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Original Research Article

High Sensitivity Troponin T Level and Cardiovascular Performance in Patients with Liver Cirrhosis

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ABSTRACT

Introduction: Cirrhotic cardiomyopathy is a new entity reported in patients with advanced liver cirrhosis. High sensitivity cardiac troponin T (hs-cTnT) is a biological marker specifically derived from the cardiac muscle. Recent data suggest that cardiac troponins are valuable for screening asymptomatic individuals for subclinical cardiovascular disease.

Objective: To evaluate hs-cTnT level in post hepatitis C cirrhotic patients and correlating the results with severity of liver disease, carotid intima media thickness (CIMT) and cardiac performance.

Methods: The study involved 40 cirrhotic patients without established cardiovascular diseases in addition to 20 apparently healthy volunteers serving as control. Demographic details, clinical data, laboratory data, echocardiographic study of left ventricular systolic and diastolic function, abdominal ultrasonography, ECG, and B-Mode Carotid ultrasonography for evaluation of CIMT were obtained. Sera were collected for measurement of high sensitivity troponin T levels. Correlations of hs-cTnT level with echocardiographic parameters, Child-Pugh functional scoring of cirrhosis as well as with abdominal ultrasound and laboratory parameters were investigated.

Results: There was a highly statistically significant increase in hs-cTnT level in patient group compared to control group. We found a positive correlation between hs-cTnT level and portal vein diameter, serum K, Child-Pugh Score, corrected QT interval in ECG, left ventricular mass, interventricular septal diameter, peak velocity of atrial filling A and CIMT. There was a negative correlation between hs-cTnT level and liver diameter, serum sodium, serum albumin and echocardiographic E/A ratio.

Conclusion: Elevated hs-cTnT in cirrhotic patients can be one of the tools used for early diagnosis of subclinical cirrhotic cardiomyopathy.

Key words: High Sensitivity Cardiac Troponin T (hs-cTnT); Cirrhotic Cardiomyopathy; Liver Cirrhosis; Carotid Intima Media Thickness; Echocardiography.

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INTRODUCTION

Liver cirrhosis is associated with significant cardiovascular abnormalities.[1,2] It became clear that liver cirrhosis is associated with increased resting cardiac output, decreased systemic vascular resistance, reduced myocardial contractility or systolic incompetence especially under stress conditions, in addition to increased left ventricular thickness associated with diastolic dysfunction and electrocardiographic abnormalities in the form of prolonged QTc interval.[3] These abnormalities are formally described as cirrhotic cardiomyopathy (CCM), which is defined as chronic cardiac dysfunction in patients with liver cirrhosis in the absence of known cardiac disease and irrespective of the cause of cirrhosis.[4,5]

One third of the patients with cirrhosis have evidence of cardiomyopathy.[6] Furthermore, more than 80% of patients with end stage liver disease (ESLD) have parasympathetic or sympathetic dysfunction characterized by abnormal response of heart rate or blood pressure to physical activity.[7]

Most of HCV patients, who developed heart disease, have chronic inflammation of the myocardium and later, dilated cardiomyopathy attributable to necrosis and loss of myocytes. However

proliferative stimuli induced by HCV infection may promote myocyte hypertrophy and hypertrophic cardiomyopathy.[8]

Chronic hepatitis C virus (HCV) infection has been associated with atherosclerosis. HCV infection was found to be associated with a higher risk of coronary artery disease.[9] ESLD could be considered as a coronary artery risk equivalent, and it is therefore important to appropriately risk-stratify these patients even in the absence of symptoms.[10] It was found that the prevalence of coronary artery disease (CAD) in liver transplanted (LT) candidates can reach up to 26% and is associated with increased mortality and cardiovascular morbidity.[11] Carotid intima media thickness (CIMT) has been used to assess clinical and subclinical atherosclerosis.[12]

Concerning the role suggested to be played by HCV in producing carotid artery plaque and thickening of CIMT, Ishizaka et al. suggested a possible role for HCV in the pathogenesis of carotid artery remodeling.[13] Moreover, Boddi et al suggested a local pro-atherogenic action of the virus as they found HCV RNA sequences within the carotid plaque tissues taken from two HCV positive patients who underwent carotid revascularization.[14]

