Original Article

Prognostic Role of Cardiac Troponin I after Percutaneous Coronary Intervention in Stable Coronary Disease

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Abstract

Background:
Cardiac troponin I is a highly sensitive and specific marker of myocardial necrosis. In addition to the strong diagnostic role of cardiac troponin I its prognostic value has become increasingly well established for patients presenting with acute coronary syndrome. However, there have been conflicting reports on the value of troponin in the setting of PCI is stable and unstable coronary disease. ***Objective:*** *To assess the role of cardiac troponin I in predicting outcome after PCI.* ***Methods and results:*** *CTnl was measured immediately before and at 8 hrs and at chest pain after PCI in 80 consecutive patients with stable coronary artery disease. Twenty of them with post procedural CTnl level 50.4 ng/ml were excluded because of the inability to do repeat estimation of CTnl at chest pain. Among the rest sixty patients, thirty had post procedural troponin <0.4 ng/ml were considered as group I and thirty had post procedural rise of CTnI >0.4 ng/ml were considered as group II. CTnl level, 0.4 ng/ml was consider as cut off value for grouping patients was based on ACC/ AHA/ ISCAI 2005 guide line definition of peri procedural myocardial infarction. The study end point was the following adverse cardiac events-recurrent angina, cardiogenic shock, significant arrythmias, congestive heart failure, Q wave MI, repeat PCI/ CABG, death during hospital stay and at 30 days follow up. In this study base line parameters like age, sex, BMI, risk factors, anginal class, base line ECG and LVEF showed no statistically significant difference between the two groups. Angiographic parameters such as types of lesion and procedural complications shows statistically significant difference between two groups. In-hospital adverse cardiac events after the procedure was significantly higher in group II than group 1 (P<0.01). The mean duration of post procedural hospital stay was also significantly higher in group II than group 1 (P<0.01). At 30 days follow up there has no incremental risk of adverse cardiac events.* ***Conclusion:*** *CTnl rise at peri procedural myocardial infarction level was observed in 37.5% of this study patient. This level of CTnl was significantly predictive of an increased risk of adverse cardiac events at hospital follow up.*

Key Words: *stable coronary disease; PCI; Cardiac troponin I.*

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Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

Introduction: In spite of relentless effort and research coronary artery disease remains the leading cause of death worldwide[1].

National data on incidence and mortality of coronary heart disease are few in Bangladesh. The prevalence of coronary heart disease was estimated as 3.3 per thousand in 1976 and 17.2 per thousand in 1986 indicating five folds increase of the disease by 10 years[2].

Treatment options for coronary artery disease include-medical therapy, percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG).

Percutaneous coronary intervention (PCI) is a technique that has been continually refined and modified. Percutaneous angioplasty for angina was conceived and developed by Andreas Gruntzig in 1977, followed by the first stenting of coronary arteries by Sigwart and Puel in 1986, primary angioplasty for acute myocardial infarction in 1988, brachytherapy in 2000 and drug-eluting stent in 2001. Over the years, technology advances in equipment and devices have improved safety as well as short and long term outcome. PCI now includes intracoronary balloon angioplasty, implantation of intracoronary stents, brachytheraphy, rotational atherectomy, directional atherectomy, extraction atherectomy, laser angioplasty and other catheter devices of treating coronary atherosclerosis. In this study, intracoronary stent implantation with or without balloon angioplasty were included as PCI.

Though much progress has been made with PCI since its first inauguration, its glory in many cases is thwarted by some adverse situations. To accurately stratify patients according to their risk of future adverse events, a quest for risk predictors is ongoing worldwide, but only a few powerful and independent predictors of early and late major adverse cardiovascular events have been found. These include traditional risk factors, such as acute coronary instability, diabetes mellitus, reference vessel diameter, and lesion and/or stent length. Nonetheless, risk assessment is still incomplete if based only on such "traditional" prognostic variables[3]

For this reason, since the last decade, biochemical markers have been carefully investigated; among these, several have been related to the risk of death, myocardial infarction and repeat revascularization after PCI.

