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Original Research Article

STUDY OF RISK FACTORS, CLINICAL PROFILE AND ANGIOGRAPHIC PROFILE IN YOUNG PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Abstract

Background: An increasing trend of coronary artery disease (CAD) in young patients under 40 years of age has been noticed among the South Asian population in recent years, particularly in Indians but there is limited data on clinical and angiographic profiles in these patients.

Methods: This is an observational study done on young (aged less than 40 yrs) thrombolysed STEMI patients presented to a Tertiary care Hospital. Clinical, Biochemical, and Angiographic data were analyzed. $P<0.05$ is considered as statistically significant and multivariate logistic regression analysis was done to determine the predictors of in-hospital outcomes.

Results: 100 patients were included in the study with a mean age of 34.3 ± 4.70 years and male to female ratio of 19:1. 10%, 16%, 59%, and 75% of patients were hypertensive, diabetics, alcoholics, and smokers respectively. 47% of patients have dyslipidemia and 66% of patients have hyperhomocysteinemia. 38% had insignificant CAD and 14% of patients had multivessel CAD. The mean Gensini score in our study population was 49 ± 35. 38% of patients had experienced in-hospital MACE and regression analysis has shown Killip class III/IV and Low LVEF as independent predictors of in-hospital MACE and TG/HDL ratio had a significant correlation with CAD severity.

Conclusion: Low LVEF and severe hemodynamic instability (Killip class III/IV) at presentation were poor prognostic markers and a TG/HDL ratio of 4.77 was found to be an independent predictor of severe CAD with a sensitivity of 77% and specificity of 82%.

Introduction

Coronary artery disease (CAD) is the leading cause of mortality worldwide and by 2020, will be the leading cause of disability. The most common among all acute coronary syndrome (ACS) patients is acute ST elevation MI in India [1]. Acute Myocardial infarction (AMI) represents one of the catastrophic events in the natural history of coronary artery disease. Although individuals younger than 40 years of age account for only 3% of all patients with coronary artery disease, they are not entirely immune from CAD [2]. In recent times the trend of the disease has changed. Acute MI has been found in young age groups more frequently in past years [3]. It has also been observed that CAD tends to occur at a younger age in Indians than in other groups, with more severe and extensive angiographic involvement. Briefly, 4-8% of patients with acute MI are less than 40 years of age. Persons in the middle age group are in the maximal productive phase of their lives with maximum family and social responsibility. Getting affected by the disease at this age leads to tremendous loss to the family and community. Moreover, it carries significant morbidity, psychological impact, and financial burden for the patient and their family when it occurs at a young age as the productive age group is being affected.

Premature coronary heart disease has been demonstrated to be three times higher in Indians when compared to subjects of similar age in the Western World. A contributory factor, that may explain the abnormalities in the lipid profile of Indians, is the high-risk atherosclerotic diet, which promotes overnutrition, obesity, and diabetes. There is also a possibility that genetic abnormality may account for the abnormal lipid

levels and premature coronary heart disease in Indians [4]. Studies have indicated that in young South Asians tobacco use, ghee intake, raised fasting glucose, high cholesterol, and parental history of cardiovascular disease are associated with premature acute myocardial infarction [5]. Smoking (72%), hypercholesterolemia (52%), and a family history of coronary artery disease (35%) were among the more prevalent risk factors in young adults. Other less common causes related to MI in young adults are hypertension, diabetes, reduced levels of physical activity, obesity, etc. Cardinal features of coronary artery disease among Indians compared to other populations [6]. 2-4-fold higher prevalence, 5-10 years earlier onset of first MI, 5-10-fold higher rate of MI and death in young (< 45 years), Lower prevalence of conventional risk factors, Higher prevalence of emerging risk factors. Although there have been many studies related to myocardial infarction (MI) in aged patients, comparatively few studies are there on young patients and South India in particular. Since there have been no previous studies aimed at STEMI in the younger population in this part of our country, we aimed to identify the risk factors, clinical profile, and coronary angiographic characteristics of young adults presenting with ST-elevated myocardial infarction. We selected an age cut-off of 40 years to define a premature CAD, based on previous Indian studies [7, 8].

Material and Methods

This prospective observational study was conducted in the Department of Cardiology. Institutional ethical approval was obtained from the ethics committee. All the patients were informed of the study before the data collection based on the procedure and each participant provided informed consent before participating in the study.

