

**Study of risk factors of acute myocardial infarction in adults below 35 years old in north Indian tertiary care hospital**

<sup>1</sup>Vijay Kumar, Senior Consultant Cardiologist, Medanta Heart Institute, Patna, Bihar, India

**Corresponding Author:** Vijay Kumar, Senior Consultant Cardiologist, Medanta Heart Institute, Patna, Bihar, India

**How to citation this article:** Vijay Kumar, “Study of risk factors of acute myocardial infarction in adults below 35 years old in north Indian tertiary care hospital”, IJMACR- March - 2023, Volume – 6, Issue - 2, P. No. 259 – 271.

**Open Access Article:** © 2023, Vijay Kumar, et al. This is an open access journal and article distributed under the terms of the creative commons attribution license (<http://creativecommons.org/licenses/by/4.0>). Which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

**Type of Publication:** Original Research Article

**Conflicts of Interest:** Nil

**Abstract**

Previous studies regarding risk factor of acute myocardial infarction in young patients are mainly from western countries. There has been a scarcity of data in Indian patients with only one study from Eastern India specifically addressing the above topic. Furthermore, previous reports have mainly studied risk factor in patient less than 40 years. We intend to study the younger population (less than 35 years) as there has been a rise in myocardial infarction in younger patients, especially in South Asians.

**Introduction**

Coronary artery disease (CAD) has become the major public health problem all over the world. It is the most common cause of mortality in the entire world<sup>1</sup>. The prevalence of CAD is four-fold higher in urban India and two-fold higher in rural India than in United States<sup>2</sup>. Ischemic heart disease (IHD) although relatively uncommon in young adults constitutes an important problem for such patients because of its devastating effect on their more active lifestyle. When the afflicted

individual is under the age of 40, the tragic consequences for family, friends, and occupation are particularly catastrophic and unexpected. In addition these patients may have different risk factor and angiographic profiles, clinical presentations and prognosis compared to older patients. There are several risk factors for IHD in young adults, most of whom have coronary atherosclerosis. As the number of atherosclerosis risk factors increases, so does the severity of coronary atherosclerosis in young adults. IHD in the absence of atherosclerosis, uncommon in older patients accounts for approximately 20% of cases in patients under age 45. Incidence of CAD in young Indians is about 12%–16%, which is higher than any other ethnic group.<sup>3, 4</sup> Median age of first heart attack in Indians is 53 years. About 50% of the CAD related-deaths in the young below 50 years, and about 25% of acute myocardial infarction (AMI) in India occurs under the age of 40 years.<sup>5, 6</sup> MI in young can be divided in to two groups, those with angiographically normal coronary arteries and those with coronary artery disease

(CAD). The prevalence of MI with normal coronary arteries has always been considered low, varying between 1% and 12%. However, it actually involves a considerable number of patients.<sup>7-9</sup> Young MI patients have higher percentage of normal coronary arteries. The MI in them can be caused by arteritis, thrombosis, embolisation or spasm. As is the case with venous thrombosis, coronary thrombosis can be seen in hypercoagulable states, such as protein C and protein S deficiency, antiphospholipid syndrome or nephrotic syndrome.<sup>10-13</sup> Coronary artery spasm can cause MI in patients with cocaine abuse<sup>14</sup> and also in association with alcohol binges.<sup>15</sup> Other unusual causes include hypertension, coronary aneurysms, mediastinal irradiations, valvular abnormalities, infective endocarditis. In the second group of young MI (those with CAD), it is mostly a result of atherosclerotic process, which starts in early childhood. Although traditional risk factors like hypertension, insulin resistance, diabetes mellitus, smoking, hyperlipidemia, physical inactivity and obesity explain most of CAD, 15-20% of those with CAD have no identifiable risk factors and therefore miss the opportunity for primary prevention.<sup>16</sup> Cigarette smoking appears to be the most common risk factor in young patients. The extent of smoking appears to be inversely related to the age at which IHD occurs. The other significant risk factors in this age group include deranged lipid profile, positive family history, obesity, hypercoagulable states, coronary anomalies, diabetes mellitus, and oral contraceptive use in young woman. Apart from traditional risk factors of myocardial infarction, recently many reports have suggested that increased lipoprotein A [Lp(a)], and hyperhomocysteinemia plays an important role in myocardial infarction and lower homocysteine levels are

