Nephrol Dial Transplant (2009) 24: 1568–1573
doi: 10.1093/ndt/gfn730
Advance Access publication 14 January 2009

Prognostic value of elevated cardiac troponin I in ESRD patients with sepsis

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Abstract

Background. Elevated cardiac troponin (cTn) levels have been reported to predict adverse cardiovascular outcomes in asymptomatic ESRD patients. However, the prognostic value of elevated cTn levels associated with sepsis in ESRD patients is unknown. Therefore, this study aimed to elucidate the clinical implications of elevated cTnI levels in ESRD patients with sepsis.

Methods. Of the 305 ESRD patients in whom cTnI was measured between January 2003 and December 2005, sepsis developed in 121 patients during follow-up. Based on cTnI levels at the onset of sepsis, patients were classified as elevated cTnI group (ET, $n = 50$, >0.2 ng/ml) and lower cTnI group (LT, $n = 71$, ≤ 0.2 ng/ml). Study endpoints were short- and long-term mortality. Short-term mortality was defined as death occurring within 90 days after sepsis, and patients who survived during this period were followed till death after 90 days.

Results. Before sepsis, the median concentration of cTnI was 0.05 (0.01–3.59) ng/ml and it was significantly increased to 0.11 (0.01–22.0) ng/ml when sepsis supervened ($P < 0.01$). Compared to the LT group, the short-term mortality rate was significantly higher in the ET group ($P < 0.05$). After adjustment for age, diabetes, serum albumin and CRP levels, presence of shock and previous cardiovascular disease history, the ET group had a greater odds ratio of short-term mortality (OR 5.13, $P < 0.01$). In addition, the Kaplan–Meier plot for long-term survival revealed a significantly higher mortality rate in the ET group. In a multivariate Cox regression analysis, the elevation of cTnI levels was an independent determinant for long-term mortality (HR 5.90, $P < 0.01$).

Conclusion. This study showed that elevated cTnI levels were significantly associated with short- and long-term mortality in ESRD patients with sepsis. Therefore, elevated cTnI levels in these patients should not be overlooked and be followed for adverse outcomes.

Keywords: cardiac troponin I; ESRD; sepsis

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Introduction

Cardiovascular events and infection are the leading causes of death in patients with end-stage renal disease (ESRD) [1]. It is generally accepted that inflammation has an important role not just in the pathogenesis of atherosclerosis but also in the initiation of acute coronary syndrome (ACS) [2]. Uraemia *per se* is considered as chronic microinflammatory status, and inflammatory markers have been demonstrated to be independent predictors in dialysis patients [3]. Sepsis is also an inflammatory state and may exert a synergistic effect on inflammation and atherosclerosis. In the general population, acute lower respiratory infection and acute urinary tract infection were associated with an increase in myocardial infarction or stroke [4]. In addition, sepsis with bacteraemia was associated with cardiovascular morbidity and mortality in patients with ESRD [5]. Cardiac troponins (cTns) are biomarkers that are previously introduced for diagnosis and risk stratification in patients with ACS [6]. Serum cTn levels are elevated in 31–85% of critically ill patients with sepsis [7,8]. The elevation of cTn levels accompanied by sepsis and septic shock has been reported to be associated with left ventricular dysfunction and poor prognosis in the general population [8,9].

Elevated cTn levels are common in asymptomatic ESRD patients, but their prognostic value is still under debate because it is often elevated in the setting of even mild degrees of renal failure without ACS [10]. Several studies revealed a low specificity of cTn for the assessment of ACS in dialysis patients [11,12]. Despite these limitations, elevated cTn levels were predictive for cardiovascular events and mortality in asymptomatic ESRD patients [13–15]. However, their prognostic value combined with sepsis in ESRD patients is currently unknown. Therefore, we undertook this study to elucidate the clinical implications of elevated cTnI levels in asymptomatic ESRD patients with sepsis.

