ORIGINAL ARTICLE

Association of cardiac troponin I level with in-hospital and late mortality in dialysis patients

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Background: Cardiovascular diseases (CVDs) are highly prevalent among the end-stage renal disease (ESRD) patients. Prognostic value of cardiac troponin I (cTnI) in patients with asymptomatic ESRD is less conclusive. This study was an observational study to evaluate correlation of first admitted cTnI level with early and late (during 6 months) hospitalization and mortality of ESRD patient admitted due to non-acute coronary and non-heart failure causes in ESRD patients. **Materials and Methods:** In this prospective observational study, 460 dialysis patients without overt CVD who were admitted at two university hospital were included and followed during 6 months. Patients' demographic information and laboratory investigations including cTnI level and cause of admission were recorded. The association between cTnI level with in-hospital and late mortality was evaluated. **Results:** cTnI level was higher in female (35.9%), hemodialysis patients (28.1%), and patients with permanent catheter vascular access (29.4%). There were significant differences in level of triglyceride (TG), low-density lipoprotein (LDL) cholesterol, and high-density lipoprotein (HDL) cholesterol between patients with normal and abnormal cTnI levels ($P < 0.05$). Patients with abnormal cTnI levels had higher level of TG and LDL cholesterol and lower level of HDL cholesterol. cTnI levels were associated with higher in-hospital and 6-month follow-up mortality rate. In logistic regression analysis, only female gender (odds ratio [OR] =1.89, confidence interval [CI] =1.22–3.076) and TG (OR = 1.007, CI = 1.003–1.01) were positively and HDL cholesterol level (OR = 0.994, CI = 0.98–0.99) was negatively associated with increased cTnI level. cTnI level was associated with early (OR = 4.81, CI = 1.64–14.89) and late (OR = 4.31, CI = 1.61–10.96) mortality. **Conclusion:** Although in this study, cTnI level is not directly associated with cardiovascular disorders and admission and readmission causes, it is a strong predictor of early and late mortality.

Key words: Cardiac diseases, cardiac-specific troponins, end-stage renal disease

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INTRODUCTION

Cardiovascular diseases (CVDs) are highly prevalent among the end-stage renal disease (ESRD) patients.[1,2] In hemodialysis (HD) patients, mortality relating to CVD is as much as 50%, and mortality after myocardial infarction (MI) is 52% at 2 years.[3]

It considers, as the main cause of death among this population.[4] However, diagnosis of CVDs in ESRD patients is challenging, as they are usually asymptomatic.[5] Moreover, left ventricular hypertrophy and electrolyte imbalance that cause electrocardiogram (ECG) abnormalities make the ECG data useless.[6] Cardiac troponin I (cTnI, 209 amino acids, and molecular weight of 23,875 Dalton) above the reference cutoff point derived from general population are crucial parameters in diagnosis of acute MI.[7] Although cTnI is the preferred serum marker for diagnosis of CVDs,[8,9] it is elevated in ESRD patients, even without any acute coronary syndromes (ACSs).[10] Some other studies showed cTnI has a predictive role for morbidity and mortality in ESRD[11] and angina equivalents.[12]

Elevated level of cTnI may suggest subclinical myocardial injury. Prognostic value of cTnI in patients

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with asymptomatic ESRD is less conclusive.[13] However, cTnT was consistently associated with all causes of mortality and CVD events in patients with ESRD, but data are not as robust for cTnI.[12]

Therefore, this study was designed to evaluate correlation of first admitted cTnI level with early and late (during 6 months) hospitalization and mortality of ESRD patient admitted due to nonacute coronary and nonheart failure causes.

MATERIALS AND METHODS

Inclusion and exclusion criteria

All ESRD patients were admitted in two university hospitalsfor any causes except for acute MI (ST-elevation MI [STEMI]) and decompensated heart failure (DHF) including presence of clinically acute pulmonary edema or severe HF (ejection fraction ≤30% in echocardiography) (overt CVDs) and had been cTnI level checked on time of admission entered in our study. All ESRD patients who admitted for surgical reasons were excluded from the study.