associated with lower rates of coronary heart disease. Some 10% to 20% of cases of Coronary heart disease have been linked to elevated level of serum Homocysteine. Study done by Boushey et al. based on meta-analysis of 27 studies indicated that an elevation in homocysteine levels  $>15 \mu\text{mol/L}$  was associated with an increased risk of CHD, peripheral arterial disease, stroke and venous thromboembolism<sup>17</sup>. It was calculated that 10% of all CAD risk in population was due to elevated homocysteine. Prolonged lowering of homocysteine by 3-4  $\mu\text{mol/L}$  was associated with a 30-40% reduction in risk of CAD<sup>18</sup> Lp (a) is considered to be 10 times more atherogenic than LDL-C.<sup>19</sup> Relative risk of CAD is increased three-fold in males if Lp (a) levels are above 30 mg/dl.<sup>20,21</sup> Adverse effects are enhanced by high LDL-C and low values of HDL-C. Elevated levels of Lp[a] have been associated with a family history of myocardial infarction (MI) in asymptomatic individuals,<sup>22</sup> as well as with clinical MI,<sup>23</sup> Coronary artery disease,<sup>24</sup> and restenosis of coronary artery vein grafts. These associations have been observed in both white and Asian populations, primarily in middle aged or older men. Certain hematologic risk factors are potential predictors of CAD in young. For example, the factor V Leiden mutation in the factor V coagulation cascade gene, a well known risk factor for venous thromboembolic disease, also has been shown to increase the risk for MI. Levels of Lipoprotein (a) & homocysteine are genetically determined. It may be possible that there is increased prevalence of hyperhomocysteinemia and elevated lipoprotein (a) levels in Indian subcontinent leading early age of onset of first MI in Indians. Recently in studies it has been found that measuring APO-B and APO-A1 is better than measuring LDL-C and HDL-C as the type of HDL-C and LDL-C (size and

density) is more important rather than the absolute values. Small dense LDL-C particles are more atherogenic.<sup>25</sup> Young patients with IHD have a greater prevalence of anatomically normal epicardial coronary arteries than do older patients with IHD. However approximately half of the patients have single vessel atherosclerotic disease. The remainder have multi-vessel disease. The prevalence of left main coronary artery stenosis is approximately 5%. Multi-vessel disease is more likely in patients with multiple risk factors and diabetes. Study of risk factors in young adults with myocardial infarction is of particular interest considering the years of potential life lost. Because young, asymptomatic patients do not undergo medical investigations leading to the discovery of CAD, and also many young patients do not have traditional risk factors for CAD, the true prevalence of the disease has been grossly underestimated. Therefore, this study is done to find out the association of recently identified risk factors along with traditional risk factors with the premature onset of CAD in young patients hospitalised in our hospital who are 35 years old or younger.

#### **Aims & Objectives**

1. To study the risk factors for AMI in patients less than 35 years.
2. Type of presentation- chest pain or heart failure.
3. Type of MI-ST Elevation MI (STEMI) or Non-ST Elevation MI (NSTEMI).
4. Disease pattern in coronary angiography.

#### **Specific Objectives**

1. To evaluate cause of AMI at very early age in Indians.
2. To assess the relationship of new emerging risk factors such as homocysteine and lipoprotein A levels with decreasing age for Am

#### **Methods**

**Study Area:** The study was carried out on patients admitted with AMI in the department of Cardiology, Medanta Heart Institute, Patna

**Study Population:** This is a hospital based study done on patients 35 years or younger admitted to hospital with AMI. A detailed clinical history, physical examination, Electrocardiography (ECG), Biochemical, echocardiographic evaluation was done using a pre-test proforma.

#### **Inclusion criteria**

All patients aged 35 years or younger admitted to the department of Cardiology Medanta Heart Institute, Patna diagnosed with AMI. Most studies have used a cut-off point of 40 years and below to define young MI<sup>26,105,106</sup>, hence in this study patient aged 35 years and younger were included.

The final diagnosis of AMI was based on the following criteria:

- Ischemic chest pain lasting 20 minutes or more
- ECG evidence of myocardial injury-  
≥ 0.1mv ST elevation in two contiguous leads other than V2-V3 where the cut-off point of ≥ 0.2 mv in men ≥ 40 years; ≥ 0.25 mv in men < 40 years or ≥ 0.15 mv in women.

OR

New horizontal or down sloping ST depression ≥ 0.05 mv in two contiguous leads and/or T wave inversion ≥ 0.1 mv in two contiguous leads with prominent R wave or R/S ratio >1.

Positive biomarkers- CK-MB, cardiac Troponins.

#### **Exclusion Criteria:**

1. Patients < 18 years of age
2. Patients > 35 years of age
3. Patients who refused to give consent

**Study Period:** Two and a half year from May 2015 to April 2017.

**Sample Size:** Total of 50 patients were analysed

**Sample Design:** Purposive sampling

**Study Design:** Cross-sectional study of CAD risk factors among North Indians.

Subjects studied were men and women aged 18-35 years.