Cardiac troponin I (cTn I) is a highly sensitive and specific marker of myocardial injury and necrosis[4]. Previous studies have found CTnI elevation to predict short and long term adverse clinical outcomes in acute coronary syndromes, including unstable angina and acute myocardial infarction[5]. Elevation of cardiac troponins was detected in 30% to 44% of patients undergoing catheter-based coronary interventions[6]. This rate may be higher than creatine kinase (CK) and creatine kinase- MB (CK-MB) elevation, which has been reported in 5% to 26% of patients undergoing percutaneous transluminal coronary angioplasty[6].

The definition of myocardial necrosis following coronary intervention has been traditionally based on CK and /or CK-MB levels[7]. The actual cut off level that may carry prognostic value is a matter of controversy, yet several recent investigators have recommended a value CK-MB > 3 times the upper normal range[7] as a cut off associated with incremental adverse prognostic risk. However other studies have found no adverse long term clinical out comes in patients with CK or CK-MB elevation[8]. More over conflicting results concerning the prognostic value of cardiac enzyme elevation were reported in studies comparing directional coronary atherectomy with balloon angioplasty[9]. In those studies the significance of myocardial damage was not assessed by cTn I, which is probably a more sensitive and specific marker of myocardial damage[10]. Fuchs et al (2000) showed that 19% of the patients with CTnI level> 3 times the upper normal range had CK-MB<3 times the upper normal range[11]. Such discrepancy points to the potential advantage of CTnI as a more sensitive predictor for adverse clinical events.

The patient with post procedural rise of cardiae Troponin I (cTn I) are more likely due to lesion complexity, increased thrombus load and post procedural complications such as side branch occlusion, multistent use, sapheneous vein graft intervention and glycoprotein IIb/IIIa use[12]. The timing of the peak elevation after PCI is unclear[13]. Minor elevations do not appear to have prognostic value, where as marked (greater than five time) elevation are associated with worse 1 year out come[14].

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

In patients with stable CAD without acute myocardial infraction troponin-I elevation after PCI did not predict mortality[15] and a post-PCI elevation of more than three times the normal limit had no incremental risk of adverse 8 months clinical out comes[11]. A Meta analysis of 2605 patients suggested that use of low cut off concentrations after PCI does not correlate with an increased incidence of composite adverse events (cardiac death, myocardial infarction, by pass surgery or repeat PCI of the target vessel) and some multiple of the cut off may be more appropriate for the prediction of adverse events[16]. On this background of controversial reports of CTnI, it was tried to evaluate the prognostic role of CTnI following PCI in patient with stable coronary disease in this study.

METHODS

Study population: This prospective non randomized clinical study comprised of 60 subjects who were admitted for PCI in NICVD, Dhaka Bangladesh from January 2006 to December 2007. Patients with symptoms of stable angina or functional evidence of silent ischemia undergoing PCI to native coronary arteries were divided into groups. One group with 30 consecutive patients with troponin I < 0.4 ng/ ml at 8hrs & at chest pain after PCI. Another group with same number of patient with troponin I >0.4 ng/ ml at 8hrs.

The exclusion criteria were patients with acute coronary syndrome, Previous PTCA or bypass surgery, Left ventricular EF <30%, Serum creatinine >2.5mg%, Conditions known to be associated with raised troponin I level.

Study protocol: Initial evaluation of patients by history & clinical examination were performed & recorded is patient's data collection form. Demographic profile including age, sex, BMI, height & weight were recorded. Risk factors of IHD, base line laboratory investigations, 12 lead ECG, echocardiography were noted for each patient.

All patient were under gone pre-procedural & post procedural estimation of serum cardiac troponin I at 8hrs & at chest pain.

Observation during the procedure: Per-procedural data includes number of vessels involved, site of lesion, type of lesion, length of lesion, device used and anticoagulant used, occlusive dissection, non-occlusive dissection, abrupt vessel closure, side branch occlusion, thrombus, hypotension, arrhythmias were recorded.

