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CLINICAL STUDY

The Relationship between Cardiac Troponins and Left Ventricular Mass Index in Patients with Chronic Renal Failure

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Keywords cardiac troponins, fluctuation, chronic renal failure, left ventricular hypertrophy, left ventricular mass index

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INTRODUCTION

It is well known that patients with end-stage renal disease (ESRD) have elevated cardiovascular mortality. The prevalence of ischemic heart disease in this population is 10–20 times higher than that in the general population.[1] Cardiac troponin T (cTnT) and cardiac troponin I (cTnI) are important serologic markers for the detection of myocardial cell injury and risk stratification of patients with acute coronary syndrome.[2–4] However, some investigators reported that the cardiac troponins are elevated in a high percentage of patients with ESRD without evidence of acute myocardial ischemia.[5–8] Although elevated serum levels of cardiac troponins have been associated with mortality in hemodialysis patients, the precise mechanism of elevated cardiac troponins in ESRD is not exactly clear. Early studies, in which first generation troponin assays were used, suggest that the elevation of cTnT may be due to the elevated expression of cTnT in myopathic skeletal muscle in patients with

renal disease[9]; the presence of cardiovascular risk factors, especially diabetes mellitus; and the presence of coronary artery disease.[10–12] In recent studies, the presence of left ventricular hypertrophy (LVH), which is common in ESRD patients, has been associated with elevated cTnT in patients with ESRD.[12–14] However, in previous studies, investigators used generally single measurement of cardiac troponins. Therefore, the change of the cardiac troponins with time and its relationship with LVMI in patients with ESRD is not completely known. The aim of this study was to evaluate the relationship between LVMI and the fluctuations in the cardiac troponins at the end of the follow-up.

METHODS

We included the patients with ESRD without clinical coronary artery disease. ESRD was definite as follows. First, glomerular filtration rate (GFR) was estimated according to the Cockcroft-Gault equation:

$$(140 - \text{age}) \times \text{ideal body weight (kg)}/72 \times \text{stable serum creatinine (mmol/L)} \times (0.85 \text{ for female patients}),$$

and then ESRD is defined as GFR < 15 mL/min/(1.73 m^2). Diabetic and hypertensive nephropathy in approximately half of patients were the most common causes of ESRD.[15]

Patients with angiographically proven stenosis (>50% of the luminal diameter), acute coronary syndromes, history of myocardial infarction, chronic stable angina pectoris, previous coronary revascularization, regional wall-motion abnormalities in echocardiography, and significant ECG changes suggestive of myocardial ischemia were excluded from the study. One hundred and twenty-nine patients met these criteria and were enrolled for the study. Nineteen patients (15%) died before the follow-up troponin assays and echocardiographic examinations. The causes of death were cardiovascular (50%), infection, and other/unknown causes. This study was performed on the remaining 110 patients, for whom follow-up echocardiographic and troponin assessments were available. The baseline LVMI of deceased patients was significantly higher than these 110 patients included in the analysis (170 ± 54 vs. 140 ± 58, $p = 0.036$). The incidence of death was significantly higher in the patients with an elevated cTnT and cTnI. Follow-up echocardiography and troponin assays were performed after 18.2 ± 2.2 (14–23) months.

Blood Sampling Protocol and Echocardiographic Examination

Blood samples were collected for complete analysis and then centrifuged, and aliquots were stored at −70°C for subsequent measurements of troponins. The cTnT was measured using the Elecsys System 2010, an enzyme-linked immunosorbent assay (third generation) from Roche Diagnostics (Mannheim, Germany). We used a level of 0.1 ng/mL as the cTnT positivity. cTnI was measured using the Axsym System, a microparticle enzyme immunoassay from Abbott Laboratories (Chicago, Illinois, USA). The cutoff value chosen for an abnormal cTnI was 0.5 ng/mL.