The study included young adult patients aged below 40 years who had been diagnosed with acute ST-elevation myocardial infarction (STEMI) and were admitted to the intensive cardiac care unit of the Department of Cardiology. The diagnosis of MI was made based on clinical history, ECG findings, and cardiac enzymes.

Inclusion criteria

1. Patients diagnosed with MI based on the ECG and Cardiac enzymes
2. Aged below 40 years
3. Those with ST elevation MI

Exclusion criteria

1. Aged above 40 years
2. With old MI, NSTE-ACS
3. Patients who were not thrombolysed
4. Complete data on the patient is not available
5. Not willing to participate in the study

A detailed history was taken in all the patients about the onset and duration of chest pain and a thorough physical examination was done as per the proforma. The first ECG along with right-sided chest leads was recorded at the earliest after admission and subsequently at 8-hour intervals on the first day, daily ECG for the duration of stay in ICCU and after that as per need. Patients were monitored for any clinical changes and ECG changes. Cardiac enzymes (CK-MB & Troponin), Serum Lipid profile, Homocysteine levels, Lipoprotein(a) levels, and hs-crp levels were done in all the patients. Transthoracic Echocardiography was done for all the patients. Due to local Government policy, a Coronary Angiogram was done 2 days after thrombolysis which is followed by coronary angioplasty after 2 days ineligible patients, and no patient in our study underwent primary PCI.

Statistical Analysis: Statistical analyses were performed using Medcalc® (V.16.8.4) for Windows, by MedCalc software (https://www.medcalc.org/) and Microsoft Excel 2007 for Windows by Microsoft Cooperation. - Categorical and numerical variables were expressed in percentage and mean (±standard deviation) respectively. Numerical variables were tested with independent samples t-test, and categorical variables were tested using the Chi-square test.

Results

The mean age of the study population is 34.3 ± 4.70 years. Male (95%) to female (5%) and the gender ratio among the study population was 19:1. Among the study group, 10% and 16% had Hypertension and Diabetes respectively. A family history of coronary artery disease is present in 12% of the population. The risk factor of smoking was present in 75% of the study population. The mean duration of smoking among the study population is 9.32 ± 6.37 pack years. The risk factor of Alcohol consumption was present in 59% of the study population.

The mean value of creatine kinase (CK-MB) among the study group is 158.22 ± 72.59 IU/L (28-375 IU/L). The mean values of hs-CRP and Lp (a) in our study were 0.63 mg/l (0.04 – 4.6 mg/l) and 16.44 ± 8.56 mg/l (2.3 – 39 mg/l) respectively. The mean value of Homocysteine was found to be 22.14 ± 21 µmol/l (3.1 – 75µmol/l). The mean level of Triglycerides was 186.5 ± 65.07 (96 – 451) mg/dL, Total cholesterol was 173.16 ± 35.78 (101 – 266) mg/dL, HDL cholesterol was 40.94 ± 10.19 (23 – 79), LDL cholesterol was 95.95 ± 32.05 (36 – 184) mg/dL and that of VLDL cholesterol was 36.70 ± 13.66 (19 – 98) mg/dL. The average TG/HDL ratio was 4.56 ± 2.09. The mean value of left ventricular ejection fraction at the presentation before thrombolysis is 43.21 ± 6.61 % (32 – 61%), 11 (11%) patients had severe MR, Mean E/A ratio was 1.24 ± 0.77 and Mean TAPSE was 1.71 ± 0.4 cm. Among these STEMI patients, 38% had insignificant CAD on Coronary angiogram (30 out of 80 patients among the anterior wall MIs and 8 out of 20 patients in the inferior wall MI). Among the 62 patients who had significant lesions in an invasive coronary angiogram, 48 patients had single-vessel disease, 13 had Double-vessel disease and 1 patient had Triple-vessel disease. Mean Gensini score (as calculated by an independent examiner) in our population was 49 ± 35.

Among the study population, 38 patients (38%) had in-hospital MACE (1 patient may experience more than 1 MACE). 18 patients had an Ejection fraction less than 40% after thrombolysis, and 21 patients needed hemodynamic support in the form of either and/or inotropic, vasopressor, or ventilator support (invasive/non-invasive). Finally, 6 patients had experienced clinically apparent ventricular arrhythmias namely ventricular tachycardia and/or ventricular fibrillation (VT/VF).