Troponin complex, is a complex of three regulatory proteins (troponin C, troponin I, and troponin T) that is integral to muscle contraction[15] in skeletal and cardiac muscles. Cardiac troponin is a highly specific cardiac marker which is valuable and sensitive in the diagnosis of myocardial necrosis.[16] Myocardial necrosis signified by troponin is not necessarily due to an acute coronary syndrome (ACS), other diseases, such as heart failure (HF), pulmonary embolism, renal failure and sepsis can be associated with an elevated troponin level.[17]

Pateron et al. found that there were 10 out of 32 cirrhotic patients without previous cardiac disease, have elevated troponin level.[18] Coss et al. revealed that elevated serum troponin level before liver transplantation is associated with post-transplant cardiovascular events.[19] However, there are still few data about the relationship of serum hs-cTnT level and the severity of liver cirrhosis and its correlation with abnormalities of cardiac structure and function in cirrhotic patients.

AIMS

The general objective of this study was to evaluate serum hs-cTnT levels in post hepatitis C virus liver cirrhosis. The specific objective was to correlate the results with Child-Pugh functional scoring of cirrhosis, ultrasound and laboratory parameters that define the severity of liver disease as well as with CIMT and echocardiographic parameters of cardiac performance.

PATIENTS AND METHODS

Patients: Inclusion criteria

The present study was carried out in 2015 and 2016 and included 40 patients, from Theodor Bilharz Research Institute Hospital, having post hepatitis C virus liver cirrhosis, which was diagnosed based on the results of laboratory tests (hepatitis C virus antibody, low serum concentrations of albumin, high INR and low platelet count) in addition to abdominal ultrasonographic findings (irregularity of the liver's surface). They were classified according to Child-Pugh functional scoring of cirrhosis. Twenty apparently normal healthy volunteers matched for age and sex and with normal liver ultrasound, normal liver function tests and negative hepatitis markers were also included in the study as a control group.

Exclusion criteria

- History of heart disease,
- Diabetes mellitus,
- Hypertension (blood pressure >140/90 mmHg),
- Hyperlipidemia,
- Acute or chronic kidney disease,
- Pregnancy,
- Liver masses.

Methods

All patients and normal volunteers were subjected to the following:

1) Thorough history taking and physical examination.

2) Blood sampling for serum electrolytes, HBs antigen and HCV antibody in addition to liver and renal function tests.

3) Special Investigations: hs Troponin T: Aliquots were stored at −80°C until assayed for serum hs-cTnT. Serum high sensitive cardiac troponin T was determined by the cTnT 4th-generation electrochemiluminescent immunoassay "ECLIA" on cobas e411 immunoassay auto analyzers (Roche Diagnostics GmbH, Mannheim, Germany) according to the instructions of the manufacturer. The limit of the blank was 3ng/L and the maximum of the master curve was 10000 ng/L.

4) Resting 12 leads ECG: The duration of QT interval was calculated manually. The QT intervals were corrected in accordance with heart rate using the BAZET formula (QTc = QT/√RR), QTc > 440 ms was considered prolonged.[20]

5) Abdominal ultrasound scanning using a Toshiba Memo 30 scanner equipped with a 3.5 mHz linear transducer was performed by a member of the study team who was unaware of all other clinical and laboratory data.

6) Echo-Doppler study: Echocardiographic measurements were performed according to the recommendations of the American Society of Echocardiography.[21] M-mode, two-dimensional, and Doppler echocardiography were conducted using a high resolution Toshiba Memo 30 scanner equipped with a 2.5 MHz transducer by three blinded members of the study team and measurements were averaged. Using M-mode, we measured the interventricular septum (IVSd) and left ventricle posterior wall (LVPWd) thicknesses at end diastole. Left ventriclular end-diastolic (LVIDd) and end systolic (LVIDs) diameters were determined. Left ventricular mass (LVM) was calculated according to Devereux et al. equation:

LVM (gm) = 1.04 x {(LVIDd + IVSd + LVPWd)3 – LVIDd3} × 0.8 + 0.6.[22]

The diameter of left atrium was determined from parasternal long axis view at end systole. The velocity of early mitral inflow (rapid-filling wave) (E cm/s), peak velocity of the late filling wave due to atrial contraction (A cm/s), were measured and E/A ratio was calculated. All measurements were completed by three blinded members of the study team and measurements were averaged.

7) Carotid Duplex: High resolution B mode ultrasonography of both the common

carotid arteries were performed using an ultrasound machine (Toshiba Memo 30 scanner) equipped with a 7.5 MHz high resolution transducer. The maximum CIMT was measured at the common carotid artery, 2 cm before the bifurcation, as the distance between the first and second echogenic lines of the anterior and posterior arterial walls during end diastole.[23] These measurements were completed by three blinded members of the study team and measurements were averaged.

