Pattern of Dyslipidemia in different type of Myocardial Infarction

Md. Zahid Alam¹, Aparna Rahman², Shabnam Jahan Hoque³, S.M. Rezaul Irfan⁴, Md. Babul Miah⁵, Mohammad Shakhawat Hossain⁶

Abstract

Background: Dyslipidemia is one of the main risk factors with prognostic significance in relation to coronary heart disease. Aggressive treatment has been recommended in acute coronary syndrome (ACS). We examined pattern of dyslipidemia in ST Elevation myocardial infarction (STEMI) and Non- ST elevation myocardial infarction (NSTEMI). We also compare the lipid status in between two types of myocardial infarction (MI).

Methods: This cross sectional observational study was carried out enrolling 100 subjects with ST elevation and Non ST elevation Myocardial Infarction, in the Department of Cardiology, BIRDEM General Hospital, Shabbag, Dhaka, over a period of six months from January 2012 to June 2012. Fasting lipid profile was done in next morning of admission in both type of MI.

Results: Mean age and gender difference was significant between STEMI and NSTEMI. Mean Cholesterol (chol), Triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) were not statistically significant between male and female groups. All mean cholesterol, TG, HDL, LDL were significantly high in older age group. The Mean cholesterol (220.7±28.15 vs208.4±20.9), triglyceride (182.8±34.4 vs 147.4±28.9), HDL (35.14±5.7 vs 41.65±3.8) and LDL (160.7±26.2 vs148.3±16.8)were also statistically significant between STEMI and NSTEMI groups (p<0.05).

Conclusion: Dyslipidemia is the dominating coronary risk factors. It could be concluded that significant differences are observed between two types of MI. Lipid status is relatively more uncontrolled in ST elevated MI and must be managed with all possible therapeutic modules to minimize further complications.

Key Words: ST-segment Elevation Myocardial Infarction (STEMI), NSTEMI (Non-ST-segment Elevation Myocardial Infarction), Acute Coronary Syndrome (ACS), Myocardial Infarction (MI), Cholesterol, Triglyceride(TG), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL).

Introduction:

Coronary artery disease (CAD) particularly myocardial infarction secondary to atherosclerosis of coronary arteries remains the leading cause of morbidity and mortality worldwide.¹ Atherosclerosis is a chronic, multifocal immuno-inflammatory; fibroproliferative disease of medium sized and large arteries mainly driven by lipid accumulation.² Elevated levels of total and low density lipoprotein cholesterol (TC and LDL-C),elevated levels of triglycerides (TG) and low levels of high density lipoprotein cholesterol(HDL-C) are important risk factors for CAD.³ LDL-C is considered as ‘bad cholesterol’ since too high level of this cholesterol is associated with an increased risk of coronary artery disease and stroke. The fundamental etiologic mechanism shared by all the forms of ACS is an imbalance between myocardial oxygen supply and demand. The most common cause of ACS is thrombus formation over a preexisting atherosclerotic plaque that has undergone disruption or erosion.⁴ The contrasting scenario is for the thrombus to fully occlude the arterial lumen, resulting in an acute Q-wave myocardial infarction (commonly with ST elevation). Unstable angina (UA)/ NSTEMI primarily caused by a nonocclusive thrombus benefit from a treatment regimen including anti-thrombotic and antiplatelet agents.⁵,⁶

Methodology:

This prospective observational study was done in the Department of Cardiology, BIRDEM General Hospital, Shahbagh, Dhaka during the period of January 2012 to June 2012 with the general objective to assess the pattern of dyslipidemia in hospital patient with ST Elevation versus Non ST Elevation Myocardial Infarction. During the study period 100 consecutive subjects aged 25-75 years suffering from STEMI and NSTEMI who presented with chest discomfort, palpitation or shortness of breath with either ECG change( ST elevation / depression ,T wave changes) or raised Troponin I were enrolled. Patient with chronic stable angina, unstable angina, non-cardiac chest pain, congenital or valvular cases and shortness of breath for other than ischemic heart disease were excluded from our study. Study subjects were collected from admitted patient in CCU referred from emergency department and also from in-patient department of the respective discipline with acute coronary syndrome. We did fasting lipid profile in next morning of admission and assess the pattern and differences of all parameter of lipid in two types of MI. Dyslipidemia was considered according to ATP III guideline with Serum Total cholesterol > 200 mg/dl ,TG >150 mg/dl, LDL >100 mg/dl, HDL <40 mg/dl (male) and <50 mg/dl ( female). Ethical permission was approved by appropriate authority. Data were analyzed using SPSS version 17.0 and p value <0.05 was considered as significant.
Results:

Age distribution of the study subjects (n=100)

100 consecutive patients were included in the study, of which 42 had STEMI and 58 had NSTEMI. The mean age and age distribution of the STEMI and NSTEMI groups were given in Table I.

Table I: Age distribution of the study

<table>
<thead>
<tr>
<th>Age group (Years)</th>
<th>STEMI (n=42)</th>
<th>NSTEMI (n=58)</th>
<th>Statistical test</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-39</td>
<td>06 (14.3)</td>
<td>11 (19.0)</td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td>08 (19.0)</td>
<td>14 (24.1)</td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td>16 (38.1)</td>
<td>19 (32.8)</td>
<td></td>
</tr>
<tr>
<td>60 and above</td>
<td>12 (28.6)</td>
<td>14 (24.1)</td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>48.36±10.18</td>
<td>51.29±11.55</td>
<td>0.024</td>
</tr>
</tbody>
</table>

Table II: Association between lipid profile with age (n=100)

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>25-39 (n=17)</th>
<th>40-49 (n=22)</th>
<th>50-59 (n=35)</th>
<th>≥60 (n=26)</th>
<th>Statistical test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>195.7±20.1</td>
<td>215±12.14</td>
<td>220±2.14</td>
<td>218±13.14</td>
<td>0.001</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>152.8±24.4</td>
<td>190±19.3</td>
<td>170±29.4</td>
<td>200±9.3</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>37.14±2.9</td>
<td>33.14±1.7</td>
<td>31.4±2.3</td>
<td>39.14±1.7</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>120.7±10.2</td>
<td>150.7±13.2</td>
<td>157±8.5</td>
<td>148±2.65</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Gender distribution of the study subjects (n=100)

In STEMI group, male were 33% and 67% were female. In NSTEMI group, 43% subjects were male and 57% were female (Fig-I). The male female ratio 1:2 (STEMI) and 1:1.3 (NSTEMI).

Table III: Association between lipid profile with sex

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>Male (n=39)</th>
<th>Female (n=61)</th>
<th>Statistical test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>209.2±28.1</td>
<td>218.7±21.3</td>
<td>0.058</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>167.8±35.1</td>
<td>174.3±30.1</td>
<td>0.326</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>37.4±3.8</td>
<td>38.7±4.9</td>
<td>0.163</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>152.3±18.2</td>
<td>159.1±24.2</td>
<td>0.136</td>
</tr>
</tbody>
</table>

Lipid profile of the study subjects (n=100)

Mean cholesterol, triglyceride, HDL and LDL were statistically significant between STEMI and NSTEMI groups.

Association between lipid profile with sex (n=100)

Mean cholesterol, triglyceride, HDL and LDL were not statistically significant between male and female groups which are shown in Table III.
Table IV: Lipid profile of the study subjects

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>STEMI (n=42)</th>
<th>NSTEMI (n=58)</th>
<th>Statistical test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>220.7±28.1</td>
<td>208.4±20.9</td>
<td>0.013</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>182.8±34.4</td>
<td>174.4±28.9</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>35.14±5.7</td>
<td>41.65±3.8</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>160.7±26.2</td>
<td>148.3±16.8</td>
<td>0.005</td>
</tr>
<tr>
<td>TC/HDL</td>
<td>6.2</td>
<td>5.0</td>
<td>-</td>
</tr>
<tr>
<td>TG/HDL</td>
<td>5.2</td>
<td>4.1</td>
<td>-</td>
</tr>
</tbody>
</table>

Discussion:

CAD is a complex and multifactorial process that manifests as stable angina, unstable angina or myocardial infarction. Myocardial infarction comprises a group of symptoms associated with obstruction of the coronary arteries. Myocardial infarction usually consists of ST elevation myocardial infarction and non ST elevation myocardial infarction. Both types of MI are frequently triggered by a thrombus formation on an eroded or ruptured atherosclerotic, lipid-rich plaque characterized by a thin fibrous cap. With the aim to show pattern of lipid profile in patients with ST Elevated versus Non ST Elevated Myocardial Infarction this present study was carried enrolling 100 subjects in the Department of Cardiology, BIRDEM General Hospital, Dhaka. The findings of the study are discussed on basis of related previous study concerning the chief objective of the study.

In STEMI group, male were 33.3% and 66.7% were female. In non-STEMI group, 43.1% subjects were male and 56.7% were female. The difference was statistically significant between STEMI and NSTEMI. Other study findings were comparable with the gender distribution of this present study. Mean total cholesterol (209.2±28.1 Vs 218.7±21.3), triglyceride (167.8±35.1 Vs 174.3±30.1), HDL (37.4±3.8 Vs 38.7±4.9) and LDL (152.3±18.2 Vs 159.1±24.2) were higher in female than male but statistically insignificant (p>0.05). Our results do not match with other study where these parameters (TG, HDL, LDL) were higher in male than female. Addulla Abdelaziz et al also describe high level of HDL in female than male, which is consistent with our study.

It was observed that mean age of STEMI and NSTEMI groups were 48.36±10.18 and 51.29±11.55 years respectively with an age range from 33 to 68 years. Majority of (38.1%, 32.8%) the respondents (STEMI vs NSTEMI) were found in the age group of 50-59. STEMI vs Non-STEMI subjects were found in 28.6% and 24.1% cases respectively above 60 years age group. Mean age difference was significant between STEMI and NSTEMI. Burazeri et al found that mean age of the study subjects with STEMI was 59.1±8.7 years in their study. Mean ±SD of total cholesterol, HDL, LDL were significantly high in 50-59 age group. Triglycerides was high in above 60 years age group. Differences of all parameter of lipid profile is statistically significant in different age group.

Dyslipidemia, manifested by elevated levels of total and low density lipoprotein cholesterol, triglycerides (TC, LDL-C, TG), low levels of high density lipoprotein cholesterol (HDL-C) is an important risk factor for CAD. These were the common laboratory findings among the subjects with STEMI and NSTEMI. Regarding pattern of dyslipidemia in our study [table IV], we found that high levels of TC (more than 200mg/dl), LDL (more than 130 mg/dl), TG (more than 150 mg/dl) were found in both STEMI and NSTEMI patients. Low levels of HDL (less than 40 mg/dl) were also found in both types of MI. Our results also revealed that the TC/HDL ratio was more than five (TC/HDL>5) and TG/HDL ratio was more than four (TG/HDL>4) in both groups. According to the American Heart Association, the goal is to keep TC/HDL ratio < 5 and TG/HDL < 4. A higher ratios indicates a higher risk of heart disease; a lower ratio indicates a lower risk. Our results also revealed that there was significant difference of lipid parameter between patients with STEMI and NSTEMI.

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There was a study regarding the comparison of lipid profile between MI and UA. Guler et al.\textsuperscript{16} and Esteghamati et al.\textsuperscript{17} who reported that low levels of HDL were significantly low in subjects with MI compared to those with UA. In our study group HDL is significantly low in STEMI than NSTEMI.

**Conclusion**

In conclusion, this study revealed that the pattern of dyslipidemia were same between two groups. But there is a significant difference of all parameters of lipid profile between STEMI and NSTEMI. Based on these results, we can recommend to pay more attention to serum lipids for the prevention of acute coronary syndrome. Furthermore, we have to control lipid profile more aggressively in STEMI to prevent further unwanted complications. Our study limitation is the absence of correlation of in hospital outcome with lipid status. Original sample size was small in relation to huge number of population, as well as the study period was only six months so large sample could not be included.

**References:**