**Parameters studied:**

**A. History**

- Age
- Sex
- H/O of hypertension
- H/O of diabetes mellitus
- H/O of smoking and duration
- H/O of alcohol consumption and duration
- H/O of substance abuse- cocaine
- Family H/O premature CAD (defined as CAD occurring below the age of 65 in women and 55 in men)
- Symptoms- chest pain or dyspnea

**A. Physical Examination**

- Systolic and diastolic blood pressure was taken in both upper limbs in seating position
- Height/Weight
- Waist-Hip ratio
- Body Mass Index

**B. Lab Values**

- Lipid profile Total Cholesterol
- High density lipoproteins (HDL) Low density lipoproteins (LDL) Triglycerides (TG) Apo A-1 Apo B Lp (a)
- Inflammatory markers Homocysteine, C-reactive protein Glucose metabolism Fasting glucose

**Electro Cardiography** Stemi Or Nstemi

**Coronary Angiography**

Single vessel or multi-vessel disease Discrete lesion or diffuse disease

**Study tools:** Preformed proforma to record patient data, ECG, Cardiac enzymes, lipid profile, inflammatory markers and other relevant blood investigations, echocardiography (2D-ECHO and Doppler), catheterization lab to analyse disease pattern on coronary angiography.

**Study technique**

1. Proper informed and written consent was taken from all patients in the study
2. Detailed history was taken as per preformed proforma.

**Statistical Tools Employed**

The statistical analysis was done using SPSS (Statistical Package for Social sciences) version 17/20 statistical Analysis Software. The values were represented in Number (%) and Mean + SD. The following Statistical formulas were used.

1. **Mean:** To obtain the mean, the individual observations were first added together and then divided by the number of observations as per following formula:

$$\bar{X} = \frac{\sum X}{n}$$

No. of observation (n)

Here summation is denoted by the sign  $\Sigma$ . The individual observation are denoted by the sign X, number of observation is denoted by n, and the mean by  $\bar{X}$ .

**Standard Deviation**

For sample size of more than 30 following formula were used to calculate standard deviation ( $\sigma$ ).

$$\sigma = \sqrt{\frac{\sum (X - \bar{X})^2}{n}}$$

For sample size of less than 30 following formula were used to calculate standard deviation ( $\sigma$ )

$$\sigma = \sqrt{\frac{\sum(X - \bar{X})^2}{n - 1}}$$

4. To test the significance of difference chi square test and student 't' test were used as appropriate.

**Chi square test:**

$$\chi^2 = \frac{\sum(O - E)^2}{E}$$

Where O = observed frequency

E = Expected frequency

**Student 't' test:** To test the significance of two means the student 't' test were used

$$t = \frac{\bar{X}_1 - \bar{X}_2}{s \sqrt{\frac{1}{n_1} + \frac{1}{n_2}}}$$

Where  $s^2 = \frac{(N_1 - 1)SD_1^2 + (N_2 - 1)SD_2^2}{N_1 + N_2 - 2}$

Where  $\bar{X}_1, \bar{X}_2$  are means of group 1 and group 2  
 N1, N2 are number of observation group 1 and group 2  
 SD1, SD2 are standard deviation in group 1 and group 2

**Analysis of Variance:** Analysis of Variance (ANOVA): The ANOVA test is used to compare the within group and between group and between group variances amongst the study groups i.e. the three different sealers. Analysis of variance of these three sealers at a particular time interval reveal the differences amongst them. ANOVA provided "F" ratio, where a higher "F" value depicted a higher inter-group difference.

F = Mean of Sum of Between Group Differences / Mean of Sum of within Group Differences

Differences	Sum of Squares	Df	Mean Square	F
Between Groups	A	N <sub>1</sub>	X=A/N <sub>1</sub>	X/Y
Within Groups	B	N <sub>2</sub>	Y=B/N <sub>2</sub>	

6. Level of significance: "P" is level of significance

- P>0.05 Not significant
- P<0.05 Significant
- P<0.01 Highly significant
- P<0.001 Very highly significant

**Results**

In this study the risk factors, clinical and angiographic profile of 50 young patients of age between 18-35 years with AMI were studied from period May 2015 to April 2017 presenting to our hospital.

No of patients were fifty in this study. Most of the patients (66%) being within the age of 31-35 years and 2% of the patients being in the age group of 21-25 years. The youngest patient was 22 years old and the oldest was 35 years old. 80% of the patients were males. The mean age of female patients was 33.3 years while for males was 31.82 years.

Table 1 & Figure 1 show the presenting symptoms of the patients. The most common symptom in young patients with AMI was chest pain, which was present in 94% of the patients, followed by sweating (50%) and breathlessness (26%).

**Table 1: Frequency of the Symptoms**

Symptoms	No of patients	Percentage
Chest pain	47	94%
Sweating	25	50%
Breathlessness	13	26%

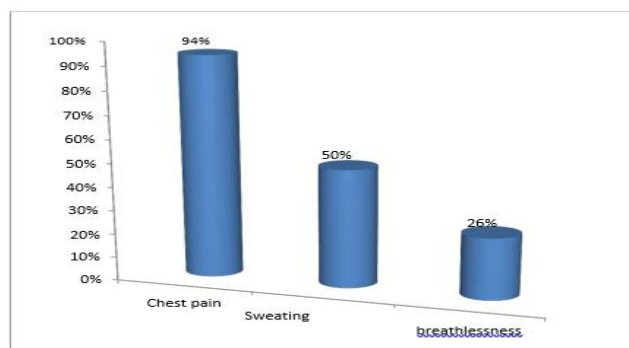


Figure 1: Frequency of Symptoms

Table 2 displays the percentage of various risk factors in young AMI patients. Smoking was most common risk factor for AMI (72%) in the young adults, hyperlipidaemia being the second common risk factor (68%).

