients with diabetic nephropathy had higher cTnT level than those with chronic renal dysfunction from other causes[16,17], the authors also found this phenomenon in the present study.

In the present study, the authors found that the magnitude of change of cTnT within the first 24 hours of admission showed no significant difference between AMI and the control group. The rate of change of cTnT from 0-6 hour and from 0-12 hour also showed no significant difference between AMI and the control group. However, the rate of change from 0-24 hour was significantly higher in the AMI group. The application of this finding was somewhat limited because there was a high degree of variation in this rising rate of cTnT in both groups (Table 2) so the rate of change, which could be considered as diagnostic, could not be identified.

The authors reanalyzed the presented data by using the levels and increments of cTnT at a specific time after the onset of chest pain in AMI group compared with the levels and increments of cTnT at a

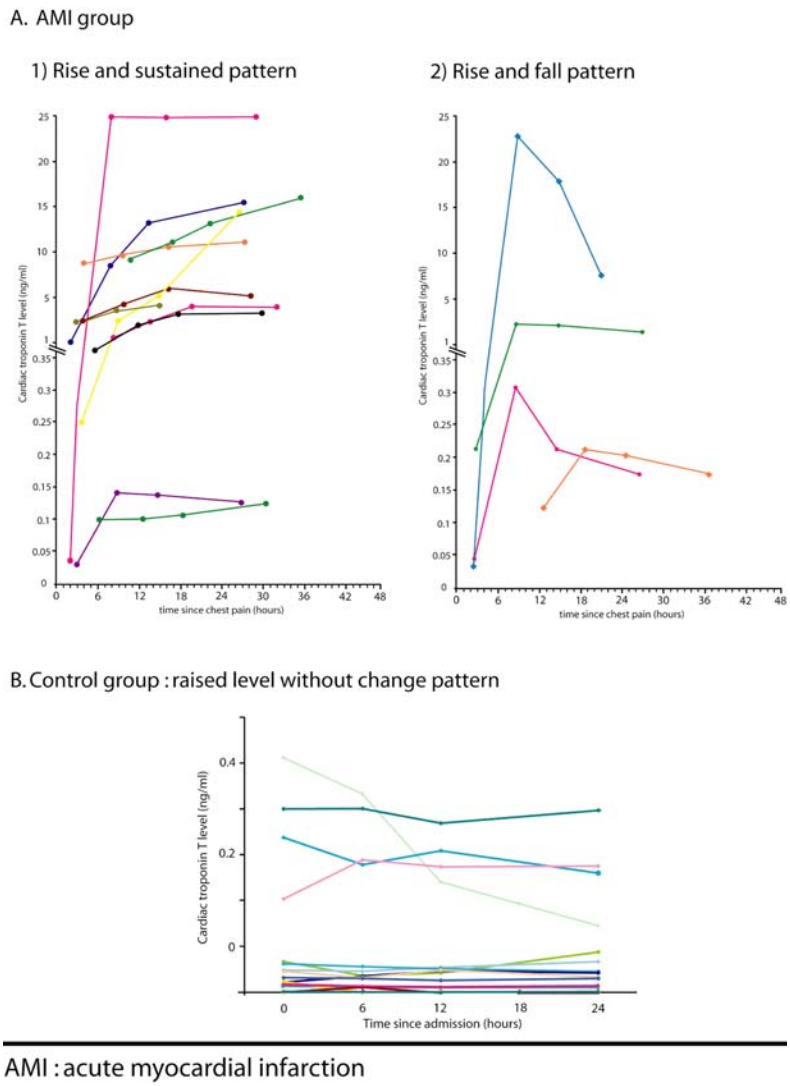

Fig. 3 Patterns of cardiac troponin T in AMI and control group

specific time after admission in the control group (this group had no chest pain and the pattern of changes of cTnT level was a raised level without changes, Fig. 3). The authors found that the magnitude and rate of change of cTnT in AMI group were significantly higher than in the control group in the first 18 hours after chest pain (Table 4). The application of this finding was also limited because there was also a high degree of variation in the magnitude and rate of change of cTnT as in the previous analysis.

Conclusion

The level of cTnT within the first 24 hours of admission at least 0.1 nanogram per milliliter was diagnostic for AMI in patients with chronic renal dysfunction with highly acceptable accuracy. The sensitivity and specificity of test was much better if patients with chronic renal replacement were excluded. The magnitude and rate of change of cTnT in the first 18 hours after AMI were higher than in those admitted without AMI but variations were too high for diagnostic use.

Acknowledgement

This study was supported by a grant from the Faculty of Medicine Research Fund, Chiang Mai University.

