



Risk factor profile and Coronary artery disease pattern in young adults from Western part of India.

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ABSTRACT

Background : upcoming epidemiological studies point towards increasing incidence of coronary artery disease(CAD) along with earlier occurrence of the disease affecting the person in their prime time of life. We must be aware of this trends and should search for the specific reason for this occurrence.

Objective : The aim of the present study is to identify the presence of conventional and newer novel risk factors (hs-CRP, Homocysteine) among the young CAD patients and compare their risk factors with the coronary angiography(CAG) findings.

Materials and Methods : conventional and newer biomarkers of CAD were collected for all young patients presenting with acute coronary syndrome and their coronary angiographic study results were collected. Analysis of the risk factors parameters and CAG results were done, to compare this with the general population

Results : out of 152, 138 were male. Hypertension and Diabetes were 10.5% and 11.8% respectively. very high number of patients were Smoker 80% . only 42% patients had their BMI within normal limit. Cholesterol and LDL level were not significantly high. STEMI was the major presenting pattern with angiography showing single vessel disease and single lesion pattern higher in number

Conclusions: There is an increasing trend towards development of CAD in younger population with the trend towards less prevalent diabetes and hypertension and significant positive association with the smoking. Young CAD patients tend to present more frequently with ST elevation MI and more likely to have single vessel disease on CAG.

KEYWORDS : High sensitive C reactive protein, Homocysteine, Young coronary artery disease, Coronary angiography.

Introduction:

Coronary artery disease (CAD) is traditionally considered as disease of the male and dis-ease of old persons affecting in sixties of life. There is inadequate understanding of the pathophysiology of CAD and ACS and both are believed to be same and single disease. Young individuals are considered immune and if CAD or ACS affects young it is considered as exception and work up for non conventional risk factors and hyper coagulable state workup starts in the community. However, now with increasing availability of the re-search papers focusing on the epidemiology of CAD and ACS, it is clear that CAD is no more restricted to patients in sixties and there is increasing trend towards affection of the society in the young age that is < 40. [1]

Premature CAD is defined as CAD occurring below the age of 65 in women and 55 in men. [2][3][4]CAD in the young is defined as CAD occurring in patients less than 40 years of age. The traditional risk factors of HTN, hypercholesterolemia with high LDL and low HDL, smoking, and diabetes do not completely explain the higher prevalence of severe CAD.[5][6]

A British study found that among all patients admitted to a coronary care unit, the rate of a first myocardial infarction was five times higher among Indians than among native English when all age groups were examined. Yet, among the 30-39 age group, the rate among Indians was ten fold higher. Similarly, in Malaysian study, the incidence of CAD in young Indians was fifteen fold higher as compared with Chinese, and ten fold higher as compared with Malays. Inflammatory markers like C reactive protein and hs-CRP are also reported to be higher in CAD and reports of link with young CAD.[7][8][9][10]

there is an immense need for the data of these patients affected with CAD before the age of 40. If we can find out any single and more reason which makes these young population susceptible to this disease, it will give insight into the treatment and prevention of the disease in community. There are trials studying increased titres of certain biochemicals like malondialdehyde and protein carbonyl levels and lymphocyte DNA damage, glutathione lever, but it has not converted into practical benefit converting into clinical end point reduction.[11][12][13] we have performed this cross sectional study to collect the data

of young CAD patients and to analyse to find out the association of risk factors with the development of CAD.

Materials and Methods

Total of 152 patients of < 40 years of age, presenting with acute coronary syn-drome were included in this prospective, cross sectional study over the period of jan 2009 to dec 2013, in department of medicine, Sheth L G general hospital. Institutional ethical approval was obtained for the study protocol and written informed consent was taken from all participants.

The main inclusion criteria for the study were 1) Acute coronary syndrome as de-fined by cardiac markers elevation with either dynamic ECG changes or typical chest pain. 2) age less than 40 years at the time of development of first index event. All 152 patients underwent detailed history, clinical examination, biochemical tests (Blood sugar - fasting and post prandial, fasting lipid profile, total counts, haemoglobin, platelet count, ESR, HsCRP and Homocysteine), and CAG. The clinical presentation was divided into STEMI or NSTEMI, based on the ECG changes. Thrombolysis detailed were obtained including type of the drug administered and window period. life style and socio economical data were also obtained which included intake of alcohol, tobacco intake (smoked and smokeless), type of diet, daily exercise and intensity of daily exercise. family history of the premature CAD was also obtained.

Results

(1) Sex Distribution:

Out of total 152, 138 (90.7%) were male and only 14(9.3%) were female.

s(2) Incidence in various age groups:

The youngest person in our study was 26 years old. Out of total patients 7/152 (4.6%) were between 26-30 years of age, 36/152 (23.68%) between 31-35, 50/152(32.89%) between 36-40 and 59/152 (38.81) were between 41-45 years of age.

