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Assessment of troponin I level in patients with acute myocardial infarction and its impact on clinical outcome: An observational study

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Abstract--Introduction: Coronary Artery disease is one of the major causes of Mortality in the world that included Myocardial ischemia and infarction. Cardiac troponins are (troponin- I and Troponin -T) the markers of myocardial ischemia and they are the sign of Myocardial damage. They can provide important diagnostic and prognostic information. Aim : In the present study the correlation of clinical presentation, complication and outcome with reference of different level (mild, moderate and severe) of elevated troponin enzyme level was studied. Material and method: 100 patients presented with acute myocardial infarction were studied for clinical presentation (hemodynamics, heart failure, mechanical complication, angina, shock) and ICU stay and total hospital stay, recurrent angina, heart failure, re-infarct, morbidity and mortality was recorded. Their cardiac troponin- I level was measured and correlated accordingly in group 1 (mild elevation: cTnI level baseline (0.004) to ten times), group 2 (moderate elevation: cTnI level ten times to hundred times) and group 3 (severe elevation: cTnI level more than hundred times). RESULTS: Significantly greater number of patients with recurrent angina were having severely elevated Troponin-I level. Relocation of patient in ICU was more with patient with severely elevated level. Severity of cardiac failure and Troponin-I levels were found to be significantly associated with each other. Overall mortality rate was highest in patients with severely elevated Troponin-I level. Conclusion: There is a significant association between the cTnI levels and mortality and heart failure in the present study.

Keywords--cardiac troponin- I level, heart failure, cardiac mortality, acute myocardial infarction, coronary artery disease.

Introduction

Coronary artery disease (CAD) is the pre-eminent cause of mortality and loss of Disability Adjusted Life Years (DALYs) around the globe^[1]. Past few decades have seen alarming rise in the CAD and cardiovascular mortality in South Asian countries especially India. India is facing epidemiologic transition in the incidence of CAD^[2]. The conventional risk factors are incapable of explaining this increased risk^[3]. Epidemiologic studies from different parts of the country indicate a prevalence of CHD to be 7% to 13% in urban and 2% to 7% in rural populations. Its prevalence is 3 times higher in the migrant Asian Indian as compared to native Indian. The prevalence is also high (nearly 21%) amongst diabetic population^[3].

There are two facets of CAD: stable CAD referring to ischemia occurring due to a reversible supply/demand mismatch^[4] and unstable CAD which includes patients with acute coronary syndrome (unstable angina, non-ST elevation myocardial infarction, ST elevation myocardial infarction). The treatment of stable CAD (stable angina) includes anti-anginal medication, medication to modify atherosclerosis and aggressive treatment of causative risk factors. Patients with stable CAD refractory to medical treatment usually require coronary angiography to be followed by either percutaneous or surgical revascularization^[5]. Acute

coronary syndromes (ACS) are the result of plaque rupture with resultant thrombus and present as unstable angina, non-ST elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). It usually manifests as chest pain, although chest pain can be a sign of a fatal heart attack, it can also stem from many other benign conditions^[5]. The survivors of Myocardial Infarction (MI) have high risk of recurrent infarction and the annual mortality rate amongst them is at least five to sixfold higher as compared to individuals who do not have CAD^[1].