Study method

Demographic data including age, gender, history of smoking, cause of hospitalization, cause of original kidney disease, and type and time of dialysis were recorded.

From all patients, hemoglobin (Hb), fasting blood sugar (FBS), and cTnI level were measured on hospital admission. Lipid profile was recorded from patients' latest laboratory data. This study is a prospective observational study, which included 460 ESRD patients (more than 18 years old) on more than 3-month renal replacement therapy (HD or peritoneal dialysis) and admitted to two university hospitals in Isfahan, Iran, for 6 months until May 24, 2018.

All patients who were discharged from hospital were followed up in the end of 6 months. Patients who missed on follow-up due to unwillingness to cooperate were excluded from our study. cTnI level was measured in the hospitals laboratory with either VIDAS High Sensitivity Troponin I (Abbott Diagnostics Ireland, Longford co., Ireland)[14] or Immunochromatographic Toyo Troponin I Test (Türklab Tibbi Malzemeler San., Izmir, Turkey).[15] The 99th percentile reference range value of the VIDAS Troponin I Ultra kit is 26.2 pg/mL confidence interval (CI) =23.3–29.7 pg/mL. The ARCHITECT STAT High Sensitive Troponin-I assay is designed to have within-laboratory (total) imprecision of <10% coefficient of variation (CV) with controls or panels across the range of 10–50,000 pg/mL. A value of more than 30 pg/mL was considered to be elevated.

The normal level of the immunochromatographic kit was <0.5 ng/mL. cTnI level of more than 20 ng/mL was defined as elevated.

Follow-up

All the patients were followed up in the end of 6 months by researchers through the provided phone number. We justified them to inform the researchers, whether they had readmitted to the hospital, dead or not. If a patient did not admitted, follow-up continued to end of 6 months and patient's general health information was recorded. We used another checklist to record our follow-up data. The checklist included causes of hospital readmission such as ACS, MI, DHF, cerebrovascular accident, intracranial hemorrhage, arrhythmia, arterial-venous fistula (AVF), arterial-venous graft (AVG) and permanent catheter thrombosis and infection, the need for revascularization (pharmacological or surgical intervention), gastrointestinal bleeding, infectious diseases, respiratory failure, insufficient dialysis.

Statistical analysis

Patients' baseline characteristics were compared by the Chi-square test and independent *t*-test for parametric and the Mann–Whitney test for nonparametric variables. Logistic regression analysis was used to adjust for confounding parameters. Analyses were done using IBM, NY, USA SPSS23 PC software. $P < 0.05$ was considered statistically significant.

Ethical considerations

This observational study was a nephrology fellowship thesis (no: 297130) and approved by Isfahan Kidney Disease Research Center and Deputy of Research and Technology, Isfahan University of Medical Sciences (Ethical code IR.MUI.MED.REC.1399.016).

In this study, there was not any change in the treatment protocols. Moreover, all patients were treated according to the attending physician's practice. Patients' verbal consent was taken to use their medical data in our research study anonymously.

RESULTS

A total number of 460 ESRD on regular dialysis patients were entered the study. cTnI level was abnormal in 122 (27%) patients according to Table 1; abnormal cTnI level was more frequent in females 65 (35.9%) than males 57 (20.4%) patients ($P < 0.001$). Furthermore, there was a correlation between the cTnI level and the type of dialysis. Patients who underwent the peritoneal dialysis had lower cTnI compared to HD patients. Furthermore, patients with permanent catheter had significantly higher cTnI levels than patients with AVF/AVG vascular access for HD. While there was

Table 1: Association of demographic and laboratory parameters with cardiac troponin I level in our patients