**Table 2:** Risk factors of MI in young adults

Risk Factors	Percentage (number)
Smoking	72%(36)
Hyperlipidemia	68%(34)
Homocysteine level >15mg/dl	42%(21)
BMI > 25	32%(16)
Diabetes mellitus	24%(12)
Hypertension	18%(9)
Family history of premature CAD	18%(9)

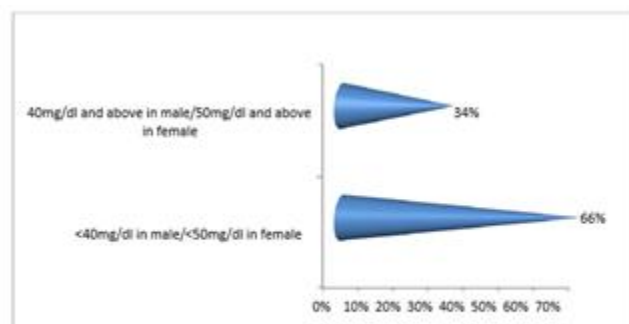
32% patients had BMI value of >25kg/m<sup>2</sup>. 24% of the patients were diabetic, of which 10% were newly detected. Hypertension and a family history of premature coronary artery disease (CAD) each formed 18% of the riskfactors.72% of the patients had multiple risk factors for AMI. 26% had a single risk factor, while 4% had none of the risk factors. Substance abuse (cocaine) was present in 4% patients.

The serum triglyceride (TG) levels was elevated in 76% of the patients in which 54% patients had levels between 150-199 mg/dl (borderline high) and 22% had levels above 200 mg/dl (high). The mean TG 177.2 mg/dl [Table3].

**Table 3:** Risk factors (lipid profile) of MI in young adults

Risk Factors (lipid profile)	Percentage (number)
Hyperlipidemia	68%(34)
Triglyceride >150mg/dl	76%(38)
HDL-C <40mg/dl	68%(34)
LDL-C > 130mg/dl	54%(27)
Lp (a) >30mg/dl	26%(13)

HDL-C was below 40 mg/dl in 68% of patients overall but when taking gender distinction into account (<40 mg/dl in males and < 50 mg/dl in females) it was below normal in 66% of patients. The mean HDL-C value was 30 mg/dl [Figure 3].



**Figure 3:** HDL-C levels

LDL-C levels was below 100 mg/dl in 16% of the patients, it was borderline high in 18% of the patients, high in 20% of the patients while 16% of the patients had very high levels of LDL-C. The mean LDL-C value was 133.9 mg/dl. Lipoprotein (a) levels were high (>30 mg/dl) in 24% of the patients. The mean Lp(a) was 25.63 mg/dl.

The Apo B/Apo A-1 ratio was normal in 9 patients. It was elevated in 82% of the patients with 8% being under the moderate risk category and 74% under high risk category. The mean ratio was 1.69.

Anterior wall MI was found in 64% of the patients and 28% of the patients had inferior wall MI. Lateral wall MI was present in 4% of the patients. In our study, 64% patients had single vessel disease (SVD) which was seen on coronary angiography, followed by double vessel disease (DVD) (24%) and 4% had multi-vessel disease. **Normal coronary angiography was found in 8% of cases [Figure 4].**



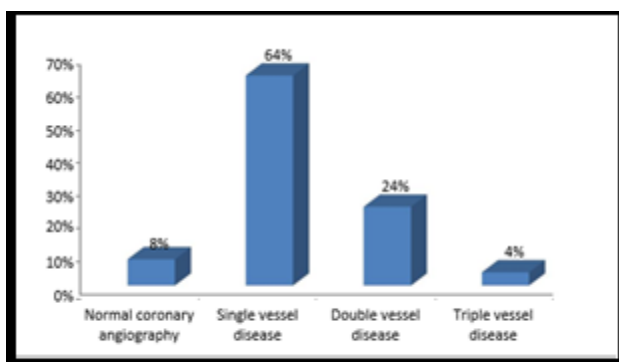


Figure 4: Vessel involvement on angiography

68% of the patients had involvement of the Left Anterior Descending (LAD) Artery, 34% had involvement of the Right Coronary Artery (RCA) and 16% had involvement of the Left Circumflex Coronary Artery (LCX) [Figure 5].