Post procedural follow up began immediately after completion of the intervention itself and was done hourly for the first 6 hours and then every morning and evening or even more frequently if indicated. Twelve- leads ECG were done: Immediately and twenty four hours after the procedure, before discharge and as necessary. Cardiac Troponin I level was measured in every patient at eight hours following the procedure. A second sample was taken thereafter on the basis of chest pain if the initial CTnI value was ≤0.4 ng/ml. Duration of follow up after intervention: All patients were followed throughout the period of index hospitalization till discharge or death. Patients who survived were also followed at day 30 over telephone.

Post Procedural evaluation

The following events & parameters in hospital & at 30 days were considered for assessment of adverse out come-

1. Recurrent anginal pain
2. Significant arrythmias.
3. Cardiogenic shock.
4. Congestive cardiac failure.
5. Q wave MI.
6. Need for Repeat PCI or CABG
7. Death

Estimation of serum cardiac troponin I: (Jaffe, Babuin & Apple 2006)

Serum cardiac troponin I was analyzed quantitatively with Abbott i STAT Assay system. The assay had a detection of lower limit 0.02 ng/ml with 99th percentile level of 0.08 ng/ml.

As according to ACC/ AHA 2005 guide lines for percutaneous coronary Intervention marked elevations of post procedural troponin I (greater than 5 times the 99th percentile of upper reference limit) appear to have prognostic value, CTnIlevel 0.4 ng/ml was considered as cut off value for grouping patients in this study.

Data collection and analysis:

Data were collected in a pre-designed form. The data obtained, were expressed in frequency, percentage, mean ± standard deviation, as applicable.

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

Comparison between groups was done by chi-square test, Student's 't' test, Fisher's exact test or others, as applicable. Computer-based SPSS (Statistical Package for Social Science) programme was used for data analysis. All p values <0.05 were accepted as significant.

RESULTS

This prospective study was conducted in National Institute of Cardiovascular Diseases (NICVD), Dhaka during the period of January 2006 to December 2007. The objective of the study was to assess the role of cardiac troponin I in predicting in-hospital and short term (30 days) outcome after PCI in stable coronary disease. A total of 60 patients were studied, among them 30 patients had post procedural CTnI ≤ 0.4 ng/ml and were considered as group I and rest 30 patient had post procedural CTnI > 0.4 ng/ml and were considered as group II.

Table I: Age and sex distribution of study patients (N=60).

Age in years	Group I (n=30)		Group II (n=30)		t value/ chi value	p value
	n	%	n	%		
29-39	5	16.7	5	16.7		
40-49	11	36.7	10	33.3		
50-59	13	43.3	10	33.3		
≥60	1	3.3	5	16.7		
Mean ±SD	48.4±9.1		49.3±9.7		0.370	[b] 0.692 NS
Sex						
Male	28	93.3	27	90.0	0.22	[a] 0.500 NS
Female	2	6.7	3	10.0		

NS= Not significant
Group I= Patients with CTnI≤ 0.4 ng/ml
Group II= Patients with CTnI>0.4 ng/ml
a= p value reached from chi square test
b= p value reached from unpaired t test

Table I shows the age and sex distribution of the study subjects. The mean age of group I was 48.4±9.1 years and for group II 49.3±9.7 years. Among group I patients 28(93.3%) were male and 2(6.7%) were female. Whereas among group II patients, 27(90%) were male and 3(10%) were female. No statistically significant age and sex difference (p>0.05) was found between two groups of patient.

Table II: Distribution of patients by pattern of risk factors (N=60).

Risk factor	Group I (n=30)		Group II (n=30)		chi value	p value
	n	%	n	%		
Smoking	18	60.0	20	66.7	0.29	0.592 NS
Hypertension	12	40.0	13	43.3	0.07	0.793 NS
Diabetes mellitus	7	23.3	6	20.0	0.10	0.754 NS
Dyslipidaemia	5	16.7	4	13.3	0.133	0.500 NS
F/O IHD	5	16.7	5	16.7	-	-

F/o IHD = Family history of ischaemic heart disease
NS= Not significant

Group I= Patients with CTnI≤ 0.4 ng/ml
Group II= Patients with CTnI>0.4 ng/ml
p value reached from chi square test