The progression of CAD is highly unpredictable. Atherosclerosis of coronary artery progresses at variable rates varying from gradual increase in luminal narrowing to an abrupt progression to total luminal occlusion^[6]. The acute coronary syndrome is diagnosed based on the presence of typical patient symptoms, EKG changes and alterations in the cardio-specific markers of myocardial damage. Cardiac troponins are known as specific markers of myocardial damage. Three types of cardiac troponins found in striated muscle cells of heart are Troponin C (cTnC), Troponin I (cTnI) and Troponin T (cTnT). Nearly 92–94% cTnT, 96–97% TnI are bound to troponin-tropomyosin protein complex whereas 6–8% and 2.8–4.1% TnI and cTnT are found in loose state in cytosol. Cytosolic fraction is the first to be released into the circulation from injured cardiomyocyte sarcolemma. This fraction is detected in blood during the early stages of Ischaemia. With the persistence of ischaemia, necrosis of cardiomyocyte occurs over several days, which releases bound troponin. Cardiac troponin levels are detectable in the serum throughout this time period (5-10 days) or may be up to 15 days in extreme cases^[7]. cTnI and TnT provide important diagnostic and prognostic information^[8]. Although it has been found that troponins can be elevated and reflect worse prognoses in many situations where ACS is excluded, such data can affect the validity of cardiac Troponins (cTn) as valuable markers for ACS without classic symptoms. This may call for a revision of the troponin cut-off values to make a diagnosis of ACS. Furthermore, it opens a new field of study to determine appropriate management of patients with elevated cTn levels in whom ACS has been excluded. Thus, this study was undertaken to study association of Troponin I level, in patients with acute MI and clinical outcome so to assess prognosis, anticipate complications and stay in intensive care.

Aim

To study the association between Troponin I level and time lapse since initiation of chest pain, severity of heart failure, duration of hospital stay, and mortality in patients with Myocardial infarction in patients with Acute Myocardial Infarction

Materials and Method

This observational study was conducted amongst the adult patients (18 years or above) presenting with acute MI at the Department of Medicine, MY hospital, Indore (M.P.) after obtaining approval from the Institutional Ethical Committee (ECR/397/Inst/MP/2013/RR-20). Pregnant females and patients with conditions other than Acute MI, which may cause elevation of serum cTnI were excluded from the study. Such conditions included demand ischemia (in the absence of ACS), supra ventricular tachycardia/atrial fibrillation, left ventricular

hypertrophy, direct myocardial damage, cardiac contusion, direct current cardioversion, cardiac infiltrative disorders, chemotherapy, pericarditis/myocarditis, cardiac transplantation (immune-mediated reactions), myocardial strain and non-cardiac causes, that ultimately leads to heart involvement like; pulmonary embolism, pulmonary hypertension or COPD, chronic renal insufficiency, end stage renal disease, sepsis/systemic inflammatory processes, intracranial pathology like stroke, sepsis/septic shock, strenuous exercise, patient presented with psychiatric illness. Informed written consent was obtained from all the participants and confidentiality of the information obtained from them was maintained. The study included a sample of 100 patients. The patients were enrolled using convenience sampling technique. The assessment of clinical features at the time of admission in terms of hemodynamic, heart failure, mechanical complication, ICCU and hospital stay, relocation of patient in ICCU due to recurrent angina, heart failure, re-infarct, morbidity and mortality was done. The statistical analysis was done using SPSS (Statistical Package for Social Sciences) 20.0 version, IBM, Chicago.

Results

The study included 100 patients with the mean age of 53.16 ± 13.41 years, attending the hospital with acute MI. The male:female ratio was 1.6:1. The patients were classified into 3 categories/groups viz. group 1 (mild elevation: cTnI level baseline (0.004) to ten times), group 2 (moderate elevation: cTnI level ten times to hundred times) and group 3 (severe elevation: cTnI level more than hundred times), based on their cTnI levels. The age-wise and gender-wise distribution of patients belonging to three groups has been presented in table 1. History of previous hypertension (56%), diabetes (37%), previous history of CAD MI (14%), and smoking (49%; none of the females had habit of smoking) were found to have no significant association with the cTnI levels (p value $> .05$).