Research Ethics: All patients were provided by informed consent, and the ethical committee of the hospital approved this study which was conducted in accordance with the Helsinki Declaration (1975).

Statistical Analysis: Statistical analysis was performed using SPSS version 17. Data were expressed as the mean ± SD for numerical variables. p≤ 0.05 was considered to be statistically significant, and p ≤0.01 was considered to be highly significant.

RESULTS

The demographic data, clinical data and electrocardiographic findings of patients and controls are shown in table (1).

Heart rate and QTc in ECG showed statistically significant increase in patient group compared to control group. While systolic and diastolic blood pressure showed statistically significant decrease in group 1 compared to group 2.

The results of routine laboratory investigations are illustrated in table (2); Serum levels of K, ALT, AST, total & direct bilirubin and INR showed statistically significant increase in patient group in comparison to control group. However, there was a statistically significant decrease in serum Na, albumin, Hb and platelets in patient group compared to control group. There was a highly statistically significant increase in hs-cTnT in patients compared to controls (figure 1).

Table 1: Demographic data, clinical data and electrocardiographic findings of patient and control groups

	Group1 (patients)		Group2 (Control)		*p* value
	Mean	SD	Mean	SD	
Age (years)	53.7	9.4	50.8	6.3	0.2184
Gender					0.4612
Male	24 (60%)		10 (50%)		
Female	16 (40%)		10 (50%)		
SBP (mmHg)	92.6	10.3	123.3	8.1	0.0001
DBP (mmHg)	59.0	8.6	78.7	4.6	0.0001
Pulse	94.8	8.7	70.2	7.3	0.0001
ECG QTc (msec)	462.43	23.7	371.24	9.3	0.0001

P < 0.05= significant, *P* < 0.01= highly significant. [SBP: Systolic blood pressure, DBP: Diastolic blood pressure, QTc: corrected QT interval in ECG.]

Table 2: Laboratory data of patient and control groups

	Group1 (patients)		Group2 (Control)		*p* value
	Mean	SD	Mean	SD	
Na (mEq/L)	130.21	4.19	141.24	1.99	0.0001
K (mEq/L)	4.63	.69	4.09	.23	0.0001
Creat (mg/dL)	1.20	.55	1.06	.15	0.2701
BUN (mg/dL)	26.51	19.70	32.59	10.29	0.2018
ALT (U/L)	31.77	29.01	14.12	2.06	0.0089
AST (U/L)	59.69	46.20	14.24	4.12	0.0001
Tbil (mg/dl)	3.09	3.56	.52	.11	0.0021
Dbil (mg/dl)	1.52	2.21	.12	.02	0.0001
Albumin (gm/dL)	2.42	.61	4.19	.10	0.0001
WBCs (10^9/L)	6.60	3.16	5.78	.31	0.2537
Hb (gm/dl)	10.38	.36	13.11	.57	0.0001
PLT count (10^9/L)	136.63	68.54	294.71	48.62	0.0001
INR %	1.59	.49	1.03	.03	0.0001
Troponin (ng/L)	22.59	25.05	4.64	1.48	0.0023

p < 0.05= significant, *p*< 0.01= highly significant. [Na: serum sodium, K: serum potassium, Creat: creatinine, BUN: blood urea nitrogen, ALT: alanine aminotransferase, AST: aspartate aminotransferase, T bil: Total bilirubin, D bil.: Direct bilirubin, WBCs: white blood corpuscles; PLT: platelets, INR: international normalized ratio.]

The results of the abdominal ultrasonography are shown in table (3). There was a statistically significant decrease in liver diameter in mid-clavicular line in group 1 compared to group 2. Also, there was a statistically significant increase in portal vein diameter and splenic span in patient group compared to control group. In patient group, ascites was mild in 8 patients (20%), moderate in 21 patients (52.5%) and severe in 9 patients (22.5%) while there was no ascites in 2 patients (5%). All subjects of control group had no ascites.

In patient group, Child Pugh Score was Class A in 9 patients (22.5%), Class B in 22 patients (55%) and Class C in 9 patients (22.5%).