Variables	Troponin I			*P* (variable)
	Normal	Abnormal	Total	
Sex				
Male	222 (79.6)**	57 (20.4)	279 (60.7)	<0.001
Female	116 (64.1)	65 (35.9)	181 (39.3)	
Age (year)*	55.64±17.12	57.25±18.48	56.07±17.38	0.38
Dialysis time (month)*	28±34.71	35.17±42.84	29.90±37.13	0.30
Type of dialysis				
Hemodialysis	291 (71.9)	114 (28.1)	405 (88)	0.03
Peritoneal	47 (88.8)	8 (13.2)	55 (12)	
Vascular access				
AV fistula/AV graft	72 (75.8)	23 (24.2)	95 (23.5)	0.03
Permanent catheter	219 (70.6)	91 (29.4)	310 (76.5)	
Smoking				
Yes	51 (82)	22 (18)	73 (15.9)	0.44
No	287 (75.1)	100 (24.9)	387 (84.1)	
Causes of renal failure				
DM				
Yes	132 (72.9)	49 (27.1)	181 (39.3)	0.83
No	155 (72.4)	59 (27.6)	279 (60.6)	
HTN				
Yes	183 (74.4)	63 (25.6)	246 (53.4)	0.63
No	24 (42.1)	190 (47.1)	214 (46.6)	
GN				
Yes	23 (76.7)	7 (23.3)	30 (6.6)	0.68
No	315 (73.3)	115 (26.7)	430 (93.4)	
Urologic				
Yes	20 (71.4)	8 (28.6)	28 (6.0)	0.36
No	318 (73.6)	114 (26.4)	432 (93.9)	
Other				
Yes	123 (73.7)	44 (26.3)	167 (36.3)	0.94
No	215 (73.4)	78 (26.6)	293 (636)	
Hb (g/dL)*	10.18±7.12	10.23±3.24	10.19±6.16	0.08
Cholesterol (mg/dL)*	170.91±67.62	175.34±66.23	172.08±62.21	0.65
TG (mg/dL)*	124.06±66.53	168.31±94.86	135.65±77.35	<0.001
HDL (mg/dL)*	97.15±80.69	63.90±54.98	88.23±75.75	<0.001
LDL (mg/dL)*	60.54±36.98	76.22±39.35	64.70±38.21	<0.001
FBS (mg/dL)*	139.24±82.61	131.13±76.34	137.40±81.18	0.31

*Mean±SD; **Frequency (%). AV=Arteriovenous; DM=Diabetes mellitus; HTN=Hypertension; GN=Glomerulonephritis; HDL=High-density lipoprotein; LDL=Low-density lipoprotein; FBS=Fasting blood sugar; SD=Standard deviation; TG=Triglyceride; Hb=Hemoglobin

a positive correlation between cTnI and triglyceride (TG) and low-density lipoprotein (LDL) cholesterol, we found a negative correlation with high-density lipoprotein (HDL) cholesterol level. Factors such as age, duration of dialysis, and tobacco consumption failed to show a correlation on the level of cTnI. In addition, ESRD etiological factors including diabetes mellitus (DM), hypertension (HTN), glomerulonephritis, urological diseases, and laboratory findings such as Hb, cholesterol, and FBS were not correlated with the cTnI level.

Table 2 lists the causes of hospitalization of patients, including cardiovascular, respiratory, gastrointestinal, and infectious causes (gastroenteritis, pneumonia, osteomyelitis, catheter infection, and sepsis). The level of cTnI has no correlation with the type of hospital admission causes such as gastrointestinal, respiratory, or infectious diseases. Cardiac cause of admission had a trend toward significance with two-sided *P* value and was significant when considered one-sided ($P = 0.045$). Furthermore, no correlation exists between the cTnI level and readmission due to cardiovascular or non-CVDs. Furthermore, early and late mortality rate was seen in people with high cTnI level.