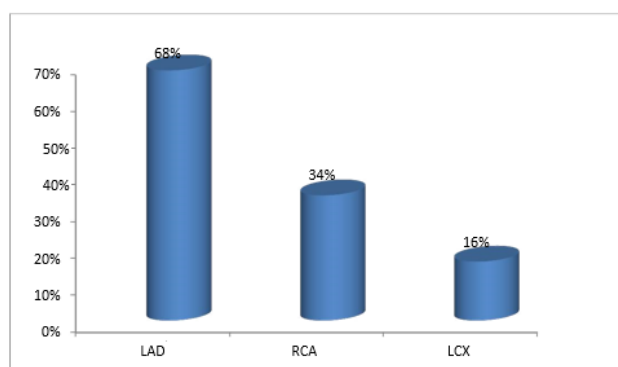


Figure 5: Major vessel involvement on angiography

## Discussion

This study was conducted in a group of patients who presented to our hospital with AMI. The study was conducted in the department of cardiology, Medanta Heart Institute, Patna for a period of 2<sup>1/2</sup> years from May 2015 to April 2017. This study was done with an intention to evaluate the risk factors contributing to the rising incidence of AMI at a younger age in Indian population. AMI is rare before 35 years of age, but there is a rising trend in young population now a days. Most of the patients belonged to higher range of age group that is 31-35 years which contributed 66% of the patients and only

2% of the patients were below 25 years of age. The mean age was 33.3 years for females and 31.82 years for males. The distribution of age group showed a striking increase in the disease with increasing age. This in accordance to previous studies which also showed increasing trend with increasing age even in young patients. In a study done by Sricharan K.N. et al. also the maximum number of the patients (70%) were within in the age of 35-40 years and 3.33% patients being in the age group of 25-30 years.<sup>26</sup> Also in a study by Jayesh Prajapati et al. the mean age was 34.5±4.7 years.<sup>27</sup> Male sex is an important risk factor for IHD. In our study 80% of the patients were male. Study by Jayesh et. al. also 89.9% patients were male.<sup>28</sup> Thus the demographic profile of our patients was similar to previous studies.

Tobacco smoking is an established conventional coronary risk factor for CAD. Casual association between tobacco chewing (smokeless tobacco) and CAD is found in some case control studies.<sup>28</sup> Tobacco increases the risk of cardiovascular disease by raising blood pressure, damaging vascular endothelium, increasing LDL-cholesterol oxidation, and lowers the HDL-cholesterol. Smoking was found to be most prevalent risk factor (72%) in our study. This was in line with a previously reported prevalence of 77% in Swiss patients aged ≤35 years by Schoenenberger and colleagues.<sup>104</sup> Studies done in India (Jayesh et al, Sricharan K.N. et. al and Gohar Jamil et. al.) also showed smoking the most prevalent risk factor.<sup>[27-30]</sup> Thus an effort should be made to educate people about the hazards of cigarette smoking and people should be educated at an early age to avoid smoking and smoking cessation programmes need to be established In this study 24% of the patients were diabetic, of which 10%

were newly detected and hypertension was present in 18% of the patients. BMI more than 25kg/m<sup>2</sup> was seen in 32% of the patients. Family history of premature CAD was present in 18% of the patients. In a study done in London among young patients with MI, positive family history of premature CHD was found in 39% of participants.<sup>31</sup> In a study by Zimmerman et al, family history of CAD was more common only in young men<sup>32</sup>, while in young patients with MI, positive family history of premature coronary artery disease was found in 39% participants. Study by Sricharan et.al.<sup>26</sup> and Gohar Jamil et. al.<sup>27</sup> also showed similar trend. However, in the study by Zhahid Hassan et. al. as compared to diabetes mellitus (12%), hypertension was more prevalent (40%)<sup>25</sup>. The excess burden of CAD among South Asians appears to be primarily due to dyslipidemia that is characterised by: high levels of ApoB, triglycerides, Lipoprotein(a), borderline high levels of LDL-C, low levels of HDL-C and ApoA1. Total cholesterol levels and LDL-C levels are correlated with extent and severity of CAD in Asian Indians as in whites. But at any given total cholesterol or LDL-C level, Asian Indians have a greater CAD risk than whites. In our study hyperlipidemia was present in 68% of the patients, with 54% of the patients having increased LDL-C levels and LDL-C levels were in very high range in 16% of the patient. HDL-C levels was below 40 mg/dl in 68% of patients. In previous studies also hyperlipidaemia was found to be a common risk factor. In study by Sricharan et. al hyperlipidemia was the second most common risk factor (36.67%).<sup>106</sup> Hyperlipidemia was documented in 46 percent of study population in the study by Zhahid Hassan et al.<sup>25</sup> Similar results were obtained in study by Jayesh Prajapati et. al.<sup>28</sup> Therefore; Asian Indians with

dyslipidemia should be treated as aggressively as if they had a CAD risk equivalent—similar to the treatment of patients with diabetes or heart disease.