Patients and methods

Study population and data collection

This is an observational cohort study with 305 ESRD patients who had been treated with haemodialysis (HD)

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or peritoneal dialysis (PD) between January 2003 and December 2005. During this period, cTnI concentrations were measured as a surveillance test for cardiovascular risk assessment. These patients were followed, and sepsis developed in 127 patients. Of these, six patients in whom cardiovascular events occurred during the follow-up period were excluded. Therefore, a total of 121 patients were included in the analysis. All patients were older than 18 years and were maintained on dialysis therapy for >3 months. Sepsis is defined as the systemic inflammatory response to infection. In association with infection, it is manifested by two or more of the following conditions: (1) temperature >38°C or <36°C; (2) heart rate >90 beats/min; (3) respiratory rate >20 breaths/min or PaO_2 <32 mmHg and (4) white blood cell (WBC) count >12 000/mm^3, <4000/mm^3 or >10% immature (band) forms.

Demographic data and clinical parameters were collected with a retrospective review of medical records: age, gender, diabetes, dialysis modality, dialysis duration, previous cardiovascular disease (CVD) history, primary cause of sepsis, bacteraemia and presence of septic shock. Septic shock was defined as acute circulatory failure (systolic blood pressure <90 mmHg, mean arterial pressure <65 mmHg or a reduction in systolic blood pressure <40 mmHg from the baseline) despite adequate volume resuscitation. In addition, baseline laboratory data for WBC count, haemoglobin, platelet count, serum albumin, C-reactive protein (CRP), CK-MB and cTnI were collected at the onset of sepsis.

Measurement of cTnI concentrations

The cTnI levels were measured by means of a one-step enzyme immunoassay based on the sandwich principles (AccuTnI, Beckman Coulter, Inc., USA). The detection limit was 0.01 mg/l, and there was an interassay coefficient of variation of 10% at 0.06 ng/ml. As described above, we obtained data for cTnI that were measured before sepsis as well as at the onset of sepsis. The mean duration between the two periods was 11.2 ± 8.9 months. Patients were divided into elevated cTnI group (ET, $n = 50$, >0.2 ng/ml) and lower cTnI group (LT, $n = 71$, ≤0.2 ng/ml) according to cTnI levels at the onset of sepsis.

Study endpoints

The date and cause of death were obtained by reviewing the hospital records. Study endpoints were short- and long-term mortality. Short-term mortality was defined as all-cause death occurring within 90 days after the onset of sepsis. Patients who survived during this period were followed for the development of death from any cause after 90 days, and these patients were subjected to long-term survival analysis. In addition, cardiovascular deaths that were caused by myocardial infarction, congestive heart failure or sudden cardiac death were further assessed for the long-term survival analysis.

Statistical analysis

All data were expressed as mean ± SD; however, data with skewed distribution were expressed as median with range.

Table 1. Patient characteristics

Number of patients	121
Age (years)	66.1 ± 11.8
Sex (male/female)	53/68
DM, *n* (%)	82 (67.8%)
Dialysis duration (months)	25.3 ± 28.3
Dialysis modality (HD/PD)	61/60
Previous CVD history, *n* (%)	33 (27.2%)
CAOD, *n* (%)	22 (18.0%)
CVA, *n* (%)	17 (13.9%)
SBP (mmHg)	137.5 ± 34.7
DBP (mmHg)	77.1 ± 18.0
MAP (mmHg)	97.3 ± 27.4
Septic shock, *n* (%)	30 (24.8%)
Types of infection	
Pneumonia	80
UTI	14
CAPD peritonitis	11
DM foot	5
Others	14
Bacteraemia, *n* (%)	48 (39.7%)
WBC (/mm^3)	11 591 ± 5942
Haemoglobin (g/dl)	9.6 ± 1.7
Serum albumin (g/dl)	2.6 ± 0.7
Platelet (×10^3/mm^3)	194 ± 95
CRP (mg/dl)	10.04 ± 8.89
cTnI (ng/ml)	0.11 (0.01–22.0)[a]
CK-MB (ng/ml)	1.7 (0.50–191)[a]