We stratified abnormal cTnI titer in three groups, including more than one time to three times, more than three times to six times, and more than six times of upper limit of the normal range and correlated with early and late mortality. As shown in Table 3, early mortality correlated with more than one till six times, and late mortality has a significant

Table 2: Outcome of end-stage renal disease patients based on cardiac troponin I level

Outcome	cTnI normal, *n* (%)	cTnI abnormal, *n* (%)	*P*
Admission cause			
Cardiovascular			
Yes	36 (63.2)*	21 (36.8)	0.059
No	302 (74.9)	101 (25.1)	
Gastrointestinal			
Yes	30 (75)	10 (25)	0.82
No	308 (73.3)	112 (26.7)	
Respiratory			
Yes	32 (71.1)	13 (29.8)	0.70
No	306 (73.7)	109 (26.3)	
Infection			
Yes	49 (79)	13 (21)	0.28
No	289 (72.6)	109 (27.4)	
Readmission			
Cardiovascular			
Yes	33 (66)	17 (34)	0.21
No	305 (74.4)	105 (25.6)	
Noncardiovascular			
Yes	115 (70.6)	48 (29.4)	0.32
No	223 (75.1)	74 (24.9)	
Early death			
Yes	7 (38.9)	11 (61.1)	0.001
No	331 (74.9)	111 (25.1)	
Late death			
Yes	14 (46.7)	16 (53.3)	<0.001
No	324 (75.3)	106 (24.7)	

*Frequency (%). cTnI=Cardiac troponin I

Table 3: Comparison stratified cardiac troponin I level with early and late mortality

	Nl. CTnI level, *n* (%)	>1–3 times, *n* (%)	>3–6 times, *n* (%)	>6 times, *n* (%)	*P*
Early mortality	7 (1.99)	8 (13.6)	3 (15.0)	0	<0.001
Late mortality	18 (5.1)	3 (5.1)	1 (5.0)	8 (27.6)	<0.001

cTnI=Cardiac troponin I

Table 4: Factors predicting in hospital mortality in studied patients

Variables	*P*	OR
Female	0.007	1.89 (1.22–3.076)
TG	0.002	1.007 (1.003–1.011)
HDL	0.021	0.994 (0.988–0.999)
cTnI	0.005	4.81 (1.640–14.897)
Age	0.003	1.06 (1.019–1.093)
FBS	0.037	1.004 (1.000–1.008)

TG=Triglyceride; HDL=High-density lipoprotein; FBS=Fasting blood sugar; OR=Odds ratio; cTnI=Cardiac troponin I

correlation only with more than six times of upper limit of the normal range.

In logistic regression analysis, baseline cTnI level as a dependent variable was analyzed with age, gender, dialysis type, time on dialysis, vascular access type, Hb, TG, HDL cholesterol, LDL cholesterol, and FBS level, as independent an covariates. Among all values, only female gender (odds ratio [OR] =1.89, CI = 1.22–3.07, $P = 0.007$) and TG (OR = 1.007, CI = 1.003-1.01, $P = 0.002$) were positively and HDL cholesterol level (OR = 0.99, CI = 0.98–0.99, $P = 0.02$) was negatively associated with increased cTnI levels.

In addition as shown in Table 4, in the logistic regression model, early mortality as an independent variable was examined with age, cTnI, cardiac causes, HTN, FBS, TG, and albumin as covariates, but only cTnI level (OR = 4.81, CI = 1.64–14.89 $P = 0.005$), age (OR = 1.06, CI = 1.01–1.09, $P = 0.003$), and FBS (OR = 1.004, CI = 1.000–1.008 $P = 0.037$) were associated with early mortality.

AG7-Furthermore, in the logistic regression model, late mortality at the end of 6 months as a dependent variable was examined with gender, age, type and time dialysis, vascular access type, cause of renal failure, cause of admission, readmission at the end of 6 months follow-up, cTnI level, smoking, Hb level as covariates but only cardiac disease on admission (OR = 10.68, CI = 2.100–14.202, $P = 0.024$), cTnI level (OR = 4.31, CI = 1.61–10.96, $P = 0.002$), age (OR = 1.03, CI = 1.01–1.04, $P = 0.049$), Hb (OR = 0.77, CI = 0.63–0.97, $P = 0.018$), and HTN as a cause of primary kidney failure (OR = 0.34, CI = 0.120–0.87, $P = 0.023$), were associated with late mortality [Table 5].