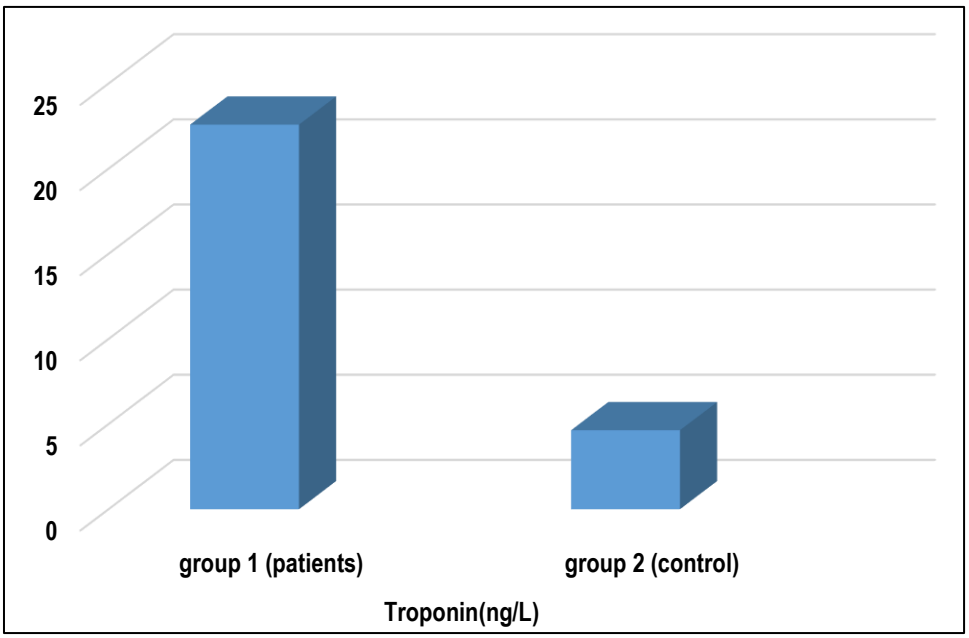

Figure 1: Troponin level of group 1 and 2

Table 3: Abdominal sonography of patient and control groups

	Group 1 (patients)		Group 2 (Control)		*p* value
	Mean	**SD**	**Mean**	**SD**	
Liver diameter (cm)	12.33	1.63	14.65	0.49	0.0001
PV diameter (mm)	14.59	1.04	6.38	0.50	0.0001
Splenic span (cm)	16.3	1.5	8.6	1.6	0.0001
Ascitis					
No	2 (5%)		20 (100%)		
Mild	8 (20%)				
Moderate	21(52.5 %)				
Severe	9 (22.5%)				
Child Pugh Score					
Class A	9 (22.5%)				
Class B	22 (55%)				
Class C	9 (22.5)				

PV: portal vein, $P < 0.05$= significant, $P < 0.01$= highly significant

Table 4: Echocardiography and CIMT of patient and control groups

	Group1 (patients)		Group2 (Control)		*p* value
	Mean	**SD**	**Mean**	**SD**	**Mean**
IVSd (cm)	1.04	0.12	0.82	0.11	0.0001
LVPWd (cm)	1.03	00.10	0.83	0.12	0.0001
LVIDd (cm)	4.97	.64	4.85	0.46	0.4585
LVM (gm)	194.57	50.11	127.66	28.23	0.0001
LA (cm)	3.93	0.57	3.45	0.34	0.0010
E velocity (m/s)	0.62	0.27	0.64	0.16	0.7616
A velocity (m/s)	0.88	0.48	0.60	0.15	0.0139
E/A ratio	0.7	0.4	1.1	0.5	0.0014
CIMT (mm)	1.01	0.12	0.44	0.14	0.0001

$p < 0.05$= significant, $p < 0.01$= highly significant. [IVSd: interventricular septum thickness (diastole); LVPWd: posterior wall thickness (diastole); LVIDd: left ventricular internal diameter (diastole); LVM: left ventricular mass; LA: left atrium diameter; E: peak velocity of early filling; A: peak velocity of atrial filling; E/A ratio; CIMT: carotid intima-media thickness.]

Regarding the echocardiographic and CIMT data, there was a statistically significant increase in LVM (figure 2), LA diameter, A velocity and CIMT (figure 3) in addition to a statistically significant decrease in E/A ratio (figure 4) in patient group compared to control group as shown in table (4). The results of correlation analysis are shown in table (5). There was positive correlation between troponin and portal vein diameter, Child-Pugh Score, corrected QT interval in ECG, left ventricular mass (figure 5), interventricular septal diameter, peak velocity of atrial filling (A) and CIMT (figure 6). While, there was a negative correlation between hs-cTnT level and liver diameter in mid-clavicular line, serum sodium, serum albumin and E/A ratio (figure 7).