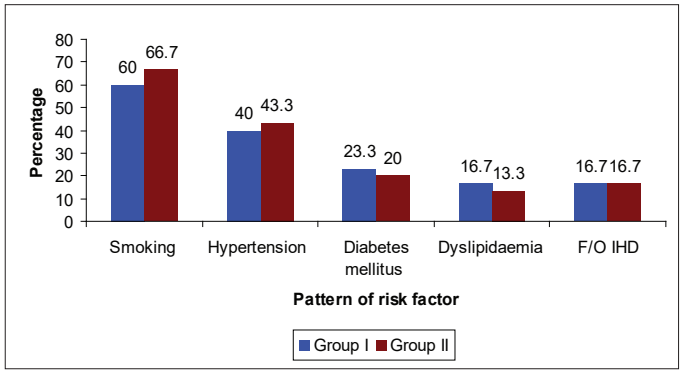

Fig: Bar diagram showing the distribution of patients by pattern of risk factors (N=60).

Table II shows the percentage distribution of risk factors among the study subjects. In group I highest percentage of risk factor was smoking (60%) followed by hypertension (40%), diabetes mellitus (23.3%), dyslipidaemia (16.7%), F/O IHD (16.7%). whereas in group II, highest percentage of risk factor was smoking (66.7%) followed by hypertension (43.3%), diabetes mellitus (20%), F/O IHD (16.7%), dyslipidaemia (13.3%). No statistically significant difference (p>0.05) was found in terms of risk factors between the two groups of patient.

In this study the mean BMI was 24.8 ± 4.1 in group I patients and 25.3 ± 4.4 in group II patients. No statistically significant mean difference (p>0.05) was found between two groups of patient. It was found that among group I patients, 21 (70%) had normal body mass index, 6(20%) were over weighted and 3(10%) were obese, whereas among group II patients, 20 (66.7%) had normal body mass index, 6(20.0%) were over weighted, 4(13.3%) were obese.

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

This study shows that among group I patients, majority had class II stable angina (40%), followed by class III (33.33%) and class I (26.7%) angina. But among group II patients majority had class II stable angina (46.7%), followed by class III (30%) and class I (23.3%) angina. No group had patient with class IV stable angina. No statistically significant difference (p>0.05) was found between two groups of patient.

The mean percentage of base line ejection fraction of group I patients was 50.5 ±6.9% and that of group II patients was 49.9±6.1%. The mean difference of ejection fraction between the two groups was not statistically significant (p>0.05).

Here among the patients of group I, 13 (43.3%) had type A lesion, 12(40%) had type B lesion, 5(16.7%) had type C lesion. Where as among group II patients majority of them had type B (43.3%) lesion, followed by type C (40%), type A (13.3%) and mixed type (3.3%) lesion. The difference was statistically significant between two groups for type C (p<0.05) and type A (p<0.01) lesion.
In this study in group I highest percentage of patients had single vessel PCI (73.3%), followed by double-vessel PCI (26.7%). But in group II highest percentage of patients had double vessels PCI (53.3%) followed by single vessel PCI (46.7%). No patient had triple vessel PCI. Analysis found no statistically significant difference (p>0.05) between the two groups of patient.

Table III : Distribution of study subjects by different types of intervention (N=60).

Types of intervention	Group I (n=30)		Group II (n=30)		Chi value	p value
	n	%	n	%		
PTCA only	0	0.0	0	0.0		
PTCA with stent	23	76.7	25	83.3	0.42	0.518 NS
Stent only	7	23.3	5	16.7		
Total	30	100.0	30	100.0		

NS= Not significant
Group I= Patients with CTnI≤ 0.4 ng/ml
Group II= Patients with CTnI>0.4 ng/ml
p value reached from chi square test

Table III shows the pattern of cardiac interventions done among the study subjects. It was found that in group I patients PTCA with stent was the highest approach (76.7%) followed by stent only (23.3%). Similar pattern of interventions were done among the patients in group II in which highest percentage of approach being PTCA with stent (83.3%) followed by stent only (16.7%). However the difference was not statistically significant (p>0.05).