DISCUSSION

Based on our research work in 460 ESRD patients who were admitted in hospital due to any cause except for acute MI (STEMI) or DHF, serum cTnI level was associated with female gender, dialysis modality, vascular access type, and serum TG and LDL cholesterol level but negative correlation with HDL cholesterol level. Elevated cTnI was more frequent in females than males, but patients' age was not related to cTnI level. Furthermore, positive cTnI was associated with early in-hospital and late 6-month follow-up all-cause mortality.

Patients' characteristics and cardiac troponin I level

In concordance with our results, Kalaji and Albitar[16] in 145 asymptomatic HD patients did not find the correlation with DM, smoking, and age. In contrast with our results, they found HTN and male sex were associated with troponin I level and had no association with any type of measured lipids. In our study, there was not any correlation between cTnI level and total cholesterol level. However, there was a positive correlation between the level of cTnI and level of TG and LDL, but a negative correlation between cTnI and HDL level has been established.

Table 5: Factors predicting late mortality in studied patients

Variables	*P*	OR
Cardiac disease on admission	0.024	10.68 (2.100–14.202)
cTnI	0.002	4.31 (1.614–10.964)
Age	0.049	1.03 (1.01–1.04)
HB	0.018	0.77 (0630–0.973)
HTN	0.023	0.345 (0.120–0.873)

HTN=Hypertension; HB=Hemoglobin; CTnI=Cardiac troponin I; OR=Odds ratio

In concordance with our results, Kruzan *et al*.[17] in study on 503 HD patients reported no correlation between primary cause of kidney failure and Hb level but in contrast had correlation with age and male sex.

The study by Löwbeer *et al*.[18] in their work on asymptomatic dialysis patients also found that cTnI level was only increased in 3 of 36 HD and one of 26 peritoneal dialysis (PD) patients. cTnI level was not different in patients with or without DM. It seems that in patients with ESRD, any factor that causes instability in patients' clinical condition which leads to hospitalization with different mechanisms can cause myocardial damage and rising in serum cardiac troponins.

In our study, there was a significant correlation between the types of dialysis and cTnI level; HD patients had higher cTnI level than PD. Al-Hweish *et al*.[19] in 84 dialysis patients (56 HD and 28 continuous ambulatory peritoneal dialysis) showed the same results. This low level of cTnI in PD may be due to the continuous removal of cTnI in peritoneal dialysis compared with intermittent short-time troponin removal by HD process.[19] It also appears that this difference is due to more hemodynamic fluctuations that can themselves cause more myocardial damage in short-term HD sessions compared to the persistent nature of peritoneal dialysis.

Admission/readmission causes and cardiac troponin I level

Although there was a trend toward significance for the correlation between cTnI level and cardiovascular causes of admission ($P = 0.059$), in our research, there was not any association between cTnI level and admission causes as well as readmission causes (cardiovascular and non-CVDs). It has been reported that troponins' level could be increased in diseases other than ACS including sepsis/critically ill patients, rhabdomyolysis, and acute pulmonary embolism.[20,21] In our study, we excluded patients with acute MI and DHF. Hence, increased cTnI level could be due to indirect myocyte damage in these critically ill patients that did not affect their admission/readmission frequency.

Early mortality and cardiac troponin I

Kang *et al*.[20] evaluated 305 ESRD patients (121 of them had sepsis). In agreement with our results, they found a positive association between early mortality (<90 days) and cTnI levels.