Two-dimensional echocardiography was performed on patients in the left lateral decubitus position with a Vingmed system V (Norway, GE). Because LV volume fluctuates in hemodialysis patients, measurements were performed after dialysis, when patients were closest to their euvolemic state. Two dimensional measurements were performed as recommended by the American Society of Echocardiography.[16] Values were indexed to body surface area. LV ejection fraction (LVEF) was determined by the modified biplane Simpson's rule. Measurements were averaged over three cardiac cycles. The LV mass (LVM) was calculated according to Devereux formula[17]:

$$\text{LV mass (gr)} = 0.8 \times 1.04\,[(\text{PWT} + \text{IVS} + \text{LVEDD})^3 - (\text{LVEDD})^3] + 0.6;,$$

where PWT is posterior wall thickness, IVS is ventricular septal wall thickness, and LVEDD is left ventricular end-diastolic dimension. LVM index (LVMI) was calculated as LVM/body surface area. LVH was defined as LVMI >134 gr/m^2 in men and > 110 gr/m^2 in women.[18]

At the end of the follow-up period, cardiac troponins and echocardiographic measurements were obtained from all of the patients again. All measurements were performed by the two cardiologists unaware of the original patient troponin results. The study was conducted in accordance with the Helsinki II declaration and approved by the Local Ethics Committee. All participants gave a written informed consent.

Statistical Analysis

Continuous variables were expressed as mean values ± SD, and categorical variables as percentages. Categorical variables were compared by Pearson chi-square statistic. Comparisons of continuous variables between two groups have been performed by means of paired or unpaired

Student's t test, Wilcoxon signed ranks test, or Mann-Whitney U test, where appropriate. The differences in continuous variables between three or more groups were performed by analysis of variance or Kruskal Wallis test. For all tests, a value of $p < 0.05$ was considered statistically significant. The SPSS statistical software package (version 10.0) was used to perform all statistical calculations.

RESULTS

Baseline clinical characteristics of all the patients are shown in Table 1. Baseline and follow-up echocardiographic measurements are shown in Table 2. There was no statistically significant difference between echocardiographic measurements at the baseline and after the follow-up.

At the baseline, cTnT was positive in 19 patients (17%). LVMI was significantly higher in patients with positive baseline cTnT than those patients with negative cTnT (184.6 ± 51.8 gr/m^2 vs. 130.8 ± 54.6 gr/m^2, $p < 0.001$). cTnI was positive in 27 patients (25%). LVMI was also significantly higher in patients with positive cTnI than those patients with negative cTnI (160 ± 73.9 gr/m^2 vs. 133 ± 45 gr/m^2, $p = 0.031$).

After the follow-up, cTnT was positive in 18 patients (16%). In patients with positive cTnT, LVMI was significantly higher than those in patients with negative cTnT (180 ± 46 gr/m^2 vs. 135 ± 58 gr/m^2, $p = 0.002$). cTnI was positive in 48 patients (44%). Although, LVMI was higher in patients with positive cTnI, it did not reach statistical significance (153 ± 67 gr/m^2 vs. 134 ± 50 gr/m^2, $p > 0.05$).

Table 3 shows the baseline and follow-up LVMI according to positivity and negativity of both troponins at the baseline and follow-up. LVMI was significantly higher in patients with both cTnT and cTnI positive at the follow-up than those patients with both cTnT and cTnI negative at the baseline.

The change in the troponins and LVMI from baseline to follow up was given in Table 4. After the follow-up, cTnT changed from negative to positive in 7 patients (6%), and from positive to negative in 8 patients (7%). LVMI significantly increased in patients in which cTnT changed from negative to positive at follow-up (136 ± 41 gr/m^2 vs. 174 ± 48 gr/m^2, $p = 0.028$). Although, LVMI decreased from 204 ± 63 gr/m^2 to 164 ± 33 gr/m^2 in patients in which cTnT changed from positive to negative at follow-up, it did not reach statistical significance. LVMI did not significantly change from baseline to follow-up in other two groups. cTnI changed from negative to positive in 33 patients (30%), and from positive to negative in 12 patients (11%). In contrast to cTnT, LVMI did not increase in patients in which cTnI changed from negative to positive at follow-up. There were five patients in which both troponins were negative at baseline and changed to positive at follow-up. LVMI also significantly increased in these patients.