The mean PTCA balloon inflation pressure and stent inflation pressure was 9.6±1.8 ATM and 14.5±1.4 ATM in group I where as in group II this pressure were 9.8±1.5 ATM, 15.0±1.3 ATM respectively. The mean PTCA balloon inflation time and stent inflation time was 22.5±6.5 sec and 23.4±6.3 sec in group I where as in group II this time were 23.5±9.5 sec, 23.0±8.6 sec respectively. No statistically significant mean difference (p>0.05) was found between two groups of patient in terms of inflation pressure and its duration.

Table IV: Distribution of study subjects by procedural complications (N=60).

Procedural complications	Group I (n=30)		Group II (n=30)		chi value	p value
	n	%	n	%		
None	18	60.0	6	20.0	4.59	0.023 *
Coronary spasm	3	10.0	3	10.0		
Side branch occlusion	5	16.7	8	26.7		
Abrupt vessel closure	0	0.0	3	10.0		
Coronary thrombosis	0	0.0	1	3.3		
Coronary dissection	1	3.3	4	13.3		
Significant arrythmias	0	00.0	1	3.3		
Hypotension	3	10.0	3	10.0		
Congestive heart failure	0	0.0	1	3.3		

*= Significant at the level of p<0.05

Group I= Patients with CTnI≤ 0.4 ng/ml
Group II= Patients with CTnI>0.4 ng/ml
p value reached from chi square test

Table IV shows the procedural complication of the study subjects. Among group I patients, 18(60%) had no procedural complication, remaining 12(40%) patients had various type of procedural complication as mentioned above. But patients in group II had more procedural complications, only 6 (20.0%) of them had no procedural complication. Analysis shows significant difference (p<0.05) between the two groups.
Regarding ECG changes at different stage of intervention, no statistically significant difference (p>0.05) was found between two groups before

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

intervention. Just after the procedure there were significant differences ($p<0.05$) in ECG changes between two groups. ECG changes were also found during interventional procedure which were also significantly higher in number ($p<0.01$) among the patients in group II. The most frequent changes during the procedure were sinus tachycardia (10% vs 26.7%), PVC (10% vs 23.3%), ST depression & T inversion (0% vs 16.7%), sinus bradycardia (6.7% vs 6.7%), T inversion only (0% vs 13.3%), VT (0% vs 3.3%).

Regarding in-hospital complications of the study subjects. It was found that in-hospital complications were significantly higher ($p<0.01$) among the patients in group II. In group I, two thirds (66.7%) of the patient did not have any complication, whereas among group II patients, only one eighth (13.3%) had no complication. The only complication among group I patients was recurrent anginal pain, where as among group II patients, the most frequent complications were recurrent anginal pain (50%) followed by significant arrythmias (16.7%), death (10%), Q wave MI (6.6%) and cardiogenic shock (3.3%).

In this Study the logistic regression analysis of rise of cardiac troponin I with selected variables was done. It was found that post procedural cardiac troponin I rise was significantly correlated with procedural complications ($P<0.01$) and ACC/AHA type C lesion ($P<0.05$); but no statistically significant correlation was found with other variables such as age > 65 years, Poor LV function (EF<40%) & ACC/AHA type B lesion.

It was found that the mean duration of post intervention hospital stay was higher among the group II patients (4.9±2.3 days) compared to group I patients (2.9±1.0 days) and the mean difference was statistically significant ($P<0.01$). It was found that proportion of post intervention hospital stay for 1-3 days was higher in group I patient (83.3%) compared to group II patient (33.3%) whereas hospital stay of 7 days and above was higher in group II patients (16.7%) compared to group I patients (0.0%).
In this Study in group I only 3 patients had recurrent anginal pain which was 5 in group II. Patients in no group experienced significant arrythmias, cardiogenic shock, congestive heart failure, Q wave MI or repeat revascularization in the from of repeat PCI/CABG. There was no death in any groups of patient.

DISCUSSION

This prospective study was carried out in the National Institute of Cardiovascular Diseases (NICVD), Dhaka during the period of January 2006 to December 2007. Over this period a total of 80 patients with stable coronary disease who underwent PCI were included in this study. Among them 50 patients had post procedural cardiac troponin I level ≤ 0.4 ng/ml and out of these fifty patients 20 were excluded because of their unability to do repeat CTnI estimation at chest pain. So 30 patients with post procedural CTnI ≤0.4 ng/ml were considered as group I and remaining 30 patients with post procedural cardiac troponin I > 0.4 ng/ml were considered as group II.