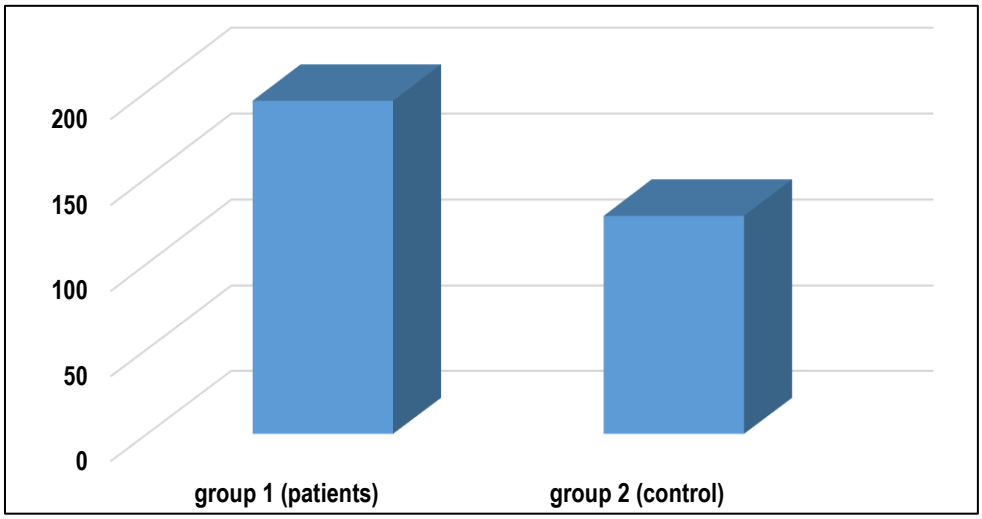

Figure 2: LVM (gm) of group 1 and 2

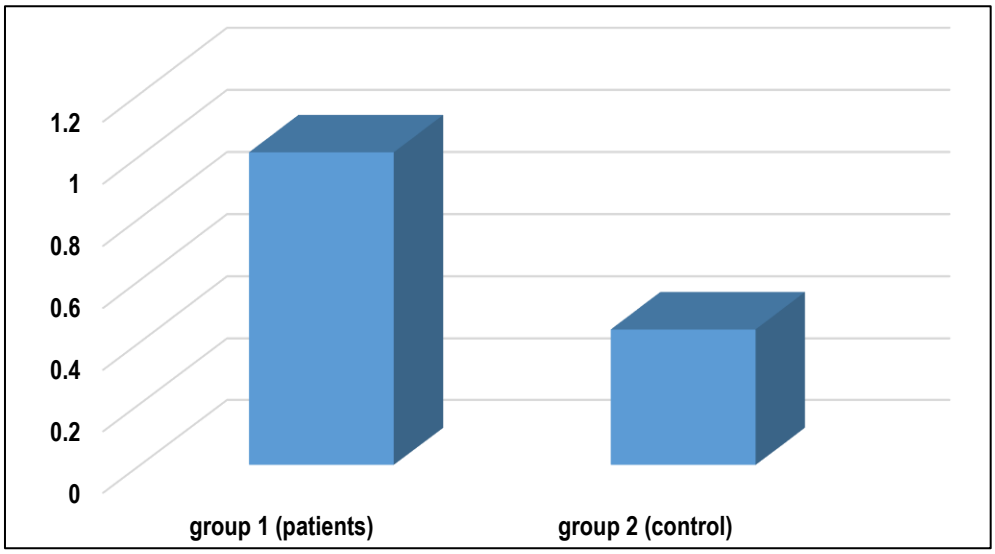

Figure 3: CIMT (mm) of group 1 and 2

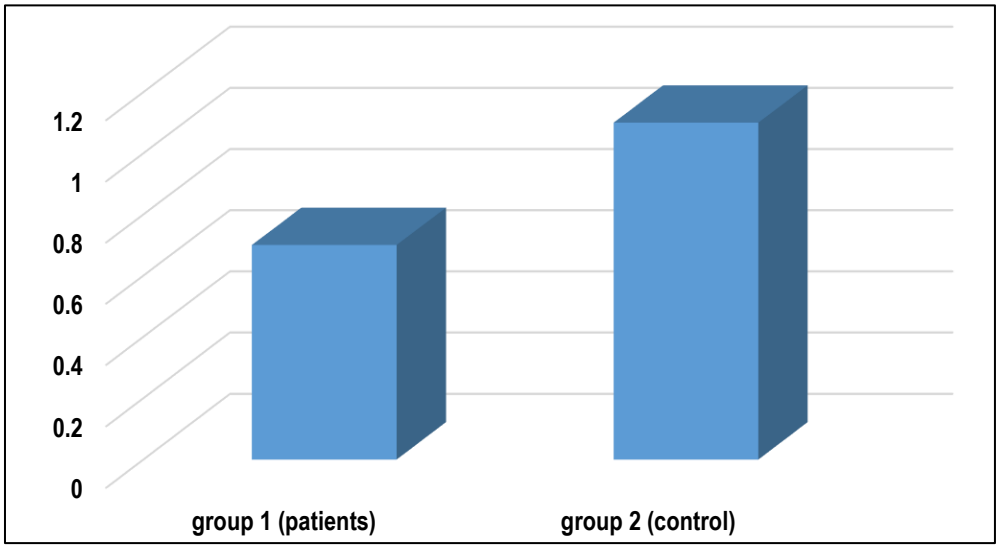

Figure 4: E/A of group 1 and 2

Table 5: Correlation between troponin and other parameters

		Liver diam.	PV diam.	Child Pugh Score	Serum K	Serum sodium	Serum albumin	QTc	IVSd	LVM	A velocity	E/A ratio	CIMT
Troponin	***r***	-.451	.436	.436	.532	-.343	-.335	.435	.289	.332	.335	-.342	.393
	p	.0001	.002	.002	.000	.008	.009	.002	.025	.010	.009	.008	.002

P < 0.05= significant, *P* < 0.01= highly significant. [Diam; diameter, PV diam: portal vein diameter, QTc: corrected QT interval in ECG, LVM: left ventricular mass, IVSd: interventricular septal diameter thickness (diastole), A velocity: peak velocity of atrial filling, CIMT: carotid intima-media thickness.]