Table 1
Baseline characteristics of patients with chronic renal failure

Patients (n)	110
Age (years)	43 ±14
Female/male (n, %)	50/60, (45/55%)
Hypertension	75 (68%)
Diabetes mellitus	19 (17%)
Smoking	24 (22%)
BMI (kg/m^2)	24
Hemoglobin level (mg/dL)	11.8 ± 2
Total cholesterol (mg/dL)	185 ± 80
Triglyceride (mg/dL)	122 ± 40
HDL (mg/dL)	35 ± 15
LDL (mg/dL)	135 ± 32
Dialysis (n, %)	
Piriton dialysis	41 (40%)
Hemodialysis	44 (37%)
Predialysis	25 (23%)

Abbreviations: BMI = body mass index, HDL = high-density lipoprotein, LDL = low-density lipoprotein

Table 2
The echocardiographic parameters in the baseline and follow-up of patients

	Baseline	Follow-up	*p* value
LV diastolic dimension (cm)	5.1 ± 0.6	5.2 ± 0.7	NS
LV systolic dimension (cm)	3.6 ± 0.7	3.7 ± 0.8	NS
EF (%)	64 ± 10	60 ± 11	NS
FS (%)	29 ± 6	27 ± 7	NS
Aortic dimension (cm)	3.1 ± 0.3	3.1 ± 0.3	NS
Left atrial dimension (cm)	3.9 ± 0.6	3.9 ± 0.7	NS
Mitral E wave (cm/sn)	0.87 ± 0.26	0.82 ± 0.26	NS
Mitral A wave (cm/sn)	0.87 ± 0.30	0.78 ± 0.32	NS
E dec (msn)	220 ± 61	215 ± 65	NS
IVRT (msn)	89 ± 17	88 ± 20	NS
Septal thickening (cm)	1.1 ± 0.2	1.1 ± 02	NS
Posterior wall thickening (cm)	1.0 ± 0.2	1.1 ± 02	NS
LVM (g)	236 ± 97	242 ± 103	NS
LVMI (g/m^2)	140 ± 58	142 ± 58	NS

Data were presented as mean ± SD.

Abbreviations: LV = left ventricle, EF = ejection fraction, FS = fractional shortening, IVRT = iso-volumetric relaxation time, LVM = left ventricular mass, LVMI = left ventricular mass index, SD = standard deviation, NS = not significant

Table 3
LVMI values according to both cardiac troponins in baseline and follow-up

	Baseline			Follow-up		
	n (%)	LVMI (gr/m^2)	*p* value	n (%)	LVMI (gr/m^2)	*p* value
Group I	73 (67%)	128 ± 47†		57 (52%)	134 ± 52‡	0.006*
Group II	9 (8%)	198 ± 51	0.001*	13 (12%)	196 ± 46	
Group III	18 (16%)	142 ± 77		35 (32%)	137 ± 67§	
Group IV	10 (9%)	172 ± 51		5 (4%)	140 ± 24	

Data were presented as mean ± SD. Abbreviations are the same as those in Table 2.
Group I = cTnT and cTnI negative; Group II = cTnT and cTnI positive; Group III = cTnT negative and cTnI positive; Group IV = cTnT positive and cTnI negative
*ANOVA analysis, between-groups
$^{\dagger}p < 0.005$, group I versus group II; $^{\ddagger}p < 0.007$, group I versus group II; $^{\S}p < 0.021$, group III versus group II.

Table 4
LVMI values and fluctuations of cardiac troponins in baseline and follow-up

				LVMI (gr/m^2)		
	n (%)	Baseline	Follow-up	Baseline	Follow-up	*p* value
cTnT	84 (76%)	0	0	130 ± 55	132 ± 59	0.351
	7 (7%)	0	+	136 ± 41	174 ± 48	0.028*
	11 (10%)	+	+	169 ± 38	184 ± 50	0.155
	8 (7%)	+	0	204 ± 63	164 ± 33	0.123
cTnI	50 (45%)	0	0	132 ± 55	130 ± 48	0.472
	33 (30%)	0	+	135 ± 40	140 ± 48	0.386
	15 (14%)	+	+	173 ± 76	182 ± 92	0.281
	12 (11%)	+	0	144 ± 70	151 ± 57	0.583
cTnT and cTnI	42 (76%)	0	0	126 ± 46	127 ± 51	0.488
	5 (9%)	0	+	149 ± 40	194 ± 42	0.043*
	5 (9%)	+	+	192 ± 37	215 ± 15	0.225
	3 (6%)	+	0	210 ± 85	170 ± 38	0.285

Data were presented as mean ± SD. Abbreviations are the same as those in Table 2. (+) = positive, (0) = negative.
*$p < 0.05$, non-parametric test-Wilcoxon test.