In this study, the occurrence of post procedural CTnI elevation and any events that occurred during and after the procedure, as long as the patients remained inside the hospital were observed. Patients were also followed up at day 30 for any adverse events over telephone by making quiry directly to the patients and or to their relatives.
The mean age of the study subject was 48.9±9.4 years with a range from 29 to 66 years. The mean age of group I patients was 48.4± 9.1 years and 49.3±9.7 years for group II patients. But the mean age difference was not statistically significant ($p>0.05$) between two groups. The age structure was consistent with Momenuzzaman et al[17] a similar interventional study regarding PTCA and plain stent.

The study revealed that highest percentage of attending patients were male (91.65%) with male/female ratio being 10.9:1. However no statistically significant sex difference ($p>0.05$) was found between two groups of patient. Similar sex difference was found by Rahaman et al[18].

Among the risk factors, smoking was the commonest in both groups (60% and 66.7% in group I and group II respectively) followed by hypertension (40%), diabetes (23.3%) Dyslipidemia (16.7%), family history of IHD (16.7%) in group I patients, whereas in group II patients after smoking, hypertension (43.3%) occupied the second highest order of frequency followed by diabetes mellitus (20%), family history of IHD (16.7%), dyslipidaemia (13.3%). Nageh et al[19] showed that smoking was the commonest association in both group of patients (70%) followed by hypercholesterolemia (63%), family history of IHD (48%), hypertension (46%),

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

diabetes mellitus (18%) However this dissimilar pattern of distribution of risk factors may be due to ethnic and cultural differences among the study population.
The mean number of risk factors was 1.7 in group I and of 1.6 in group II patients, but no statistically significant difference (p>0.05) was found between two groups of patient. Similar pattern of risk factors was found by Malik et al[20], Rahman et al[18].

The mean body mass index was 24.8±4.1 in group I patients and 25.3±4.4 for group II patients. No statistically significant mean difference (p>0.05) was found between two groups of patients. Subsets of patients in each group with stable coronary disease who underwent interventional procedure were CCS class I (26.7% vs 23.3%), C,C,S class II (40% vs 46.7%) CCS class III (33.3% vs 30.0%) and no statistically significant difference found between two groups of patient (p>0.05).

Mean percent of ejection fraction (EF) was 50.5±6.9% in group I and 49.9±6.1% in group II. Although statistically there was no significant difference (p>0.05) between two groups but mean percent of EF was higher in group I than group II. This is comparable with study of Fuchs et al[11] (48±13% vs 46±13%).

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The mean number of risk factors was 1.7 in group I and of 1.6 in group II patients, but no statistically significant difference (p>0.05) was found between two groups of patient. Similar pattern of risk factors was found by Malik et al[20], Rahman et al[18].

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In terms of number of vessels intervened, single vessel PCI was the highest (73.3%) in group I patients, whereas double vessel PCI was the highest

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

(53.3%) in group II patients. No patient had undergone triple vessel PCI in any group. Similar pattern of distribution of study subjects seen in study conducted by Ricciardi et al[21].

In the present study, it was found that type A lesion (43.3%) was the commonest coronary lesion in group I patients, followed by type B (40%) and type C (16.7%), where as in group II patients type B was the commonest coronary lesion (43.3%), followed by type C (40%), type A (13.3%), mixed type (3.3%) lesion. There was statistically significant difference (P<0.05) between two groups of study subject. This difference in lesion morphology among the study subjects were similar as Nageh et al[19]. Similar differences were also found in Kini et al's study[15].

The most common pattern of percutaneous coronary intervention was PTCA with stent (79.95%) followed by stent only (20.05%). But the distribution of various devices in Nageh et al's study[19] was as follows, balloon angioplasty-13%; stent-78%, rotational atherectomy with or without stent insertion-9%. Because of the lack of availability of non balloon devices in our NICVD setting as well as high cost and marginal risk benefit ratio of those devices, may influence our decision to limit our exercise on stent with or without PTCA. However in this study the difference between the two groups of patients was not statistically significant (p>0.05).