Table 4. Levels and change of cardiac troponin T in AMI group at a specific time after the onset of chest pain and in control group at a specific time after admission

Cardiac Troponin T	Group of patients		p-value
	AMI group*	Control group**	
0-6 hour (ng/mL)			
Mean ± SD	1.15630 ± 2.15005	0.06710 ± 0.10541	0.020
Median	2.3050	0.0230	
6-12 hour (ng/mL)			
Mean ± SD	5.73550 ± 8.20734	0.06680 ± 0.11073	0.003
Median	2.5600	0.0120	
12-18 hour (ng/mL)			
Mean ± SD	5.87410 ± 7.41765	0.06752 ± 0.10737	0.001
Median	2.2100	0.0100	
18-24 hour (ng/mL)			
Mean ± SD	5.56930 ± 6.94848	0.07050 ± 0.11162	0.001
Median	2.1700	0.0130	
Magnitude of change between 0-6 hour and 6-12 hour (ng/mL)			
Mean ± SD	4.33530 ± 8.18937	-0.02640 ± 0.13082	0.019
Median	0.6400	0.0000	
Magnitude of change between 0-6 hour and 12-18 hour (ng/mL)			
Mean ± SD	3.65800 ± 6.93330	-0.00050 ± 0.02083	0.020
Median	0.9700	0.0000	
Magnitude of change between 0-6 hour and > 18 hour (ng/mL)			
Mean ± SD	3.19320 ± 7.62355	-0.00010 ± 0.02695	0.076
Median	0.8245	0.0000	
Rate of change between 0-6 hour and 6-12 hour (ng/mL/hr)			
Mean ± SD	0.73728 ± 1.35823	-0.00020 ± 0.00414	0.017
Median	0.1850	0.0000	
Rate of change between 0–6 hour and 12-18 hour (ng/mL/hr)			
Mean ± SD	0.29183 ± 0.60146	0.00341 ± 0.01561	0.034
Median	0.0465	0.0000	
Rate of change between 0–6 hour and > 18 hour (ng/mL/hr)			
Mean ± SD	0.11922 ± 0.39890	-0.00008 ± 0.00113	0.201
Median	0.0452	0.0000	

* After chest pain, ** After admission
AMI: acute myocardial infarction, SD: standard deviation, ng/mL: nanogram per milliliter, ng/mL/hr: nanogram per millitre per hour

Table 5. Cardiac troponin T levels in control group

Stages of renal dysfunction	Mean ± SD (Range, ng/mL)	Number of patients in each cardiac troponin T level (ng/ml)		
		≥ 0.1	0.01 - < 0.1	< 0.01
Stage 3	0.016 ± 0.0184 (<0.01-0.067)	0	2	6
Stage 4	0.041 ± 0.0233 (<0.01-0.089)	0	6	2
Stage 5	0.161 ± 0.1399 (<0.01-0.400)	4	2	1

SD: standard deviation, ng/mL: nanogram per millilitre

References

1. Newby LK, Rutsch WR, Califf RM, Simoons ML, Aylward PE, Armstrong PW, et al. Time from symptom onset to treatment and outcomes after thrombolytic therapy. GUSTO-1 Investigators. J Am Coll Cardiol 1996; 27: 1646-55.
2. Cannon CP. Time to treatment: a crucial factor in thrombolysis and primary angioplasty. J Thromb Thrombolysis 1996; 3: 249-55.
3. Roberts R, Fromm RE. Management of acute coronary syndromes based on risk stratification by biochemical markers: an idea whose time has come. Circulation 1998; 98: 1831-3.
4. Henrikson CA, Howell EE, Bush DE, Miles JS, Meininger GR, Friedlander T, et al. Prognostic usefulness of marginal troponin T elevation. Am J Cardiol 2004; 93: 275-9.
5. Lamb EJ, Webb MC, Abbas NA. The significance of serum troponin T in patients with kidney disease: a review of the literature. Ann Clin Biochem 2004; 41: 1-9.
6. De Zoysa JR. Cardiac troponins and renal disease. Nephrology 2004; 9: 83-8.
7. Diris JH, Hackeng CM, Kooman JP, Pinto YM, Hermens WT, Dieijen-Visser MP. Impaired renal clearance explains elevated troponin T fragments in hemodialysis patients. Circulation 2004; 109: 23-5.
8. Freda BJ, Tang WH, Van Lente F, Peacock WF, Francis GS. Cardiac troponins in renal insufficiency: review and clinical implications. J Am Coll Cardiol 2002; 40: 2065-71.
9. Apple FS, Sharkey SW, Hoeft P, Skeate R, Voss E, Dahlmeier BA, et al. Prognostic value of serum cardiac troponin I and T in chronic dialysis patients: a 1-year outcomes analysis. Am J Kidney Dis 1997; 29: 399-403.
10. Deegan PB, Lafferty ME, Blumsohn A, Henderson IS, McGregor E. Prognostic value of troponin T in hemodialysis patients is independent of co-morbidity. Kidney Int 2001; 60: 2399-405.
11. Iliou MC, Fumeron C, Benoit MO, Tuppin P, Calonge VM, Moatti N, et al. Prognostic value of cardiac markers in ESRD: Chronic Hemodialysis and New Cardiac Markers Evaluation (CHANCE) study. Am J Kidney Dis 2003; 42: 513-23.
12. Gault MH, Longerich LL, Harnett JD, Wesolowski C. Predicting glomerular function from adjusted serum creatinine. Nephron 1992; 62: 249-56.
13. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification Am J Kidney Dis 2002; 39: S1-266.
14. 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.
15. Van Lente F, McErlean ES, DeLuca SA, Peacock WF, Rao JS, Nissen SE. Ability of troponins to predict adverse outcomes in patients with renal insufficiency and suspected acute coronary syndromes: a case-matched study. J Am Coll Cardiol 1999; 33: 471-8.
16. Li D, Keffer J, Corry K, Vazquez M, Jialal I. Nonspecific elevation of troponin T levels in patients with chronic renal failure. Clin Biochem 1995; 28: 474-7.
17. Jeon DS, Lee MY, Kim CJ, Cho JM, Moon KW, Kim BS, et al. Clinical findings in patients with cardiac troponin T elevation and end-stage renal disease without acute coronary syndrome. Am J Cardiol 2004; 94: 831-4.