(3) Socio-economic Status

based on the classification given by Prasad, In our study 11.1%

(17/152) of patients belong to higher socioeconomic class, 42.1% (64/152) from the middle class and 46.7% (71/152) from lower socio-economic class.

(4) Life Style:

In our study, 79 out of 152, (51.9%) were having sedentary life style whereas 73 (48.1%) were doing manual work.

(5) HTN

HTN as defined by JNC 7, was found in 16 out of 152 (10.5%) patients in our study.

(6) DM

Prevalence of DM in our study was 11.84% (18 out of 152)

(7) Smoking:

In our study 80.26% (122/152) patients were smokers out of them 46% were

heavy smokers.

(8) OC Pills in AS :

In our study, only 1 patient found to be on oral contraceptive pills

(10) Obesity:

In our study 63/152 (42%) of patients had BMI in normal range. 37/152 (24.34%)

were overweight, 34/152 (22.36%) were mildly obese, 12/152 (7.89%) moderate and 6/152 (3.94%) were morbidly obese. 28/152 (18.42%) of total male patients and 101/152 (66.44%) of total female patients had waist circumference > 102 and > 88 cm. respectively.

(11) Lipids in ACS :

(a) LDL Cholesterol: 42 of 152 (27.63%) had LDL < 100 mg/dL, 46 of 152 (30.26%) had LDL between 100-130, 40 of total 152 (26.31%) had LDL between 130-160, and only 18 out of 152 (11.84%) had LDL between 160-190 and 6 of total 152 (3.94%) had very high LDL > 190 mg/dL.

(b) HDL :

114/152 (75%) of total male and 11/14 (78.5%) of female had their HDL cholesterol below the normal limit for their sex.

(12) Homocysteine and Young ACS :

In our study 112/152 (73.68%) patients had elevated homocysteine level.

(13) hs-CRP:

In our study 115/152 (75.65%) of patients had hs-CRP (> 3 mg/L)

(14) Coronary Angiography :

Incidence of TVD was significantly lower and SVD and normal angiographic findings were higher among young ACS patients. Incidence of normal coronary angiogram was 16/152 (10.52%) in our study.

Discussion :

We have very good understanding of coronary artery disease in terms of etiopathogenesis and pathophysiology but, whether the same applies when disease affects young people is not clear yet. There have been many studies in this field but being a small number of patients and regional factors prevent general application of the finding and there remains need of the study in our population. All of the study performed in young CAD patients showed preponderance of male, suggestive of hormonal protection enjoyed by females or acquired risk factors adopted by male life style, smoking being the top in this list. Tobacco smoking is an established conventional coronary risk factor for CAD. Tobacco increases the risk of cardiovascular disease by raising blood pressure, damaging vascular endothelium, increasing LDL-cholesterol oxidation, and lowers the HDL-cholesterol. On an average, as per NFHS III in Gujarat; prevalence of tobacco use by any form is 60.2% in men and 8.4% in women. Tobacco consumption was found to be most common addiction in our young ACS patients. It was found in 79% which is comparable to study done by Rohit V. Ram and Atul V. Trivedi in Gujarat. In our study majority of patients were tobacco chewer rather than smoker.

On comparing the prevalence of established coronary risk factors in young CAD and in elderly, a pattern emerges that Hypertension and diabetes is present in fewer young CAD and smoking is significantly higher in numbers in young CAD. [14] However overweight and obesity also remains significantly higher in numbers suggesting a development of lifestyle which promotes diabetes, hypertension in young.

Presence of normal and near normal LDL level were common in young CAD and the same was the finding in older patients presenting with the acute coronary syndrome. We now know that rather than the LDL level, it is the plaque rupture which causes acute coronary syndrome, and oxidised LDL and lipoprotein (a) and phospholipase A enzyme may be more important in deciding development of acute coronary syndrome. We could not find any single coronary risk factor which can be statistically significant in terms of development of ACS in compare to older CAD.

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 hyperhomocysteinemia compared to those who ate non-vegetarian. Homocysteine levels >15 μmol/L are found in 75-84% of subjects in India. The prevalence of hyperhomocysteinemia in our study is which is comparable to study conducted in younger subject by A.K. Puri et al. in India.