In this study PTCA Balloon inflation pressure & time and stent inflation pressure & time did not show any statistically significant difference (p>0.05) between two groups. Average PTCA balloon inflation pressure was 9.7±1.7 ATM, stent inflation pressure was 14.8±1.4 ATM. PTCA balloon inflation time was 23±8.0 sec and stent inflation time was 23.2±7.5 sec.

Commonest procedural event was side branch occlusion both in group I and II (16.7% vs 26.7%). Coronary dissection ranks second (13.3%) in group II, whereas coronary spasm and hypotension ranks second (10% for each complication) in group I.

No abrupt vessel closure or congestive heart failure had taken place in group I, where as abrupt vessel closure, coronary spasm and hypotension ranks 3rd (10% for each complication) in group II patients. Congestive heart failure, significant arythmias, coronary thrombosis ranks 4th in group II (one case for each complication). Difference regarding these complications was statistically significant (p<0.05) which was supported by both Nageh et al19 and Fuchs et al[11].

Distribution of study subjects by ECG changes before the procedure showed no statistically significant difference (p>0.05). But per procedural and post procedural ECG changes showed statistically significant difference (p<0.05) between two groups. This can be explained by peri procedural myocardial injury[22].

Distribution of in-hospital complications following PCI were significantly higher (p<0.01) among patients within group II. Recurrent anginal pain was the commonest complication in both groups (33% vs 50%). In this study 5 patients developed significant arrythmias, 1 patient developed cardiogenic shock, 2 patients developed Q wave MI and 3 patients died in group II. Whereas no such events occurred in group I. No patient in any group required repeat PCI or CABG. Distribution of in-hospital complications with increasing tertile of post procedural CTnI showed patients with greater than 5 times elevation of CTnI from the upper reference limit were associated with increased in-hospital complication than patients with equal or less than 5 times elevation of CTnI (p<0.01). This observation was compatible with study done by Fuchs et al[11]. In terms of Major adverse cardiac events (MACE) patients with post procedural CTnI rise greater than 20 times the upper reference limit had increased number of major adverse cardiac events than patients with equal or less than 20 times the upper reference limit (p<0.01).

On logistic regression analysis it was found that procedural complications and ACC/ AHA lesion type C had statistically significant correlation (p<0.05) with CTnIrise. This correlation was supported by the statistical analysis conducted by Nageh et al[19], though variables such as poor LVEF (<40%), increased age (>65 years) were not correlated significantly (p>0.05). This disparity is probably due to small sample size of this study which was too small to give accurate result by logistic regression analysis.

The mean duration of post procedural hospital stay was 2.9±1 for group I patients and 4.9 ± 2.3 days for group II patients. The mean duration was significantly higher (p<0.05) among patients with in group II than in group I. The procedural and post procedural events were relatively more frequent in

Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022

group II patients. They were targeted for extra care in hospital settings. So the patients in group II stayed for longer period in hospital compared to group I patients.

Each patient who survived at discharge were followed at day 30 over telephone. They and or their relatives were asked for the same in hospital complications that were observed during the post procedural period. Patients in both groups experienced no complications at 30 days follow up except recurrent anginal pain. In group I, 3 (10%) patients experienced recurrent anginal pain where as in group II 5(18.5%) patients experienced recurrent anginal pain. There was no statistically significant difference between two groups ($p>0.05$).

CONCLUSION

The study reveals that CTnI rise at peri procedural MI level was observed in 37.5% of patients with stable coronary disease undergoing elective PCI. From this study it can be concluded that post procedural marked rise of CTnI (greater than 5 times the upper reference limit) is significantly associated with increased in-hospital adverse clinical out come in stable coronary disease, but has no incremental risk of short term (30 days) adverse clinical out come.

Conflict of interest: None.

Funding: The authors received no financial support for the research, authorship or publication of this article.

Ethical issue: Ethical clearance certificate was obtained prior to the conduction of this study. No ethical violation was done during the study.

Acknowledgments: Not applicable.

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Accepted reviewed version date: 14 March 2022

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Received date: 15 Feb 2022
Accepted reviewed version date: 14 March 2022