Cardiac Troponin-T in Pre-End Stage Kidney Disease

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Background: *Cardiac troponin T level (cTnT) is commonly increased in end stage chronic kidney disease (CKD) in the absence of acute myocardial infarction. There are few data available on serum cTnT concentration in patients with pre-end stage CKD.*
Objective: *To evaluate the correlation of cTnT level and severity of kidney disease in patients with CKD stage 3 and 4 and to evaluate whether there is a relationship between left ventricular mass index and cTnT level.*
Material and Method: *Patients (103) with CKD stage 3-4 between 26 and 85 years of age (mean 60.0 $\pm$ 11.9) entered the present study. Serum cTnT determined using a third-generation electrochemiluminescent immunoassay on an Elecsys 2010 analyzer (Roche Diagnostics Ltd.). All patients underwent echocardiography. Left ventricular hypertrophy (LVH) was considered when LV mass index exceeded 125 g/m^2.*
Results: *Overall, 28 patients (28.2%) had cTnT > 0.01 μg/L and two patients (1.8%) had cTnT > 0.1 μg/L cTnT concentration was commonly increased in more severe CKD (9 patients in stage 3 and 20 patients in stage 4). LVH was not associated with increased cTnT (p = 0.105).*
Conclusion: *The present study demonstrated that the elevated cTnT > 0.01 μg/L is relatively common in patients with CKD stage 3-4 who do not require dialysis treatment, however, serum cTnT level above > 0.1 μg/L is uncommon in this population. Increased serum cTnT is associated with decreased renal clearance but not LVH.*

Keywords: *Troponin T, Pre end- stage, Kidney disease*

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Coronary artery disease (CAD) is the major cause of death in patients with chronic kidney disease (CKD), responsible for up to 45% of overall mortality[1,2]. The cardiovascular mortality rate in end stage renal disease patients on hemodialysis is approximately 10 to 20 times higher than in the general population[3]. The incidence of cardiovascular disease was increased by 40% even in early stage of CKD[4]. Left ventricular hypertrophy (LVH) is present in the vast majority of patients, predisposing them to risk of cardiac death[5]. Early detection of cardiovascular disease could facilitate more aggressive treatment of those at increased risk.

The biochemical diagnosis of myocardial infarction in patients with CKD is always complicated by the fact that serum markers of myocardial necrosis such as creatine kinase, MB-fraction of creatine kinase (CK-MB) are commonly increased in end stage CKD patients, even in the absence of clinically suspected myocardial infarction[6,7]. Cardiac troponin T (cTnT) is a component of the contractile apparatus of the cardiac muscle. Because of its high tissue specificity, cTnT is a cardiospecific, highly sensitive marker of myocardial damage[8]. However, increases in serum cTnT concentration have been reported in patients with CKD in the absence of acute myocardial infarction[9]. Most studies have demonstrated that increased cTnT is a powerful predictor of mortality and associated with LVH in the hemodialysis patients[9,10].

Currently, there is few data regarding cTnT level in pre-end stage CKD (stage 3-4) patients who do not require dialysis treatment. The objectives of the present study were, therefore, to evaluate the level of

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cTnT in patients with CKD stage 3 and 4 and to evaluate whether there is a relationship between left ventricular mass index and cTnT level in such patients.

Material and Method

Patients

Between August 2005 and October 2006, 103 patients with CKD stage 3 and 4 attending nephrology outpatient clinics at Khon Kaen University Hospital were enrolled into the present study. Patients who had the following conditions were excluded; acute renal failure, receiving dialysis, myocardial infarction, congestive heart failure, recent infection, ischemic ST- T change or pathological Q wave on electrocardiogram, anginal chest pain, and glomerular filtration rate (GFR) < 15 mL/min or > 60 mL/min. Clinical history was recorded including age, sex, weight, height, primary renal diagnosis, blood pressure, history of diabetic, and smoking history. GFR was calculated using the Cockroft-Gault formula as follows:

Males: (140 - age) x weight (kg)/ 72 x serum creatinine (mg/100 mL)

Females: 0.85 (140 - age) x weight (kg)/ 72 x serum creatinine (mg/100 mL)

Patients were stratified into stage 3 CKD (GFR = 30-59 mL/min/1.73 m^2) and stage 4 CKD (GFR = 15-29 mL/min/1.73 m^2) according to National Kidney Foundation Guideline[11].