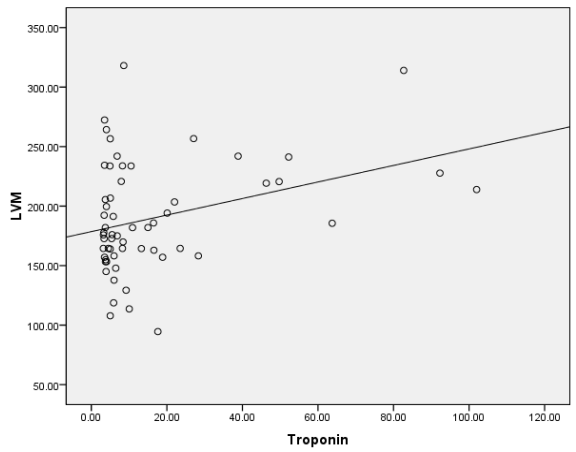

Figure 5: Positive correlation between Troponin and LVM

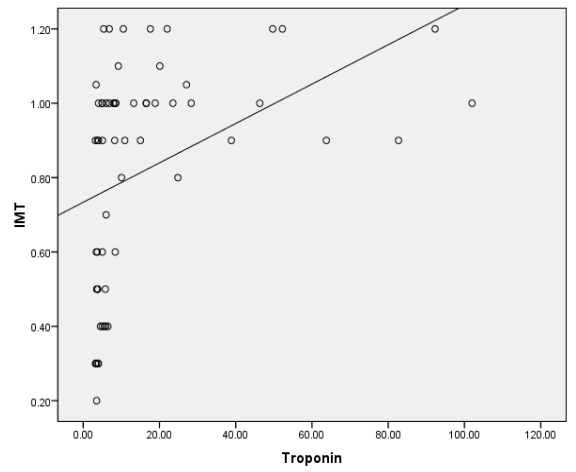

Figure 6: Positive correlation between Troponin and carotid IMT

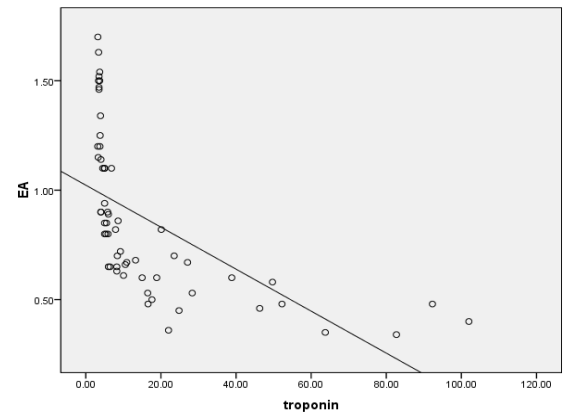

Figure 7: Negative correlation between troponin and E/A wave

DISCUSSION

Detection of circulating cardiac troponins is associated with the presence of ongoing myocardial necrosis.[24] Recent reports have associated detectable cardiac troponin levels in stable non-cardiac subjects with heightened risk of developing future cardiovascular events.[25] At present, it is unclear whether microvascular ischemic insults, or various oxidative and inflammatory mediators, contribute to myocyte injury, apoptosis and progressive myocyte loss, leading to release of troponin fragments into the circulation.[26]

Our results showed that hs-cTnT is increased in patients with post hepatitis C liver cirrhosis and its level is correlated positively with portal vein diameter, serum K and Child-Pugh Score and negatively with liver diameter in mid-clavicular line, serum sodium and serum albumin. This means that hs-cTnT level increases with more advanced stages of liver cirrhosis, and cirrhotic cardiomyopathy is more common in end stage liver diseases and so, we hypothesize that decompensated cirrhotic patients may have some degree of myocardial injury. Our results are in accord with that of Tangaroonsanti et al., who found elevated troponin I levels in cirrhotic patients who had no history of cardiac disease that increased with more advanced stages of liver disease and significantly correlated with serum potassium.[27] Also Wiese et al. observed increased circulating levels of hs-cTnT in cirrhosis, with the highest levels in patients with decompensated cirrhosis.[28]

In the present study, there was a positive correlation between prolonged QTc intervals and hs-cTnT levels which could suggest that the prolongation of the QTc interval is not only associated with myocardial ischemia, but with myocardial injury. In agreement with our findings, Rukshin et al. observed prolonged QTc interval in acute coronary syndrome that was more marked in non-Q-wave acute myocardial infarction patients than in those with unstable angina who had no evidence of myocardial injury.[29] To our knowledge, no studies have been done to correlate hs-cTnT with QTc interval in cirrhotic patients.