DM, diabetes; CVD, cardiovascular disease; CAOD, coronary artery occlusive disease; CVA, cerebrovascular attack; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean artery pressure; UTI, urinary tract infection; CAPD, continuous ambulatory peritoneal dialysis; CRP, C-reactive protein; HD, haemodialysis; PD, peritoneal dialysis; WBC, white blood cells.
Data were expressed as mean ± SD.
[a]These data were expressed as median with range

The comparisons between the two groups were made by Student's *t*-test and chi-square test. For analysis of short-term mortality, logistic regression analysis was used. The patient survival was analysed by Kaplan–Meier survival analysis, and Cox proportional hazards regression models were used to determine relationships between cTnI levels and long-term mortality outcome. Covariates with a *P*-value of <0.10 in univariate analyses were subjected to multivariate Cox regression analysis. Hazard ratios (HR) and 95% confidence intervals (CI) for long-term mortality were obtained. All probabilities were two tailed, and the level of significance was set at 0.05. All analyses were conducted using SPSS version 13.0 (SPSS Inc., Chicago, IL, USA).

Results

Patient characteristics

Table 1 details clinical characteristics of the 121 patients. The mean age was 66 years, and 43.8% were male. Diabetes (67.8%) was the most common cause of ESRD. The mean dialysis duration was 25.3 ± 28.2 (range 3–148) months, and the mean follow-up duration was 19.1 ± 18.9 months. Of the subjects, 61 patients were treated with HD and 60 with PD. A previous CVD history was present in 33 patients (27.2%): 22 patients (18.2%) with coronary artery disease and congestive heart failure and 17 patients (14.1%) with cerebrovascular events. The most common type of infection

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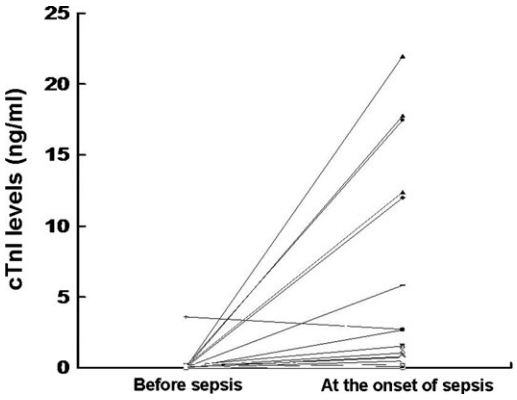

Fig. 1. Changes of cTnI levels from the first measurement of cTnI to the onset of sepsis. Prior to the onset of sepsis, the median concentration of cTnI was 0.05 (0.01–3.59) ng/ml and it was significantly increased to 0.11 (0.01–22.0) ng/ml when sepsis supervened.

was pneumonia (66.1%) followed by urinary tract infection (11.6%). Forty-eight patients (39.7%) had bacteraemia, and septic shock was evident in 30 patients (24.8%). The most common causative micro-organism was staphylococcus aureus (28.3%).

Change of cTnI levels with sepsis

Before sepsis, the median concentration of cTnI was 0.05 (0.01–3.59) ng/ml and it was significantly increased to 0.11 (0.01–22.0) ng/ml when sepsis supervened (Figure 1, $P < 0.01$). Elevated cTnI levels at the onset of sepsis compared to the levels before sepsis were found in 88 patients (72.7%), and cTnI levels >0.2 ng/ml were observed in 50 patients (41.3%).

Comparison between patients with elevated cTnI levels and those with lower cTnI levels

When the patients were divided into the two groups on the basis of cTnI concentrations at the onset of sepsis, there were no significant differences in age, gender, diabetes, previous history of CVD, dialysis duration, dialysis modality, blood pressure, mean arterial pressure and incidence of bacteraemia between the two groups (Table 2). However, patients with elevated cTnI levels had lower serum albumin levels (2.4 ± 0.7 versus 2.7 ± 0.6 g/dl, $P < 0.05$) and had higher CRP levels (12.6 ± 8.4 versus 8.3 ± 8.8 mg/dl, $P < 0.01$) and CK-MB levels (15.5 ± 34.0 versus 2.1 ± 2.1 mg/dl, $P < 0.01$) compared to those with lower cTnI levels. Also, septic shock was less common in patients with elevated cTnI levels (12.0% versus 33.8%, $P < 0.05$) (Table 2).