Lp(a) appears to be a major risk factor in Asian Indians as compared to whites. Elevated LP(a) found in 35-40% of all Indians.<sup>32,33</sup> High LP(a) levels are highly correlated with the severity of ACS, recurrent events, poor prognosis, and increased mortality.<sup>34</sup> A high level of LP(a) is shown to be the most prevalent dyslipidemia in our young patients with premature CAD. LP(a) levels are governed almost exclusively by race, ethnicity, and genetics, unlike other lipids, where the levels are influenced by age, gender, diet, and other environmental factors. The effect of LP(a) on the atherogenicity is not additive but multiplicative. It constitutes an important inherited risk factor for atherosclerosis and is also regarded as biological marker for familial CAD.

In our study Lp(a) levels were above 30 mg/dl in 24% of the patients representing an important risk factor in young patients with MI. In the study by Jayesh Prajapati et. al.<sup>28</sup> (21.5%) patients had high level of LP(a)<sup>109</sup> similar to our study. Similarly in the studies by Schaefer EJ et.al (elevated in 21.5% of patients) and by Issar HS et al the Lp(a) level were significantly higher in young patients with MI as compared with controls.<sup>34</sup>

In our study the mean Lp(a) level was 25.63 where as in a study by Kamariya et. al. and Jayesh Prajapati et.al. the mean Lp(a) levels were 44.04±8.52 mg/dl and 37.1 ± 31.9 mg/dl in young patients with MI.<sup>28,35</sup> The lower mean level of Lp(a) in our study may be due to the fact most of the patients had levels skewed around 26-30 mg/dl.

Homocysteine levels are higher among Asian Indians than others. In India, most people adhere to a vegetarian diet and vegetarians have 3.0 times higher risk of



hyperhomocysteinaemia compared to those who eat non-vegetarian.

In our study homocysteine levels were above normal in 42% of the patients. Whereas, homocysteine levels elevated in 72.55% of the patients in study done by Puri A et al.<sup>36</sup> Also in the study done by Veerendrakumar Arumalla and K. Rajashekar Reddy<sup>37</sup> hyperhomocysteinemia was found in 66% of the patients with AMI. The slightly lower prevalence of hyperhomocysteinemia in our patients might be due to increased fish consumption and fruits. So, we should encourage higher intake of fruits and avoid overcooking of vegetables to prevent hyperhomocysteinemia. Our study showed that 72% patients had multiple risk factors, 26% had a single risk factor and 4% of the patients had no risk factors. In the study by Sricharan K.K. et al.<sup>26</sup> 46.67% of the patients had multiple risk factors for AMI, 46.67% had a single risk factor, while 6.67% had none of the risk factors. The most common presenting complaint in our study was chest pain (94%) followed by sweating (50%) and breathlessness (26%). The study by Sricharan et. al. showed that the most common symptom was chest pain, which was present in 90% of the patients, followed by sweating (50%), breathlessness (20%), restlessness (6.7%) and palpitations (3.3%).<sup>106</sup> However, in study by Zhahid Hassan et. al. 69% of the patients denied any chest pain.<sup>26</sup> The chest pain was absent in our study mainly in females and diabetics. There is now compelling evidence that the ApoB/ApoA1 ratio is a better index of the likelihood of vascular events than any of the corresponding cholesterol indices: the total cholesterol/HDL-C ratio, non-HDL-C/HDL-C ratio, or LDL-C/HDL-C ratio. ApoB/A-1 ratio > 1 associated with increased CV risk. Relation between risk and ApoB

is continuous, whereas at the extremes of HDL concentration in plasma the relation to risk is not certain. In the present study the Apo B/Apo-A1 ratio was elevated in 82% of the patients. The mean ratio was 1.69. In a study by Jayesh Prajapati et.al. the mean ratio was 0.76.<sup>37</sup> In our study most common presentation was with anterior wall MI which was found in 64% of the patients. It was followed by inferior wall MI in 28% and lateral wall MI in 4% of the patients. These results are in line with the prior studies done by Sricharan et. al. and Jayesh Prajapati et.al, where also anterior wall MI was most common presentation followed by inferior wall MI.<sup>29</sup> Regarding coronary angiographic findings, LAD was the most common coronary artery involved (68%) followed by RCA in 34% of the patients. The LCX was the least (16%) involved artery. Prior studies also (Zhahid Hassan et.al, Gohar Jamil et.al, Sricharan et.al.)<sup>25-28</sup> showed LAD as the most common coronary artery involved followed by RCA and LCX.

In our study, normal coronary angiography was seen in 8% of the patients. 64% of the patients had SVD whereas, DVD and multivessel disease was present in 24% and 4% of the patients respectively. Studies done by Zhahid Hassan et.al and Gohar Jamil et.al also showed that SVD was most common in young AMI patients followed by DVD and multivessel disease.<sup>26,27</sup>

Whereas in the study by Sricharan et.al, majority of the patients (57.14%) had SVD, followed by normal coronaries (22.45%). 16.3% had DVD and 4% patients had multivessel disease.<sup>26</sup> Thus it is evident from angiographic findings that young patients usually have SVD and less diffuse disease which is highly amenable to treatment with better preservation of myocardial function if they are offered timely intervention.