Coagulopathy (16%) was found to have significant association with the cTnI levels (Chi-square value- 10.922, df- 2, p value $< .05$). Based on cTnI value 89 patients were diagnosed to have STEMI and 11 were found to have NSTEMI. Amongst these patients with MI, 10% percent patients belonged to group 1, 46% belonged to group 2 and 44% patients belonged to group 3. Overall, most of the patients (45%) stayed for 2 days in ICCU. The duration of stay in ICCU was found to have no significant association with levels of cTnI (p value $< .05$). [Figure 1] Similarly, total duration of hospital stay for 5 days or more was more for group 2 and 3 patients as compared to that for group 1 patients. However, this difference was statistically non-significant (p value $> .05$). [Figure 2] No significant association was seen between the duration of chest pain reported by the patients and cTnI levels (p value $> .05$). [Table 2]

A significantly greater proportion of patients with recurrent angina (63.6%) were having severely elevated cTnI levels (Chi-square value- 7.719, df-2, p value $< .05$). The relocation of patients in ICCU following recurrent angina was commonest amongst patients belonging to group 3 (47.7%) followed by group 2 (21.7%) and was least common in group 1 patients (20.0%). However, this difference was statistically non-significant (p value $< .05$). The severity of heart failure and cTnI levels were found to be significantly associated with each other (p value $< .05$).

[Table 3]. Overall, the mortality rate was 11% and it was highest amongst group 3 (20.5%) followed by group 2 (4.4%), whereas no mortality was recorded in group 1. The association between cTnI level and mortality was statistically significant (p value <.05). [Table 3]

Discussion

The present study was undertaken to find the association between cTnI levels and outcome in patients with Acute MI. Majority (33.0%) of the patients presenting with MI belonged to the age group of 41-50 years. Only 15% patients were belonging to age group of less than 40 years, amongst them 78.5% were males and 21.4% were females [Table 1]. Although MI is less prevalent before 35 years, now a days there is rising trend of MI amongst population owing to change in lifestyle factors^[9]. *Bhardawaj R et al.* (2014) also reported that MI in less than 40 years of age is almost exclusively seen in male^[10]. In the present study, the number of males (62.0%) experiencing MI was greater than the number of females (38.0%). *Srivastava RK et al.* (2014) also reported lesser number of females as compared to males, amongst patients with MI^[11]. The difference in the prevalence of MI amongst males and females can be attributed to the difference in prevalence of certain important risk factors such as smoking amongst them. In the present study none of the females had habit of smoking. A vast amount of literature reported smoking to be common amongst males as compared to females^[12]. Smoking has been identified as a major modifiable risk factor for MI^[11].

The levels of troponin I level begins to start within 2-3 hours after the onset of chest pain and continue to rise till they reach their peak usually after 12-48 hours^[13]. In the present also, cTnI levels begin to rise in 6% patients within 2 hours. Within 6 hours after the onset of chest pain, all the patients were having elevated levels of cTnI. However, the duration of chest pain was not significantly associated with the amount of cTnI levels. [Table 2]. In this study, the Killip class IV was found to have significant association with severely elevated levels of cTnI. [Table 3] This can be justified by the fact that patients with higher Killip class had worse clinical profile representing the existing damage to the heart^[14] and cTnI levels are also indicative of damage to heart muscles^[7].

In the present study, the mortality rate of MI patients was found to be significantly associated with elevated levels of cTnI. It has been reported that in the patients with ACS, the Troponin levels correlate with the risk of death and also patients with normal EKG findings and CK-MB levels but with elevated troponin levels are at higher risk of death than do patients with normal troponin levels^[15]. The duration of stay at hospital and specifically in ICCU were found to have no significant association with the elevated levels of cTnI. [Figure 1 & 2] This can be described with the fact, the greater the level of cTnI, the greater is the existing damage to heart which increases the mortality rate. The mortality amongst patients with severely elevated levels if cTnI was significantly high and thus it led to no longer duration of hospital stay.

Conclusion

Based on the results of the study, it can be concluded that there is a significant association between the cTnI levels and mortality, Killip's class of heart failure. However, duration of chest pain, duration of hospital stay, duration of stay in ICCU, relocation to ICCU following recurrent angina have no significant association with cTnI levels.