Elevated cTnI levels and short-term mortality

A total of 55 deaths occurred within 90 days after the onset of sepsis. Forty-four and seven deaths were attributable to sepsis and cardiovascular events, respectively. The mortality rate (60.0% versus 35.2%, $P < 0.01$) was significantly higher in patients with elevated cTnI levels compared to those with lower cTnI levels. In addition, patients with elevated cTnI levels had a greater odds of death [odds ratio (OR) 5.13, 95% CI 1.73–15.18, $P < 0.01$] in a multivariate logistic regression analysis adjusted for age, diabetes, previous CVD history, presence of septic shock, and serum albumin and CRP levels (Table 3). Besides cTnI, other factors that predicted the short-term mortality were old age (per 1-year increase, OR 1.05, 95% CI 1.03–1.14, $P = 0.001$), presence of diabetes (OR 3.00, 95% CI 1.09–8.25, $P = 0.033$) and septic shock (OR 8.00, 95% CI 2.21–28.94, $P = 0.002$), and lower serum albumin levels (per 1 g/dl increase, OR 0.17, 0.07–0.41, $P < 0.001$).

Table 2. Comparison between patients with elevated cTnI (ET) and those with lower cTnI (LT) levels

	LT group ($n = 71$)	ET group ($n = 50$)
Age (years)	66 ± 12	67 ± 12
Sex (male/female)	31/40	22/28
DM, *n* (%)	44 (62.0%)	38 (76.0%)
Dialysis duration (months)	28 ± 32	22 ± 24
Dialysis modality (HD/PD)	40/31	21/29
Previous CVD history, *n* (%)	17 (23.9%)	16 (32%)
CAOD, *n* (%)	11 (15.5%)	11 (22.0%)
CVA, *n* (%)	8 (11.3%)	8 (16.0%)
SBP (mmHg)	139.3 ± 34.3	135.0 ± 35.4
DBP (mmHg)	77.3 ± 18.6	76.8 ± 17.5
MAP (mmHg)	98.0 ± 22.4	96.2 ± 22.6
Septic shock, *n* (%)	24 (33.8%)	6 (12.0%)‡
Bacteraemia, *n* (%)	28 (39.4%)	20 (40.0%)
WBC (/mm^3)	10 750 ± 5307	12 776 ± 6548
Haemoglobin (g/dl)	9.9 ± 1.6	9.3 ± 1.6
Platelet (×10^3/mm^3)	199 ± 100	187 ± 89
Serum albumin (g/dl)	2.8 ± 0.6	2.5 ± 0.7†
CRP (mg/dl)	8.3 ± 8.8	12.5 ± 8.4‡
cTnI (ng/ml)[a]	0.04 (0.01–0.19)	0.80 (0.21–22.0)‡
CK-MB (ng/ml)[a]	1.50 (0.50–9.40)	5.15 (0.50–191.0)‡

DM, diabetes; HD, haemodialysis; PD, peritoneal dialysis; CVD, cardiovascular disease; CAOD, coronary artery occlusive disease; CVA, cerebrovascular attack; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean artery pressure; WBC, white blood cells; CRP, C-reactive protein.
[a]These data were expressed as median with range.
†$P < 0.05$, ‡$P < 0.01$ versus the LT group.

Table 3. Multivariate logistic regression analysis for short-term mortality[a]

	OR	95% CI	*P*-value
Age (per 1-year increase)	1.05	1.03–1.14	0.001
DM (versus non-DM)	3.00	1.09–8.25	0.033
Previous CVD history (versus none)	1.05	0.36–3.03	0.928
Septic shock (versus none)	8.00	2.21–28.94	0.002
Serum albumin (per 1 g/dl increase)	0.17	0.07–0.41	<0.001
CRP (per 1 mg/dl increase)	1.02	0.97–1.08	0.406
Elevated cTnI levels (versus ≤0.2 ng/ml)	5.13	1.73–15.18	0.003

[a]Adjusted for age, diabetes, previous CVD history, presence of septic shock, serum albumin and CRP levels and elevated cTnI levels.