Predictors of In-Hospital Outcome: Predictors of in-hospital major adverse cardiac events (MACE): A multivariate regression analysis was done based upon predictors in univariate regression analysis and parameters with known clinical impact. We found that only Killip III/IV Class at presentation and low LV-EF as independent predictors of in-hospital MACE. Interestingly, there is no significant association between the type of thrombolytic agent used (STK or TNK) and in-hospital MACE, after the correction of confounding variables.

Table 1: Logistic regression analysis for composite endpoint in hospital-MACE

Variable	*Univariate analysis*		*Multivariate analysis*	
	OR (95% CI)	*p*	*OR (95% CI)*	*p*
Killip III and IV (yes = 1)	3.17 (1.78 – 5.01)	0.009	2.76 (1.71- 4.17)	0.027
Systolic Blood Pressure	0.81 (0.47 – 1.92)	0.034	0.88 (0.411 – 1.98)	0.093
LV-EF	0.77 (0.52 – 1.18)	0.042	4.050 (2.12 – 6.17)	0.041
AWMI (yes =1)	2.74 (1.71 – 3.61)	0.033	2.01 (1.58 – 2.89)	0.19
H-T (hours)	1.81 (1.08 – 2.82)	0.051		
TNK (yes =1)	0.90 (0.81 – 1.22)	0.411		

Interestingly, smoking was found to be a predictor of Insignificant CAD after systemic thrombolysis, irrespective of the thrombolytic agent used. In addition, high HDL cholesterol was also found to be a predictor of Insignificant CAD (which includes complete recanalization) post-thrombolysis. The analysis did not show any superiority of tenecteplase over streptokinase in terms of recanalization, suggesting Insignificant CAD after thrombolysis.

In cardiovascular medicine, the Gensini score, one of the most widely used scoring systems, is an example where a zero value indicates no luminal stenosis within the coronary artery tree, representing patients without

coronary artery disease (non-CAD group). TG/HDL ratio, age, and DM were found to have a significant correlation with the Gensini score on bivariate analysis.

The results of multiple linear regression analysis showed that there was a positive correlation between the TG/HDL ratio and Gensini score which was significant with r = 0.611, p <0.05 even after being controlled by confounding variables.

The determinant coefficient value R2 is 0.582 and thus it can explain the variability of the Gensini score by 58.2% while other factors influence the rest. The AUROC curve has shown a significant association (p<0.05) between the TG/HDL ratio and the severity of CAD as assessed by the Gensini score. We found an optimal cut-off of 4.77 with a sensitivity of 77% and specificity of 82% for detecting severe CAD (defined as a Gensini score of more than 30).

Table 2: Logistic regression analysis for CAD post-thrombolysis

Variable	*Multivariate analysis*	
	OR (95% CI)	*p*
AWMI (yes =1)	1.17 (0.87 – 1.76)	0.441
Smoking (yes =1)	2.26 (1.35 – 3.16)	0.046
Hs CRP	1.41 (0.72 – 2.1)	0.214
TG	0.81 (0.57 – 1.05)	0.055
HDL	1.93 (1.12 – 2.88)	0.037
TNK (yes =1)	1.87 (1.11 – 2.91)	0.061

Table 3: Bivariate analysis for predictors of CAD

Variable	*Correlation coefficient (r)*	*95% CI*	*P*
TG/HDL	0.766	0.417 – 0.911	0.021
Age (years)	0.578	0.322 – 0.87	0.046
Gender (male)	-0.124	-0.457 – 0.021	0.180
DM (yes)	0.611	0.066 – 0.838	0.007
Smoking (yes)	-0.077	-0.311 – 0.188	0.056
Hypertension (Yes)	-0.218	-0.0887 – (-0.027)	0.261
STK (yes)	0.439	0.182 – 0.728	0.091

Table 4: Multivariate analysis for predictors of CAD severity

Variable	*Standardized coefficient (β)*	*p*	*R^2 Linear*
TG/HDL	0.611	0.021	0.582
Age (years)	0.511	0.133	0.217
DM (Yes)	0.244	0.092	0.302