Sample analysis

Blood samples were collected in the non-fasting state. Serum cTn-T was measured with a third-generation electrochemiluminescent immunoassay on an Elecsys 2010 analyzer (Roche Diagnostics Ltd.). The lower detection limit of this assay was 0.01 μg/L and the between- run coefficient of variation (CV) was less than or equal to 10%. Serum creatinine was measured by buffered kinetic Jaffe' reaction method on an Integra 800 analyzer (Roche Diagnostics Ltd.). The reported CV between run was 0.8% at a concentration of 1.3 mg/dL.

Echocardiography

Transthoracic echocardiography was performed using a 2.5- or 3.5-MHz phase array transducer (Sonos 5500, Hewlett-Packard, Andover, Massachusetts). Gain setting and gray scale were adjusted to optimize imaging of both epicardial and endocardial surfaces. M-mode studies were taken generally from the short-axis view at the level of largest left ventricular diameter. Left ventricular mass (LVM) was calculated using Devereux and Reichek[12] formula and indexed by body surface area. LVH was considered present when left ventricular mass index (LVMI) exceeded 125 g/m^2[2,13]. All measurements were made at the time of maximum chamber dimension using a leading edge-to-leading edge convention according to American Society of Echocardiography guidelines[14]. LV ejection fraction was used to evaluate LV systolic performance. Doppler echocardiographic measurement of mitral flow was used to determine diastolic performance of LV. The following diastolic parameters were measured: early (E) and late (A) peak mitral inflow velocities, E/A ratio (E/A), deceleration time (DT), and isovolumic relaxation time (IVRT).

Statistical analysis

Continuous variables are presented as means $\pm$ SD, and categorical variables are described with frequencies and percentages. The comparison of continuous and categorical variables was performed by the *t* test. The categorical variables were compared using the Chi-square test. A logistic regression analysis was used to examine the effect of LVMI and GFR on cTn-T concentration above 0.01 μg/L. A p-value of < 0.05 was considered statistically significant. All the analyses were performed using SPSS version 11.0 (SPSS Inc., Chicago, Illinois, USA).

Results

Table 1 summarizes the clinical characteristics of the study population. Among 103 patients, there were 52 patients in CKD stage 3 and 51 patients in CKD stage 4. Hypertension and diabetes were presented in 39.8% and 34.0% respectively. The mean age of the study population was 60 years. The patients with CKD stage 4 were older than those with CKD stage 3 (65.1 $\pm$ 10.7 vs. 55.0 $\pm$ 10.8; p = 0.000).

Overall, 29 patients (28.2%) had cTnT > 0.01 μg/L. The number of patients with elevated cTnT was significantly higher in patients with CKD stage 4 compared to those with CKD stage 3. However, two patients (1.9%) in CKD stage 4 had cTnT > 0.10 μg/L. The highest level of cTnT was 0.11 μg/L. Elevated cTn-T was associated with reduced GFR (odds ratio [OR] 0.94, 95% confidence interval [CI] = 0.90-0.99, p = 0.020; Table 2, Fig. 1). The clinical characteristics including systolic and diastolic blood pressure between patients in CKD stage 3 and those in CKD stage 4 were not statistically different.

Echocardiographic data were available in all patients. The prevalence of LVH was 36.5% in CKD

Table 1. Clinical characteristics, echocardiographic findings and troponin T level among 103 chronic kidney disease patients