Elevated cTnI levels and long-term mortality

The relationship between cTnI levels and long-term mortality was further assessed in 66 patients who survived sepsis and other complications during 90 days after the onset of sepsis. Of the patients, 20 patients (30.3%) were in the ET group and the remaining 46 (69.7%) in the LT group. A total of 22 deaths occurred after 90 days, and patients in

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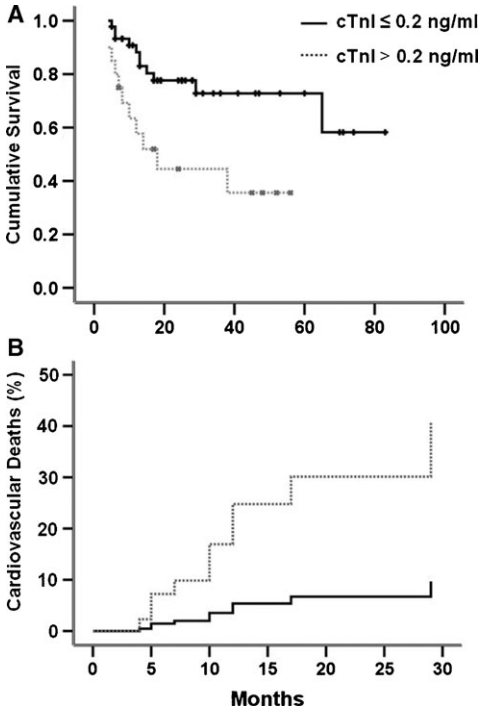

Fig. 2. Kaplan–Meier survival curve for long-term all-cause mortality (**A**) and cardiovascular mortality (**B**). The patient survival rate was significantly lower ($P < 0.01$) and cumulative cardiovascular mortality was significantly higher ($P < 0.05$) in patients with elevated cTnI levels compared to those with lower cTnI levels.

Table 4. Multivariate Cox proportional hazard models for long-term adverse outcomes

	HR	95% CI	*P*-value
All-cause mortality[a]			
Age (per 1-year increase)	1.04	1.00–1.08	0.050
DM (versus non-DM)	2.38	0.87–6.50	0.090
Previous CVD history (versus none)	3.57	1.28–9.94	0.015
Septic shock (versus none)	2.98	0.87–10.25	0.083
CRP (per 1 mg/dl increase)	0.99	0.93–1.05	0.620
Elevated cTnI levels (versus ≤0.2 ng/ml)	5.90	2.06–16.87	0.001
Cardiovascular mortality[b]			
Age (per 1-year increase)	1.01	0.96–1.06	0.674
Diabetes (versus non-DM)	0.95	0.18–5.08	0.952
Previous CVD history (versus none)	6.01	1.43–25.19	0.014
Elevated cTnI levels (versus ≤0.2 ng/ml)	5.17	1.16–23.16	0.032

[a]Adjusted for age, diabetes, previous CVD history, presence of septic shock, serum CRP levels and elevated cTnI levels.
[b]Adjusted for age, diabetes, previous CVD history and elevated cTnI levels.

the ET group had a significantly higher mortality rate than those in the LT group (55.0% versus 23.9%, $P < 0.05$). The Kaplan–Meier plot for long-term survival revealed that patient survival was significantly lower in patients with elevated cTnI levels (Figure 2A, $P < 0.01$). In addition, in a multivariate Cox regression analysis adjusted for age, diabetes, presence of septic shock, previous CVD history and serum CRP levels, patients in the ET group had a higher risk of death (HR 5.90, 95% CI 2.06–16.9, $P < 0.01$) compared to those in the LT group (Table 4). For long-term cardiovascular mortality 90 days after the onset of sepsis, the elevation of cTnI levels was identified as a significant determinant (HR 5.17, 95% CI 1.16–23.2, $P < 0.05$) after adjustment of age, diabetes and previous CVD history (Figure 2B, Table 4).