Strong evidence indicates that CRP is associated with CHD events. [15] Moderate, consistent evidence suggests that adding CRP to risk prediction models among initially intermediate-risk persons improves risk stratification. However, sufficient evidence that reducing CRP levels prevents CHD events is lacking. We have found very high Hs-CRP probably because of we have evaluated in ACS which is itself an inflammatory condition. Mean Hs-CRP level in our study were 16.3±22.5 mg/L which is correlate with study done by Tenzin Nyandak et al. in Delhi, India

There is now compelling evidence that the apolipoprotein (Apo) B / ApoA-1 ratio is a better index of the likelihood of vascular events than any of the corresponding cholesterol indices: the total cholesterol / high-density lipoprotein cholesterol (HDL-C) ratio, non-HDL-C / HDL-C ratio, or low-density lipoprotein cholesterol (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. Appreciating these distinctions should allow appropriate use of the ApoB/ApoA-1 ratio as a simple, single, summary index of the lipoprotein-related risk of vascular disease. In our study mean Apo B/A-1 ratio is only 0.76. This could be because of most of our patients have low HDL.

Young patients tend to present with STEMI rather than with unstable angina or NSTEMI, and this also reflected in the angiographic pattern like single vessel disease with the single lesion in the vessel. This also suggest the need for finding the cause for the plaque rupture in young patients rather than searching for the cause for the development of the plaque. Smoking is the aetiology which has been shown to cause LDL oxidation and making the plaque unstable. Presence of normal or near normal angiography also can be explained by the small plaque which had ruptured and caused acute coronary syndrome, with development of thrombus over it which gets lysed with the treatment and on angiography gives normal or near normal angiography result. [16] Normal Angiography may also be due to coronary emboli as the aetiology of the ACS. Coronary spasm may also be considered but none of our patient had history of cocaine use.

Although this being a prospective and real world scenario reflecting study, it has the limitation of small sample size with study focused on the patients presenting with the acute coronary syndrome, and study does not have control values in the general population of the same age group.

Conclusion :

Young coronary artery disease patients tend to present with STEMI rather than with UA/NSTEMI, had male preponderance and with smoking as the single most important risk factor with normal or near normal lipid profile. Overweight remains higher in young CAD patients and needs to be considered carefully as it may reflect develop-

ing traditional risk factors at an early age.

REFERENCES

1. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART STUDY). *Lancet* 2004; 364: 937–952. | 2. Enas EA. Coronary artery disease in women. *Indian Heart J.* 2001 May- Jun; 53(3): 282–2 | 3. Serdar Z, Aslan K, Dirican M, Sarandol E, Yesilbursa D, Serdar A. Lipid and protein oxidation and antioxidant status in patients with angiographically proven coronary artery disease. *Clin Biochem* 2006; 39: 794–803. | 4. Egred M. Myocardial infarction in young adults. *Postgrad Med J.* 2005; 81: 741–5 | 5. Reddy KS, Yusuf S. Emerging epidemics of cardiovascular disease in developing countries. *Circulation.* 1998; 97: 596–601. | 6. Goel PK. A tertiary care hospital-based study of conventional risk factors including lipid profile in proven coronary artery disease. *Indian Heart J.* 2003 May-Jun; 55(3): 234–40 | 7. Nyandak T. High Sensitive C-Reactive Protein (hs-CRP) and its Correlation with Angiographic Severity of Coronary Artery Disease (CAD). *JACM.* 2007; 8(3): 217–21. | 8. Ridker P. Clinical application of C - reactive protein for cardiovascular disease detection and prevention. *Circulation* 2003; 107: 363–369 | 9. Espiguero RA, Avanzas P, Sales JC, Aldama G, Pizzi C, Kaski JC. C-reactive protein elevation and disease activity in patients with coronary artery disease. *Eur Heart J* 2004; 25: 401–408. | 10. Imran Ahmed, Achyut Sarkar, Vascular Inflammation and Angiographic Severity of Coronary Artery Disease in Young Asian Indians *Journal of Cardiovascular Disease Research*, 2014; 5(1):15-21 | 11. Mutlu-Turkoglu U, Akalin Z, Ilhan E, Yilmaz E, Bilge A, Nisanci Y, Uysal M. In-creased plasma malondialdehyde and protein carbonyl levels and lymphocyte DNA damage in patients with angiographically defined coronary artery disease. *Clin Biochem* 2005; 38: 1059–1065. | 12. Beutler E, Duron O, Kelly B. Improved method for the determination of blood glutathione. *J Lab Clin Med* 1963; 61: 882–888. | 13. Paglia DE, Valente WN. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J Lab Clin Med* 1967; 70: 158–169. | 14. Klein L, Nathan S. Coronary artery disease in young adults. *J. Am. CollCardiol.* 2003; 41(4): 529–31 | 15. Guruprasad S, Rajasekhar D, Subramanyam G, Srinivasa Rao PVLN, Vanajakshamma V, Latheef K. High sensitivity C-reactive protein levels across spectrum and severity of coronary artery disease. *J. Clin. Sci. Res.* 2012; 3:126–30 | 16. Klein LW, Agarwal JB, Herlich MB, Leary TM, Helfant RH. Prognosis of symptomatic coronary artery disease in young adults aged 40 years or less. *Am. J. Cardiol.* 1987; 60:1269–72 |