Variable	Overall (n = 103)	Stage 3 (n = 52)	Stage 4 (n = 51)	p-value
Age (y), mean ± SD	60.0 ± 11.9	55.0 ± 10.8	65.1 ± 10.7	0.000
Male, n (%)	58 (56.3)	34 (65.4)	24 (47.0)	0.075
Smoking, n (%)	30 (29.1)	15 (28.8)	15 (29.4)	1.000
Hypertension, n (%)	41 (39.8)	20 (38.5)	21 (41.2)	0.842
Diabetes, n (%)	35 (34.0)	17 (32.7)	18 (35.3)	0.837
SBP, mean ± SD	129.6 ±14.5	129.1 ± 13.7	130.2 ± 15.4	0.708
DBP, mean ± SD	78.9 ± 9.2	80.4 ± 8.0	77.3 ± 10.1	0.092
BSA (m^2), mean ± SD	1.6 ± 0.2	1.7 ± 0.2	1.6 ± 0.2	0.000
LVM (g), mean ± SD	203.1 ± 77.0	201.4 ± 78.6	204.8 ± 76.2	0.819
LVMI (g/m^2), mean ± SD	126.0 ± 39.8	120.5 ± 41.1	131.6 ± 38.1	0.158
LVH, n (%)	44 (42.7)	19 (36.5)	25 (49.0)	0.200
MV E/A ratio	0.9 ± 0.4	1.1 ± 0.5	0.8 ± 0.3	0.003
DT (msec), mean ± SD	211.6 ± 75.3	204.4 ± 75.6	218.8 ± 75.1	0.339
IVRT (msec), mean ± SD	121.0 ± 30.9	111.7 ± 30.5	130.7 ± 28.7	0.002
LVEF (%), mean ± SD	63.5 ± 8.9	62.9 ± 11.1	64.1 ± 5.9	0.499
cTnT ≥ 0.01 μg/L, n (%)	29 (28.2)	9 (17.3)	20 (39.2)	0.016

SBP = systolic blood pressure; DBP = diastolic blood pressure; BSA = body surface area; LVM = left ventricular mass; LVMI = left ventricular mass index; MV = mitral valve; DT = deceleration time; IVRT = isovolumic relaxation time; LVEF = left ventricular ejection fraction

Table 2. Odd Ratio (OR) for increased cTn-T > 0.01 μg/L

Factor	OR	95% CI	p-value
GFR (mL/min/1.73 m^2)	0.94	0.90-0.99	0.020
LVMI (g/m^2)	1.00	0.99-1.02	0.105

GFR = glomerular filtration rate; LVMI = left ventricular mass index

stage 3 and 49.0% in stage 4. LVMI was not associated with the increase of serum cTnT level > 0.01 μg/L (OR = 1.00, 95% 0.99-1.02, p = 0.105; Table 2, Fig. 2). LV systolic function (LVEF > 50%) was normal in all patients, however left ventricular diastolic performance, assessed by mitral valve E/A ration and isovolumic relaxation time (IVRT), was statistically significantly decreased in patients with CKD stage 4 compared to those with CKD stage 3.

Discussion

The principle findings of the present study were 1) increased serum cTnT > 0.01 μg/L was relatively common in pre end stage chronic kidney disease who had no clinical setting of acute myocardial infarction; 2) elevated cTnT was associated with decreased GFR but not increased LV mass.

Increases in serum cTnT concentrations have been reported in 50% of patients with CKD and no evidence of myocardial necrosis[15]. Ooi and House[16] observed increased cTnT concentrations (> 0.1 μg/L) in 29% of hemodialysis patients without overt acute coronary disease. Roppollo et al[17] found that 25 of 49 hemodialysis patients had cTnT > 0.1 μg/L and patients with diabetes were more likely to have elevated cTnT. Most of these studies included end stage kidney disease or on hemodialysis patients, in whom increased cTnT was the predictor of cardiac and all cause mortality[18,19]. However, in pre end stage kidney disease patients who do not require dialysis therapy, the data was limited. cTnT was undetectable in nine CKD patients with GFR of 40-60 mL/min but the levels > 0.1 μg/L were detected in only four of 46 CKD patients with GFR < 40 mL/min who were not receiving dialysis[20]. Mockel et al[21] also observed increased cTnT concentrations > 0.1 μg/L in four of 20 pre end stage kidney disease patients without clinical evidence of an acute coronary syndrome. Recently, Abbas et al[22] reported that serum cTnT was increased > 0.01 μg/L in 43% of all patients with CKD stages 3-5 who were not receiving dialysis. Furthermore, they found that the elevated cTnT level was associated with decreased GFR. In that study, there was no association between cTnT level and LVMI.