Alam *et al*. studied 133 chronic dialysis patients and followed them for two consecutive months. They showed high cTnI level was associated with an increased risk of early mortality (58% noncardiac, 39% cardiac, and 3% unknown).[22]

Fonarow *et al*. assessed the level of cTnI or cTnT in 42,636 of the 77,467 hospitalized patients due to DHF.[23] They showed a significantly increased risk of in-hospital mortality in patients with high troponin levels compared with patients without detectable troponin. Admission cardiac troponin levels are significant and independent predictors of in-hospital mortality in acutely DHF patients. Although we excluded DHF patients in our study, again we have found a positive association between elevated cTnI levels with early in-hospital mortality.

Late mortality and cardiac troponin I

Herzog *et al*. study entered dialysis patients who were hospitalized for a first MI after the initiation of renal replacement therapy and showed that 59.3%, 73%, and 89.9% of dialysis patients die within 1, 2, and 5 years of acute MI, respectively.[24] In concordance with the abovementioned study, we showed that hospitalization due to CVD was associated with 10.68 times increase in 6-month follow-up mortality risk.

Kang *et al*. showed a positive association between late mortality (more than 90 days) and elevated cTnI level in 121 patients with sepsis.[20] Although in our study, serum troponin levels were associated with a 4.31-fold increase in risk of 6-month mortality, there was no association with infectious causes. In contrast with our results, Khan *et al*. studied 126 long-term HD patients and followed them for 2 years.[25] They could not find a significant difference in late mortality rate between normal and elevated cTnI groups. They also could not find association in cardiac mortality between the two groups.

In a meta-analysis performed by Khan *et al*. found no clear association between elevated troponin I levels and mortality in asymptomatic HD patients due to lack of standardized assay.[26] In our study, however, high levels of troponin I were associated with late mortality in hospitalized patients. This discrepancy could be due to the patients' different clinical conditions.

Furthermore, Apple FS and coworkers evaluated long-term mortality in 733 asymptomatic HD patients, and after adjustment for the independent risk factors, the risk of death increased 2-fold with elevated cTnI.[13]

In our patients, after adjustment for the other independent risk factors, cTnI level remained a statistically significant risk for late mortality in 6-month follow-up.

CONCLUSION

We conducted a survey on our ESRD patients who had been hospitalized for any reason other than DHF and acute STE MI. We found that high levels of cTnI were associated with increased in hospital and in the end of 6-month follow-up mortality.

We suggest that any ESRD patient who is hospitalized for any cause with higher cTnI level may need more medical care and closer follow-up.

Ethics approval and consent to participate

There was not any intervention in the treatment protocols of individuals, and patient's information remained confidential. All the required information regarding this study explained to the participants, and then, verbal informed consent was taken. Voluntary exclusion of the study did not affect the treatment plan.

Consent for publication

Patients gave their verbal consent to publish their data anonymously.

Authors' contributions

TSh and GHG: designed the study; analyzed and interpreted the data; wrote, reviewed, and read the initial and final draft of the manuscript.

Authors' contribution

1. ST: Substantial contributions to the conception and design of the work, analysis, and interpretation of data for the work
   - Drafting the work and revising it critically for important intellectual content
   - Final approval of the version to be published
   - Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy and integrity of any part of the work are appropriately investigated and resolved.
2. GG: Substantial contributions to the conception or design of the work, the acquisition, analysis, and interpretation of data for the work
   - Drafting the work or revising it critically for important intellectual content
   - Final approval of the version to be published
   - Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.
3. AN: Substantial contributions to the conception or design of the work; and interpretation of data for the work
   - Drafting the work or revising it critically for important intellectual content
   - Final approval of the version to be published
   - Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy and integrity of any part of the work are appropriately investigated and resolved.
4. SH: Substantial contributions to the conception and design of the work, analysis, and interpretation of data for the work.
   - Drafting the work or revising it critically for important intellectual content
   - Final approval of the version to be published
   - Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy and integrity of any part of the work are appropriately investigated and resolved.

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Conflicts of interest

There are no conflicts of interest.

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