## Conclusion

1. Although AMI is less common in young adults <35 years of age, it constitutes an important problem for both patient and the treating physician.
2. AMI produces a devastating impact on the active life of young patients and it also produces financial constraints on their family and the community as they are the main earning population of the country.
3. The young patients have different risk factor profile, clinical presentation, angiographic presentation and prognosis in comparison to older patients.
4. In this study most of the patients were male suggesting male sex as a risk factor for the occurrence of AMI in young.
5. Smoking remained the most prevalent risk factor in young AMI patients with cocaine abuse contributing further to the increased risk amongst youngsters.
6. Next common risk factor was dyslipidemia characterized by high levels of ApoB, TG, Lp(a), borderline elevation of LDL-C, low levels of HDL-C and Apo-A1.
7. Regarding novel risk factors for accelerated atherosclerosis, Lp(a) and hyperhomocysteinemia contributed as an important risk factor AMI apart from traditional risk factors. Although hyperhomocysteinemia was slightly less prevalent here as compared to other parts of country because of more intake of non vegetarian diet and fruits. These risk factors usually get underdiagnosed. More detailed investigations of these risk factors should be done. These risk factors can be genetically determined, so these should be properly evaluated in children of families with premature CAD.s
8. Positive family of premature CAD is also quite common in younger patients and need to be properly

evaluated. Children of families with premature CAD should be screened for the causative factors for CAD in their families, so that the preventive measures can be taken at right time.

9. Female patients tends to have higher prevalence of diabetes and multiple risk factors when they present with AMI at younger age.
10. The most common presenting complaint was chest pain followed by breathlessness and sweating unlike older patients in whom atypical presentation and breathlessness is more common.
11. Anterior wall was most common territory involved followed by inferior wall and lateral wall.
12. The prevalence of SVD was much more common than DVD and multivessel disease
13. A sizeable proportion of the patients had normal coronaries.

Since young person form the main working population of the country and are the main financial back-up of their families, important steps should be taken to keep them healthy and prevent such a devastating disease to occur in them at an early age.

The most important thing is to do risk factor modification which is a quite challenging task. Since cigarette smoking is highly prevalent in young, preventive educational programmes along with smoking cessation clinics need to be established.

Diabetics and cholesterol education should be provided to the population especially the youth by medical personal on priority basis.

Young patients usually have discrete lesion which can be treated by angioplasty and if done early, much of the myocardium at risk can rescued. Thus, all the patients should be subjected to an early revascularization and risk stratification for better outcomes and prognosis.

## References

1. WHO The global burden of disease, update 2004; 11-12.
2. Goel PK, Bharti BB, Pandey CM, Singh U, Tewari S, Kapoor A, et al. A tertiary care hospital based study of conventional risk factors including lipid profile in proven coronary artery disease. *Ind Heart J* 2003; 55:234-240.
3. Negus BH, Williard JE, Glamann DB et al. Coronary anatomy and prognosis of young asymptomatic survivors of myocardial infarction. *Am J Med* 1994; 96: 354-8.
4. Mammi MVI, Pavithran P, Rahman PA et al. Acute MI in North Kerala. A 20-year hospital based study. *Indian Heart J* 1991; 43: 93-6.
5. Bahuleyan CG. Hospital data on coronary artery disease from North Kerala. In Vijayaraghavan G (ed). *Cardiovascular Disease Prevention Trivandrum* 54-9.
6. Girija G. Risk factors profile of patients with acute MI. In Vijayaraghavan G (ed). *Cardiovascular Disease Prevention Trivandrum*. 78-83. American Heart Association Heart and Stroke Statistical Update 1997; 26-7.
7. Alpert JS: Myocardial infarction with angiographically normal coronary arteries. *Arch Intern Med* 1996, 154(3):265-269.
8. Da Costa A, Isaaz K, Faure E, Mourrot S, Cerisier A, Lamaud M: Clinical characteristics, aetiological factors and long-term prognosis of myocardial infarction with an absolutely normal coronary angiogram; a 3-year follow-up study of 91 patients. *Eur Heart J* 2001, 22(16):1459-1465.
9. Bugiardini R, Bairey Merz CN: Angina with "normal" coronary arteries: a changing philosophy. *Jama* 2005, 293(4):477-484.
10. Padler FA, Comad AR. Myocardial infarction with normal coronary artery: A case report and review of literature. *Am J Med Sci*. 1997; 314:342-5.
11. Penny WJ, Colvin BT, Brooks N. Myocardial infarction with normal coronary arteries and factor XII deficiency. *Br Heart J*. 1985; 53:230-4.
12. Hamsten A, Norberg R, Björkholm M, de Faire U, Holm G. Antibodies to cardiolipin in young survivors of myocardial infarction: An association with recurrent cardiovascular events. *Lancet*. 1986; 1:113-6.
13. Fujimura O, Gulamhusein S. Acute myocardial infarction: Thrombotic complications of nephrotic syndrome. *Can J Cardiol*. 1987; 3:267-9.
14. Ross GS, Bell J. Myocardial infarction associated with inappropriate use of cocaine for treating epistaxis. *Am J Emerg Med*. 1992; 10:219-22.
15. Moreyra AE, Kostis JB, Passannante AJ, Kuo PT. Acute myocardial infarction in patients with normal coronary arteries after acute ethanol intoxication. *Clin Cardiol*. 1982; 5:425-30.
16. Smith SC Jr. Current and future directions of cardiovascular risk prediction. *Am J Cardiol*. 2006; 97: 28A-32A.
17. Boushey CJ, Beresford SAA, Omenn GS et al. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. *J Am med Assn* 1995; 274:269-271.
18. Tambe AB. Homocysteine and atherosclerotic vascular disease. *Cardiology Today* 2000; 4: 269-718. Akram H. al Khadra "clinical profile of young