In our study, we also found a positive correlation between LVM & IVSd and hs-cTnT levels. Our study is going with that of Pateron et al. who found elevated troponin levels in cirrhotic patients who had no history of cardiac disease that was related to left ventricular mass.[30] Previous studies in patients with hypertrophic cardiomyopathy have demonstrated that LVM index together with maximal LV wall thickness had the strongest positive correlation with troponin level.[31-33] Microvascular dysfunction, due to increased pressure load and decreased capillary density, has been reported to cause ischemia in both secondary hypertrophy and hypertrophic cardiomyopathy.[34,35]

Also, we found a positive correlation between hs-cTnT and A wave velocity of mitral flow and a negative correlation with E/A ratio. This mean that elevated troponin level was correlated with

severity of diastolic left ventricular dysfunction in patients with liver cirrhosis. This is in accordance with previous studies that have been done on patients having severe sepsis and septic shock[36], chronic kidney disease[37], coronary heart disease[38] and left ventricular hypertrophy.[39] But, no studies have been done on patients with liver cirrhosis.

The present study showed that elevated hs-cTnT level in cirrhotic patients was in parallel with increased CIMT. This is in consistent with the study done by Caliskan et al[40] on peritoneal dialysis patients and they concluded that hs-cTnT is a useful biomarker for evaluating noninvasive predictors of atherosclerosis in chronic peritoneal dialysis patients.

CONCLUSION

hs-cTnT level increases in liver cirrhosis and is well correlated to Child-Pugh scoring, ultrasonographic findings, laboratory parameters that define severity of liver disease, as well as with CIMT, corrected QT interval in ECG, LVM and diastolic function.

Elevated hs-cTnT level in cirrhotic patients can be considered one of the tools used to early diagnose subclinical myocardial necrosis and cirrhotic cardiomyopathy that can prevent adaptation to acute hemodynamic disturbance such as during liver transplantation or intrahepatic portosystemic shunt.

Further researches are needed to adopt more aggressive preventive strategies based firstly on identifying risky patients with subclinical myocardial necrosis aiming at improving prognosis of these patients.

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