Tables

Table 1
Age-wise and gender-wise distribution of study participants belonging to three groups

Age in years	Number of patients						Total
	Group 1 (n=10)		Group 2 (n=46)		Group 3 (n=44)		
	Male	Female	Male	Female	Male	Female	
19-30	1	0	2	3	3	0	9
31-40	1	0	4	0	0	0	5
41-50	5	0	5	5	10	8	33
51-60	1	0	8	3	5	5	22
61-70	1	1	9	2	6	6	25
>71	0	0	1	4	1	0	6
Total	9	1	29	17	24	20	100

Table 2
Comparison of cTnI level and duration of acute chest pain

Duration of chest pain (in hours)	Number of patients				P value ^a
	Groups based on level of cTn I			Total	
	Group 1	Group 2	Group 3		
Two	0	2	4	6	>.05
Three	1	4	15	20	
Four	3	13	8	24	
Five	3	12	6	21	
Six	3	15	11	29	
Total	10	46	44	100	

^aChi-square test. Chi-square value- 12.977. df-8.

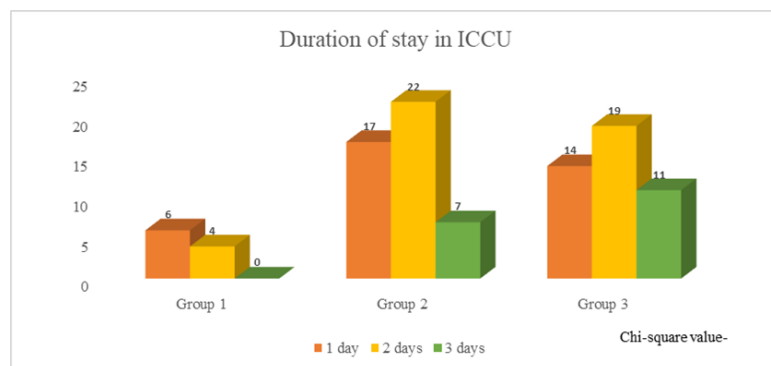
Table 3
Comparison of severity of heart failure (Killip's classification) amongst patients belonging to different groups

Heart failure (Killip's class)	Number of patients				P value ^a
	Groups based on level of cTn I			Total	
	Group 1	Group 2	Group 3		
Class I	9	26	8	43	<.05*
Class II	1	7	4	12	
Class III	0	9	15	24	

Class IV	0	4	17	21	
Total	10	46	44	100	

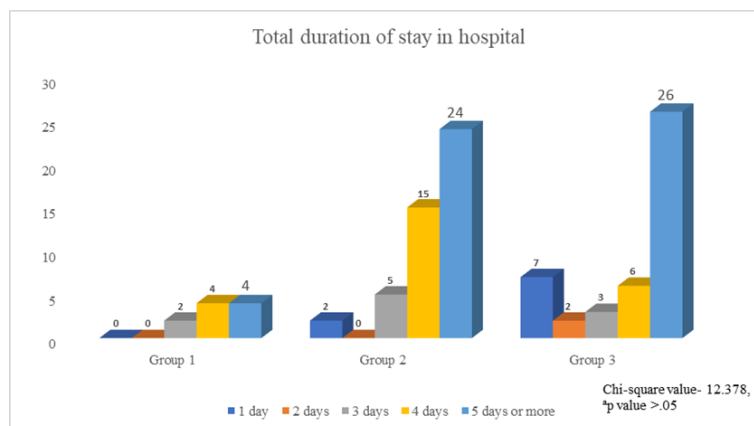
^aChi-square test. Chi-square value- 30.713, df- 6, *p value<.05 was considered statistically significant.

Figures



^aChi-square test. *p value >.05 was considered statistically significant.

Figure 1. Comparison of duration of stay in ICU of patients belonging to different groups.



^aChi-square test. *p value <.05 was considered statistically significant.

Figure 2. Comparison of total duration of stay in hospital of patients belonging to different groups

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