- patients with acute MI in Saudi Arabia *International journal of cardiology* 2003; 91,9-13.
19. Lawn RM. Lipoprotein (a) in heart disease. *Sci Am* 1992;266:54-60.
  20. Maher VM, Brown BG, Marcovina SM, Hillger LA, Zhao XQ, Albers JJ. Effect of lowering LDL cholesterol on cardiovascular risk of lipoprotein (a). *JAMA*1995; 274:1771-4.
  21. Armstrong VW, Cremer P, Eberle E, Manke A, Schulze F, Wieland H, et al. The association between serum Lp (a) concentrations and angiographically assessed coronary atherosclerosis. Dependence on serum LDL levels. *Atherosclerosis* 1986; 62:249-57.
  22. Hoefler G, Harnoncourt F, Paschlce E, Mirti W, Pfeiffer KH, Kostner GM: Lipoprotein Lp[a]: A risk factor for myocardial infarction. *Arteriosclerosis* 1988;8:398-401.
  23. Durrington PN, Ishola M, Hunt L, Arrol S, Bhatnagar D: Apolipoproteins [a], AI, and B and parental history in men with early onset ischemic heart disease. *Lancet* 1988;1:1070-1073.
  24. Armstrong VW, Cremer P, Eberle E, Manke A, Schulze F, Wieland H, Kreuzer H, Seidel K: The association between serum Lp[a] concentrations and angiographically assessed coronary atherosclerosis: Dependence on serum LDL levels. *Atherosclerosis* 1986;62:249-2.
  25. Sweetnam PM, Bolton CH, Downs LG, Durrington PN, MacKness MI, Elwood PC, et al. Apolipoproteins A-I, A-II and B, lipoprotein(a) and the risk of ischemic heart disease: the Caerphilly study. *Eur J Clin Invest.* 2000; 30(11):947-56.
  26. Coronary artery disease in the young: A study of risk factors and angiographic characterization in the valley of Kashmir. Zahid Hassan et al, *International Journal of Scientific and Research Publications*, Volume 4, Issue 7, July 2014, ISSN 2250-3153.
  27. Study of Acute Myocardial Infarction in Young Adults: Risk Factors, Presentation and Angiographic Findings: Sricharan K.N. et al. ID: JCDR/2012/3853:1995.
  28. Risk factor assessment of young patients with acute myocardial infarction. Gohar Jamil et al. *Am J Cardiovasc Dis* 2013; 3(3):170-174.
  29. Akram H. al Khadra “clinical profile of young patients with acute MI in Saudi Arabia *International journal of cardiology*2003;91,9-13.
  30. Washington, DC: US Government Printing Office; Ser. 1, No. 15, DHHS Publication 81-1317, 1981.
  31. Avogaro P, Beittolo BJ, Cuzzolato G, Quinci GB, Belussi F. Plasma level of lipoprotein A-1 and apolipoprotein B in human atherosclerosis. *Artery.* 1978; 4: 385–394.
  32. Vergani C, Trovato G, Dioguardi N. Serum total lipids, lipoproteins, cholesterol, apoproteins A and B in cardiovascular disease. *ClinChim Acta.*1978; 87: 127–133.
  33. Sniderman A, Shapuiro S, Marpole D, Skinner B, Teng B, Kwiterovich PO Jr. Association of coronary atherosclerosis with hyperapobetalipoproteinemia (increased protein but normal cholesterol levels in human plasma low density (beta) lipoproteins). *ProcNatlAcadSci U S A.* 1980; 77: 604–608.
  34. Sniderman AD, Wolfson C, Teng B, Franklin FA, Bachorik PS, Kwiterovich PO Jr. Association of hyperapobetalipoproteinemia with endogenous hypertriglyceridemia and atherosclerosis. *Ann Intern Med.* 1982; 97: 833–839.

35. Austin M, Breslow J, Hennekens C, Buring J, Willett W, Krauss R. Low-density lipoprotein subclass patterns and risk of myocardial infarction. JAMA. 1988; 260: 1917–1921.