DISCUSSION

The findings of results in this study are as follows:

- cardiac troponins are shown to have significantly fluctuations in patients with ESRD without known coronary artery disease;
- patients whose troponin T level was changed from negative to positive with time have more prominent increased LVMI; and
- although there was significant fluctuation (from negative to positive) in level of cTnI with time, an increase of LVMI was not statistically significant in these patients.

Cardiac troponins are the most important of biochemical markers for the detection of myocardial cell damage. It has been reported that elevated cardiac troponins had an important role in predicting the mortality in patients with ESRD.[8,19,20] It is well accepted that elevated serum cTnT concentrations in ESRD patients, measured by second- or third-generation cTnT assays, originate from the heart and not from the skeletal muscle. We used third-generation cTnT and cTnI assays that do not show cross-reactivity with skeletal muscle troponin isoforms in patients with ESRD. However, the fluctuation of cardiac troponins with time and its relationship with LVMI in patients with ESRD is not clear yet despite using new generation assays. However, previous studies have generally used a single measurement of cardiac troponins.[13,14,21,22] Fluctuations in the level of the cardiac troponins can appear with time.[19] Similar to our results, Robert et al.[23] have been reported that undetectable cTnI in 27% of patients at baseline was found to be detectable

in the serial follow-up. We found that fluctuation of cTnI was higher than the fluctuation of cTnT.

An increase of LVMI was more prominent in patients who had fluctuation in cardiac troponins from negative to positive (especially cTnT, as well as both cTnT and cTnI). Therefore, an increase of LVMI may be partly responsible for the changes of cardiac troponins in ESRD over time. The presence of left ventricular hypertrophy, which is an important predictor of cardiovascular mortality, is significantly correlated with elevated cTnT in patients with ESRD without acute myocardial ischemia.[5,6,8] It has been suggested that this association is explained by an elevated cTnT release from the hypertrophic cardiomyocyte due to impaired membrane permeability.[13] In addition, an increase of the myocardial fibrosis and myocyte death or decrease in the capillary density caused by LVMI may explain the elevated troponins without coronary artery disease in this population.

In contrast to fluctuation in cTnT, there was more evident fluctuation in cTnI in our study; however, an increase of LVMI with time was not statistically significant in these patients. Discrepancy between fluctuations of cTnT and cTnI at follow-up is not entirely clear, but may be explained by releasing kinetics, cellular biology, protein chemistry, and assay technology between the two troponins.[9,24,25] On the other hand, this may be attributable to instability of the cTnI molecule or dissimilar glomerular filtration of cTnT and cTnI.[26,27] In addition, recurrent myocardial microinfarction and progression of degenerative myocardial disease may be the cause of excessive fluctuations in the cTnI, which is more cardiac specific than cTnT in the setting of renal failure.[18,28,29] However, further studies are needed to investigate the cause of these different fluctuations between cardiac troponins in patients with ESRD.

LIMITATIONS

Coronary angiography was not performed to exclude CAD; a history of preexisting CAD would likely exclude some electrocardiographic and echocardiographic findings. Some patients probably had subclinical CAD. In addition, different dialysis modalities and pre-dialysis situations might have a different impact on both troponin levels and ventricular mass/morphology.

We conclude that change of troponins from negative to positive with time may be a marker of the increase of LVMI in the patients with ESRD. In light of the current data, we can conclude that cTnI is a more sensitive and specific marker than cTnT for diagnosing myocardial injury due to increased left ventricular mass. Therefore, cardiac troponins, especially cTnI, should be closely monitored in patients with ESRD.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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