Discussion

Recently, the importance of cardiac biomarkers has been highlighted because CVDs are the most common cause of death in ESRD patients [13,16]. CTnT and cTnI are two of the clinically available biomarkers whose clinical value has been validated not only in non-CKD patients but also in CKD patients [13]. Although a number of literatures have reported that the elevated cTn level is an independent predictor of cardiovascular events and mortality in ESRD patients [13–18], its utility in this population with septic condition has never been demonstrated. This study showed that sepsis could trigger or aggravate myocardial injury, and the elevated cTnI level associated with sepsis was a significant determinant of short- and long-term adverse outcomes in ESRD patients.

The elevated cTnT level as a risk factor for sepsis-related death in patients undergoing HD was previously suggested by Hocher *et al*. [19]. They measured the cTnT level at baseline and followed the patients for 2 years after blood sampling. Interestingly, in that study, the elevated cTnT level at baseline was already predictive of sepsis-related mortality later on. The authors did not give a clear explanation on why such cardiac biomarker measured even when sepsis was not evident could predict infection-related adverse outcomes. In contrast, our study revealed that the elevated cTnI level at the onset of sepsis was a significant determinant of sepsis-related mortality whereas its level at the study entry was not (data not shown). It should be noted that our study is different from the study by Hocher *et al.* in that they did not provide change of cTnT level because cTnT was measured only once at baseline. We clearly showed change of cTnI level by measuring it both at the study entry and at the onset of sepsis. Therefore, it is reasonable to presume that the cTnI levels were significantly affected by sepsis. In addition, cTnI levels >0.2 ng/ml measured at the study entry were observed in only 6% of patients. This may give low statistical power to predict adverse outcomes. Whether cTn measured prior to sepsis can predict infection-related death later on remains to be further investigated.

Among many other cardiac biomarkers that can predict adverse outcomes, such as CRP, interleukin-6, intercellular adhesion molecule-1, etc, cTnI is of our special interests because such markers are nonspecific in the inflammatory process and can be confounded by infectious diseases [20]. In addition, cTnI was preferred in this study due to lack of expression of cTnI in noncardiac tissue [21]. It is well known that troponin T isoforms can be released from noncardiac tissue such as skeletal muscle [22]. In septic shock, hypoperfusion causes ischaemia in skeletal muscle and this may consequently lead to false elevation of cTnT levels.

Myocardial dysfunction is common in patients with sepsis. Ver Elst *et al*. reported that 50% of patients with sepsis and septic shock had elevated cTnI levels, and 78% of them had LV dysfunction assessed by echocardiography [23]. In addition, an elevated cTnT or cTnI concentration has been

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suggested as a significant risk factor for myocardial dysfunction and adverse outcomes in septic patients without ACS [7,8,23–28]. Although its prognostic value in patients with sepsis has been reported, the underlying mechanism of cTnI elevation in sepsis is poorly understood. Microinjury of myocardial cells in sepsis may be attributable to several factors. These include endotoxins, inflammatory cytokines or reactive oxygen radicals induced by an infectious process that may have a direct cardiac myocytotoxic effect. In addition, inflammatory cytokines such as tumour necrosis-α and interleukin-1 are reported to be cardiodepressants. Myocardial injury due to microvascular thrombosis can also play a role. It is well known that there is a close relationship between the presence of inflammatory cytokines and a procoagulant state in patients with severe sepsis [29]. Inflammatory cytokines, including tumour necrosis factor-α, interleukin-1β and interleukin-6, are capable of activating coagulation and inhibiting fibrinolysis, whereas the procoagulant thrombin is capable of stimulating multiple inflammatory pathways [30]. In this context, the possibility of small-vessel thrombosis with subsequent myocardial microinfarction and troponin release is possible. Taken together, these findings suggest that sepsis may amplify their systemic inflammation cascade and aggravate myocardial ischaemia. In addition, most ESRD patients are predisposed to be volume overloaded and hypertensive, resulting in myocardial stretch [16]. This eventually leads to left ventricular hypertrophy and subsequent cTnI release from injured myocardium even in patients without ACS. Therefore, it can be speculated that ESRD patients are more susceptible to myocardial injury that is precipitated by sepsis-associated tachycardia and anaemia.