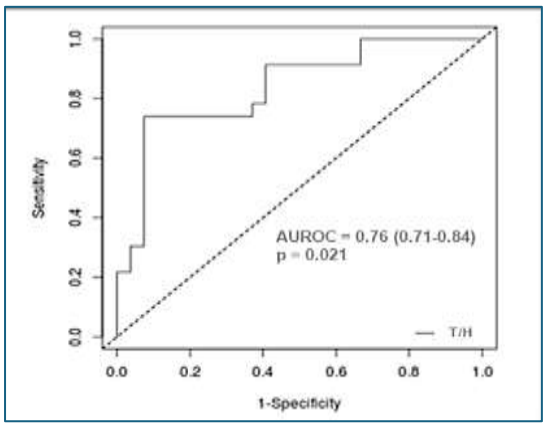

Figure 1: ROC curve between CAD severity and TG/HDL Ratio

Discussion

Despite coronary vascular disease particularly STEMI being the most significant cause of mortality, which in recent times is associated with a lower shift of mean age for presentation among south Indian adults, very limited studies have described the presentation, and outcomes of STEMI, likely because of lack of resources and funding. The present study was intended to examine demographic characteristics of young thrombolysed STEMI patients and predictors of in-hospital outcomes, including in-hospital MACE (includes mortality) and angiographic outcomes in this population group with first STEMI. The mean age of the study population was 34.3 ± 4.70 years; this is consistent with the mean age (32-36 years) of ACS in the young Indian population as documented in other studies [9-12].

Among the 100 patients in our study, males form the predominant subgroup (95%) with males: female ratio of 19:1, this disproportionate burden (more than 90%) of Young STEMI is documented in other Indian studies too [9, 10, 13] probably a higher rate of alcohol and smoking abuse, higher rate of stress exposure and relative protection of women due to hormonal effects may play a role, but the true reasons for this disparity needed to be evaluated conclusively. 10% (n=10) and 16% (n=16) of patients were hypertensive and diabetic respectively. A similar proportion of prevalence of hypertension (10-20%) [9, 10, 12, 14, 15] and diabetes (10%-25%) [12, 14, 15] in young STEMI is reported in other Indian studies; however, few studies have reported a higher incidence of Diabetes and Hypertension as high as more than 40%. The disparity for these results is unknown, but underdiagnosis before presentation may be a plausible cause. A family history of CAD is present in 12% of patients, and a similar proportion (7-18%) is consistently reported in many Indian young ACS patients. [9, 14, 15]

Alarmingly about 60% of our study population are alcoholics and 75% are smokers with a mean pack year of almost 10 pack years; these findings demand an urgent need for education and other primary prevention strategies in young STEMI patients. Of the STEMI patients presented to our unit young STEMI constituted 14.5% (924/134), a proportion which represented too in other Indian studies. Unfortunately, only 81.3% of (134/109) young STEMI patients were eligible for systemic thrombolysis (9 patients were excluded in the final data analysis). Delayed presentation resulting in ineligibility for thrombolysis was mainly pre-hospital pre-hospital. In India, the median time from the onset of symptoms to hospital arrival was 300 min in STEMI patients (more than double the delay reported in developed countries [range from 140 to 170 min]). The benefits of early thrombolytic therapy are well-established well-established. The mean absolute reduction in mortality per hour of delay is 1.6 (+/- 0.6) per 1,000 patients [16]. In the hospital, it took a further 50 min (door-to-needle time, D2NT) to undergo thrombolysis in India [17] (Guidelines recommend a D2NT of less than 30 min [18]. A similar similar delay i.e. median D2NT was 72 min (mean of 104.7 min) (in fact higher) in management is found in our study. The majority of patients have chest pain as a presenting complaint, 95%, and the majority have AWMI, 80%. Mean Killip class at Presentation was 1.47 with a mean systolic blood pressure of 113.7mmHg (+/- 23.64 mmHg). All (100%) STEMI patients received aspirin, clopidogrel, and a Statin on presentation to our hospital. Our use of aspirin and clopidogrel on admission seems to be more than satisfactory and compares well with other studies of the [19] developing world in general. An ACEI was administered to over 70% of patients at admission. ACCESS investigators who studied over 11,000 patients (including younger and older patients) with ACS in the developing world reported the use of ACEIS to be 68% [19]. Intravenous beta blockers were not used in the acute management of STEMI in the present study. However, 60% of patients received oral beta blockers during the initial assessment, Underutilization of intravenous beta blockers persists and should be analyzed in future studies. The in-hospital routine biochemical evaluation found dyslipidemia in 41% of the population group and even studies done by other Indian investigators found a similar proportion (30-50%) with a mean total cholesterol of 173.16 (+ 35.78) mg/dl [9, 11, 13]. In our study we found a TG/HDL ratio of 4.55, 3 independent investigators who evaluated this ratio in Indian young ACS patients found a similar value ranging from 4.8-5.392,94,96] We also studied the levels of lipoprotein a (LP[a]), homocysteine (HC) and hs-CRP in our population group and found that 7%, 70% and 71% of patients respectively have elevated levels. Interestingly our population had very low LP(a) levels comparatively, i.e., It was found elevated in more than 25% patients in many Indian young ACS patients. [9, 11, 13]