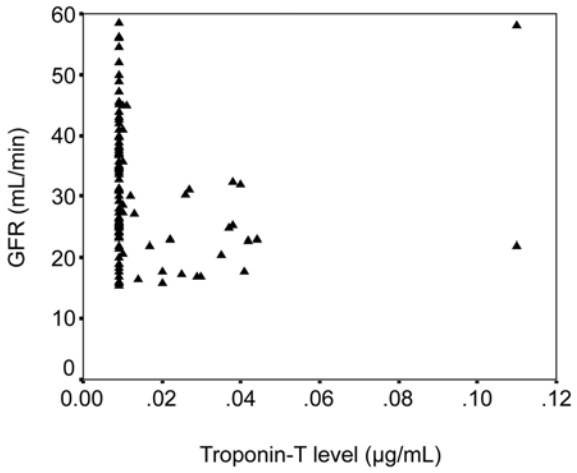

Fig 1. Relation of cTn-T with GFR in patients with CKD stage 3-4
For graphic purposes, patients with cTnT concentration < 0.01 µg/L has been assigned concentration of 0.009

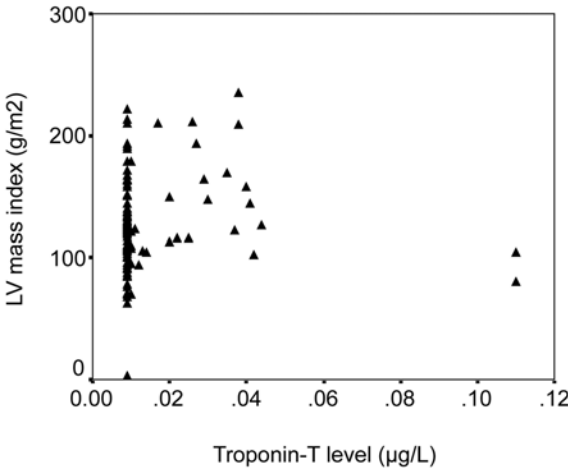

Fig 2. *Relation of cTn-T with LV mass index in patients with CKD stage 3-4*
For graphic purposes, patients with cTnT concentration < 0.01 µg/L has been assigned concentration of 0.009.

In the present study, 28.2% of patients with CKD stages 3-4 had cTnT level > 0.01 µg/L. Increased cTnT levels were more common in patients with more advanced kidney disease. The decreased prevalence of increased cTnT in this present study compared with the study of Abbas et al[22] probably reflects the relatively less severe disease in the present study (patients with end stage CKD were excluded from the present study). Only two of 103 patients in this present study had cTnT level above the cutoff value (0.1 µg/L) for the definition of acute myocardial infarction based on WHO criteria[23].

The causes of increased cardiac troponin concentrations in patients with chronic kidney disease in the absence of acute coronary syndrome are still controversial. This could have resulted from subclinical myocardial damage caused by silent ischemia or myocardial remodeling in the development of LVH, although decreased clearance process could not be completely excluded[24,25]. The correlation between LV hypertrophy and increased cardiac troponins is controversial. Although previous studies have demonstrated that cTnT was increased in the settings of LVH and cardiac failure in the absence of acute coronary syndrome[25,26]. Abbas et al[22] however, found no correlation between left ventricular mass and cTnT concentration in non-dialysis patients.

The diagnosis of acute coronary syndrome in patients with CKD is complicated by the fact that such patients may present with atypical clinical presentations and serum cardiac troponins may be elevated in the absence of significant coronary artery disease. The cutoff level of cTn T for the diagnosis of acute myocardial infarction is not clearly established. It has been suggested that a higher level of cTnT (0.5 µg/L) could be used to make a diagnosis of acute myocardial infarction in patients with end stage kidney disease[27]. However, in the present study, the authors found that cTnT level > 0.1 µg/L is uncommon in patients with CKD stage 3-4 and this cutoff level may be considered as a threshold to make a provisional diagnosis of acute myocardial infarction in such patients.

In conclusion, elevated cTnT > 0.01 µg/L is relatively common in patients with CKD stage 3-4 who do not require dialysis treatment, however, serum cTnT level above > 0.1 µg/L is uncommon in this population. Increased serum cTnT is associated with decreased renal clearance but not LVH.