ระดับการวินิจฉัยของ cardiac troponin T ในผู้ป่วยที่มีความผิดปกติของการทำงานของไตแบบเรื้อรัง

อภิชาต สุคนธสรรพ์, สุเมธ พงศ์หล่อพิศิษฏ์

วัตถุประสงค์: *เพื่อหาระดับการวินิจฉัยและแบบแผนของการเพิ่มขึ้นของ cardiac troponin T (cTnT) ในการวินิจฉัยโรคกล้ามเนื้อหัวใจตายชนิดเฉียบพลันในผู้ป่วยที่มีความผิดปกติของการทำงานของไตแบบเรื้อรัง*

วัสดุและวิธีการ: *เป็นการศึกษานำร่องแบบ cross-sectional เปรียบเทียบระดับของ cTnT ในผู้ป่วยที่มีความผิดปกติของการทำงานของไตแบบเรื้อรังและได้รับการยืนยันว่าเป็นโรคกล้ามเนื้อหัวใจตายชนิดเฉียบพลัน กับระดับ cTnT ของผู้ป่วยที่มีความผิดปกติของการทำงานของไตแบบเรื้อรังที่มาด้วยอาการอื่น โดยทำการตรวจระดับของ cTnT ณ เวลา 0, 6, 12 และ 24 ชั่วโมงภายหลังเข้ารับการรักษาในโรงพยาบาลทั้งสองกลุ่ม*

ผลการศึกษา: *มีผู้ป่วยจำนวนกลุ่มละ 23 ราย ค่าเฉลี่ยระดับ cTnT ของกลุ่มผู้ป่วยที่มีโรคกล้ามเนื้อหัวใจตายชนิดเฉียบพลันสูงกว่าของกลุ่มควบคุมอย่างมีนัยสำคัญ แต่ไม่มีความแตกต่างในระดับการเพิ่มขึ้นและอัตราการเพิ่มขึ้นของ cTnT หลังจากนอนโรงพยาบาลอย่างมีนัยสำคัญ ระดับของ cTnT ที่ใช้วินิจฉัยโรคกล้ามเนื้อหัวใจตายชนิดเฉียบพลันในผู้ป่วยที่มีความผิดปกติของการทำงานของไตแบบเรื้อรังคือ 0.1 นาโนกรัมต่อมิลลิลิตร การวินิจฉัยโดยใช้ค่า cTnT ระดับนี้มีความไวร้อยละ 90.90 และความจำเพาะร้อยละ 84.50 ความไวและ ความจำเพาะของระดับการวินิจฉัยจะเพิ่มขึ้นเป็นร้อยละ 91.30 และร้อยละ 100.00 ถ้าตัดกลุ่มผู้ป่วยที่ต้องทำการ ฟอกไตออกจากการศึกษา*

สรุป: *ระดับของ cTnT ที่ใช้วินิจฉัยโรคกล้ามเนื้อหัวใจตายชนิดเฉียบพลันในผู้ป่วยที่มีความผิดปกติของการทำงานของไตเรื้อรังใน 24 ชั่วโมงแรกคือ 0.1 นาโนกรัมต่อมิลลิลิตร ความไวและความจำเพาะของการใช้ระดับ cTnT นี้ในการช่วยวินิจฉัยจะเพิ่มขึ้นในกลุ่มผู้ป่วยที่ไม่ได้รับการฟอกไต*