Hyperhomocysteinemia is found in varying proportions in our country's young STEMI patients ranging from 19.2% in a study done by Sinha et al. [13] to as high as 70% in different studies [9, 11, 12]. The mean Ejection fraction was 43.21% which was slightly higher compared to 37% found in a study done by Surrender

Deora et al. [9] and comparable (41.5%) to a study done by Prajapati et.al. [10] Invasive coronary angiogram showed recanalized vessels or insignificant CAD, single vessel Disease (SVD), double vessel disease (DVD), triple vessel disease (TVD) in 38%, 48%, 13%, and 1% respectively. The mean Gensini score in our population was 49 ± 35, to our knowledge no Indian study in young STEMI patients specifically evaluated this score. The majority of patients have SVD similar to many Indian studies through proportion ranges from 55-70% [9-11, 15], and the mean pool from these studies showed a value of 57.5%. TVD is seen in the lowest proportion in our study; a similar observation is seen in Young STEMI patients in general and the mean pool from other Indian studies [9-11, 15] showed a value of 5.11%.

Regression analysis for recanalization and the presence of insignificant CAD in post-thrombolysis coronary angiogram showed smoking and low TG/HDL ratio as independent predictors, interestingly type of thrombolytic agent (Streptokinase [STK] vs. Tenecteplase [TNK]) has no impact on recanalization rates, partly might be due to our study's insufficient power to analyze multiple co-variables. To make the analysis more robust, we also analyzed the predictors of the severity of CAD as assessed by the Gensini score. The results of multiple linear regression analysis showed that there was a positive correlation between the TG/HDL ratio and Gensini score ($p = 0.021$) after being controlled by confounding variables. The determinant coefficient value R2 is 0.582 which means that the TG/HDL ratio after being controlled by confounding can explain the variability of the Gensini score by 58.2% while the rest is influenced by other factors (though the presence of Diabetes mellitus and advanced age were found in binary analysis this finding was not seen after correction for confounding variables). This is in line with previous studies which showed that the TG/HDL ratio is a powerful independent indicator of extensive coronary disease [20-24].

In recent years, several studies have suggested that an increase in triglycerides and a decrease in HDL is a form of atherogenic dyslipidemia. Triglycerides or HDL alone does not always describe the overall cardiovascular risk it has, whereas when combined between triglycerides and HDL to be a single ratio, it will have a better power to predict cardiovascular disease [25] because this index will indicate the presence of sdLDL (small dense LDL) [26, 27]. Though there are no Indian studies which evaluated the optimal cutoff of assessing the severe CAD (as per Gensini score), we found that a cut-off of 4.77 in AUROC having an optimal sensitivity of 77% and specificity of 82% for detecting severe CAD (defined as Gensini score of more than 30). In-hospital MACE in our population was 38%. In multivariate regression analysis, only low LVEF and Killip III/IV class at presentation were found as independent predictors of in-hospital MACE. Even in regression analysis, Tenecteplase failed to show superiority over streptokinase in the reduction of in-hospital MACE; this finding stems from the fact that we used too many variables in the regression analysis for which our study sample may not be adequately powered. The true incidence of these events varies in different parts of the world and depends on the perfusion therapy used (PCI vs. thrombolysis), the type of thrombolytic agent used (STK vs. TNK), E the extent of facilities available to tackle complications, the degree of expertise, etc. Because the definition of in-hospital MACE differs in different studies and also lacks robust data in young STEMI patients, in particular, Indians make data difficult to compare.

Conclusion

This prospective observational study involved 100 young thrombolysed STEMI patients, predominantly male. The mean age and risk factor proportions aligned with other Indian studies. Hyperhomocysteinemia was present in 66% of patients, and 7% had elevated Lp(a) levels. Most patients had anterior wall STEMI, with chest pain as the common complaint. In-hospital MACE occurred in 38% of patients. Regression analysis identified low LVEF and Killip III/IV class as predictors of in-hospital MACE, and a high TG/HDL ratio correlated with CAD severity.

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