Limitations

There are several limitations in the present study**.** First, coronary artery disease was not comprehensively investigated in all patients with increased serum cTnT. This made it impossible to definitely exclude coronary artery disease in the present study. Second, the prospectively follow up to assess the prognostic value of cTnT in CKD patients is not conducted

although it has been demonstrated that cTn-T is a marker of decreased survival.

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ระดับโทรโปนิน-ที ในผู้ป่วยโรคไตวายก่อนระยะสุดท้าย

ทรงศักดิ์ เกียรติชูสกุล, ดุจดาว สหัสทัศน์, ไชยสิทธิ์ วงศ์วิภาพร, ชลธิป พงศ์สกุล

ภูมิหลัง: *ระดับโทรโปนินจะสูงขึ้นในผู้ป่วยโรคไตวายเรื้อรังระยะสุดท้าย แม้ว่าผู้ป่วยจะไม่มีลักษณะทางคลินิกของภาวะกล้ามเนื้อหัวใจตายเฉียบพลัน อย่างไรก็ตามข้อมูลเกี่ยวกับระดับโทรโปนิน-ทีในผู้ป่วยไตวายเรื้อรังก่อนระยะสุดท้ายยังมีไม่มากนัก*

วัตถุประสงค์: *เพื่อศึกษาหาความสัมพันธ์ ระหว่างระดับโทรโปนิน-ทีกับความรุนแรงของโรคไตเรื้อรัง และหาความสัมพันธ์ระหว่างระดับโทรโปนิน-ทีและภาวะหัวใจห้องล่างซ้ายโตในผู้ป่วยไตวายเรื้อรังก่อนระยะสุดท้าย*

วัสดุและวิธีการศึกษา: *ศึกษาผู้ป่วยโรคไตวายเรื้อรังก่อนระยะสุดท้าย 103 ราย อายุ 26-85 ปี (เฉลี่ย 60 ปี) โดยตรวจวัดระดับโทรโปนินในเลือดด้วยวิธี electrochemiluminescent immunoassay ผู้ป่วยทุกราย ได้รับการตรวจคลื่นเสียงสะท้อนหัวใจผ่านทางผนังทรวงอก ผู้ป่วยที่มีน้ำหนักหัวใจห้องล่างซ้ายมากกว่า 125 กรัม พื้นที่ผิวกาย 1 ตารางเมตร จัดว่ามีภาวะหัวใจโต*

ผลการศึกษา: *การศึกษานี้พบว่าในผู้ป่วยร้อยละ 28.2 มีระดับโทรโปนินในเลือดมากกว่า 0.01 ไมโครกรัม/ลิตร แต่มีเพียงร้อยละ1.8 เท่านั้นที่มีระดับโทรโปนิน-ทีในเลือดมากกว่า 0.1ไมโครกรัม/ลิตร ระดับโทรโปนิน-ทีมีความสัมพันธ์กับระดับความรุนแรงของโรคไต โดยพบว่าผู้ป่วยไตวายระยะที่ 3 จำนวน 9 ราย และผู้ป่วยในระยะที่ 4 จำนวน 20 ราย มีระดับโทรโปนิน-ทีสูงกว่า 0.01 ไมโครกรัม/ลิตร การศึกษานี้ไม่พบว่า ขนาดของหัวใจห้องล่างซ้าย มีความสัมพันธ์กับระดับของโทรโปนิน-ที*

สรุป: *ในผู้ป่วยไตวายเรื้อรังก่อนระยะสุดท้ายที่ไม่มีอาการโรคกล้ามเนื้อหัวใจตายเฉียบพลัน ระดับโทรโปนิน-ที มีค่าสูงกว่าปกติได้บ่อย แต่น้อยมากที่มีระดับสูงมากกว่า 0.1 ไมโครกรัม/ลิตร ระดับโทรโปนิน-ทีมีความสัมพันธ์กับความรุนแรงของโรคไตวาย แต่ไม่มีความสัมพันธ์กับขนาดของหัวใจห้องล่างซ้าย*