Other factors that can potentially contribute to the cTnI elevation are use of aggressive inotropic agents and severe hypotensive episode due to septic shock, thus leading to ischaemic injury to myocardial cells [24]. However, in this study, inotropics were used only in 24.8% and severe hypotensive episodes (systolic blood pressure <90 mmHg) were observed only in 10% of patients. In addition, septic shock was less common in patients with elevated cTnI levels (12.0% versus 33.8%, $P < 0.05$). Therefore, it is unlikely that such factors are major contributors to the cTnI elevation. In addition, it has been reported that patients with previous CVD already have higher serum cTn levels than those without a history of CVD [31]. We excluded the 33 patients (27.2%) who experienced CVD prior to the study entry and reanalysed the data. As a result, the elevation of cTnI concentrations was a significant predictor of all-cause mortality in a multivariate Cox regression analysis adjusted for age, diabetes, shock status and CRP levels (data not shown).

One of the confounding factors that can affect cTn levels is residual renal function. Serum concentrations of cTn are variable depending on renal impairment, and it is often elevated in patients with CKD who have no evidence of ACS. However, it has been suggested that its prognostic value for CVD remains whatever the effect of renal clearance, although the degree of cTn elevation may be confounded by renal impairment [32]. This study includes both HD and PD patients. It is generally acknowledged that residual renal function is more preserved in patients treated with PD. Therefore, it is possible that cTnI concentrations may differ according to dialysis modalities and residual renal function should be considered when interpreting our results. However, there is no difference in residual renal function between patients treated with HD and PD and the proportion of patients receiving HD or PD was not different between the ET and the LT group. In addition, when analysed according to HD and PD patients, respectively, the elevation of cTnI concentrations still remains as an independent risk factor for mortality in HD patients as well as PD patients (data not shown).

In this study, the cut-off value of cTnI was defined as 0.2 ng/ml. This was based on the results of receiver operating curve (ROC) analysis that revealed that this value provides the best diagnostic accuracy for short-term mortality and long-term mortality compared to the cut-off value of 0.1 ng/ml. For short- and long-term mortality, the area under the curve (AUC) when the cut-off value was defined as 0.2 ng/ml was 0.629 and 0.645, respectively, whereas it was 0.582 and 0.563 when defined as 0.1 ng/ml. This finding was also compatible with ROC analysis of the long-term cardiovascular mortality as AUC with the cut-off value of 0.2 ng/ml was higher compared to that of 0.1 ng/ml (0.675 versus 0.640). However, the prognostic value of cTnI in our subjects was relatively lower because all AUCs were <0.7. This can partly be explained by the fact that most deaths were caused by sepsis itself, not by CVDs.

Several shortcomings in this study should be discussed. First, this is an observational study with a small sample size. As per an observational study design, all data were dependent on medical records, which may not represent a precise patient status. In addition, a small sample size may lead to selection bias. Second, echocardiography or coronary angiography was performed only in small number of patients; thus we could not provide data pertinent to LV function and coronary artery status. In addition, we obtained data for cTnI levels only at two time points because cTnI levels were not measured serially in the meantime. This may not reflect a precise change of cTnI levels between the two periods. However, only six patients experienced cardiovascular events from the measurement of cTnI to the study entry and these patients were excluded in the analysis. Therefore, chances of the elevation of cTnI levels by factors other than sepsis were minimized. Finally, lower AUC from the ROC analyses is another shortcoming that may provide less diagnostic accuracy. Despite these limitations, it should be noted that elevated cTnI levels in ESRD patients with sepsis were significantly associated with short- and long-term mortality in this study.

In conclusion, our findings suggest that in ESRD patients, sepsis-associated cTnI elevation may have the predictive value for short- and long-term mortality. Therefore, elevated cTnI levels in these patients should not be overlooked and be followed for adverse outcomes.

Conflict of interest statement. None declared.

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Received for publication: 13.10.08
Accepted in revised form: 5.12.08

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