Original article

Patterns and determinants of dyslipidaemia in ‘Young’ versus ‘Not so Young’ patients of coronary artery disease: a multicentric, randomised observational study in northern India

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KEY WORDS

Cholesterol
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A B S T R A C T

Aims: The aims of the study were to ascertain difference in lipid levels of ‘Young’ onset of coronary artery disease (CAD) (≤ 45 years) vs. ‘Not so Young’ onset of CAD (≥ 55 years) among north Indians and also to investigate determinants of ‘dyslipidaemia’ in CAD patients.

Methods: This was a prospective, multicentric, randomised, observational study carried in eight centres of UP, India. All blood investigations were performed employing a central laboratory.

Results: Out of a total 435 patients studied, 218 were in the ‘young group’ (YG) and 235 were in the ‘Not so Young Group’ (NSYG). Dyslipidaemia was more common in YG as evident by significantly higher levels of total cholesterol, triglycerides, low- and very low-density lipoprotein cholesterol as compared to NSYG. Diabetes, hypertension, urban lifestyle, and family history of CAD were found to be important determinants of dyslipidaemia in YG.

Conclusion: We conclude that lipid levels among north Indians are significantly higher in younger patients with CAD when compared with elderly.

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Introduction

Coronary artery disease (CAD) is one of the major causes of mortality and morbidity in elderly and middle aged populations of both the developed and developing countries. In the recent years, incidence of CAD in India has been reported to be in between 7–13%.^{1–6} South Asian ethnicity has been considered to be an independent risk factor for acute myocardial infarction (AMI). First myocardial infarction (MI) occurs at least 5–10 years earlier in Asian Indians with more severe,

extensive as well as malignant disease with increased complications and mortality.^{7–10} The INTERHEART study,¹¹ which had about 3.7% of patients from India, however, demonstrated that risk factors for AMI among south Asians were similar to that of other ethnic groups.

There is a strong possibility of genetic predisposition for CAD in the population from the Indian subcontinent which probably confers them a higher susceptibility to develop symptomatic CAD at a younger age. This may be due to a combination of one or more factors, one of which could be dyslipidaemia, with or without metabolic syndrome.⁷

Therefore, our objectives for this study were to ascertain difference in risk factors especially serum lipid levels of ‘Young’

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(CAD onset at ≤ 45 years) vs. 'Not so Young' (CAD onset at ≥ 55 years) onset of CAD in north Indian population and also to investigate patterns and determinants of 'dyslipidaemia' in patients inflicted with CAD.

Methodology

The study was carried out under the aegis of Uttar Pradesh Chapter of Cardiological Society of India (UPCSI), conceived and coordinated by the Research cell-UPCSI.

Selection of subjects and data collection

This was a prospective, multicentric, randomised, observational study, carried out on patients attending the out-patient/Emergency Departments of Cardiology or Medicine (which were mostly secondary/tertiary care teaching hospitals) in eight centres of UP; the largest state of northern India with a population of approximately 199 million. The study documents (e.g. protocol and informed consents in English and Hindi language and clinical proforma) were duly approved by respective Ethics Committees of all participating centres.

All centres employed a uniform colour coded clinical proforma for data collection designed jointly by Departments of Cardiology and Biostatistics, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India who also coordinated data collection, reporting of samples, and data analysis.

Anthropometric data, e.g. height, weight, and abdominal girth were collected using standardised study aids provided by the coordinating centre. Besides basal demographics, detailed clinical history, risk factor profile, and clinical examination with electrocardiogram (ECG) were done for each eligible patient. Risk factors for CAD were identified according to standard definitions.

We enrolled proven CAD cases from February 2007 to February 2009 after obtaining a written informed consent. Criteria for proven CAD were defined as: history of MI (chest pain with ECG changes and/or elevated cardiac enzymes) or Angiographically proven CAD ($>50\%$ stenosis in at least one major epicardial artery) or AMI cases (when recruited within 24 hours of onset of event) or history suggestive of ischaemic chest pain with ST-T wave changes on ECG, with/without positive cardiac biomarkers, diagnosed as either stable or unstable angina (in the opinion of treating physician). All consecutive patients of age at onset of disease ≤ 45 years (Young Group [YG]), were enrolled and thereafter, any subsequent patient with age at onset of disease ≥ 55 years (Not so Young Group [NSYG]), satisfying the inclusion criteria were enrolled in a 1:1 ratio, therefore, minimising selection bias.

All patients with age at onset of disease in the range of 46–54 years or taking any lipid lowering drugs in last 4 weeks or having secondary causes of dyslipidaemia (e.g. long standing uncontrolled diabetes, hypothyroidism, liver disease, nephrotic syndrome, etc.) were excluded from the study. Elderly patients ≥ 75 years, patients with debilitating disease, pregnant or lactating females, and patients of AMI reporting after

24 hours of onset of chest pain were also excluded from the study.

Of the 500 patients enrolled between February 2007 to February 2009, 453 patients were selected for final analysis (47 cases excluded for either missing blood reports or missing data regarding age of onset of disease or absence of clear diagnosis for inclusion and history of lipid lowering therapy in last 4 weeks of enrolment).

Biochemical estimations

Patients were investigated for fasting serum lipid profile and fasting plasma glucose by a central laboratory (Thyrocare Technologies Limited [CPL], Mumbai, India and Super Religare Laboratories Limited [Formerly SRL Ranbaxy Ltd.], Mumbai, India). Methodology used for serum cholesterol estimation was photometry using Bi-directionally Interfaced Fully Automated Analyzer and CHOD-PAP-Enzymatic End-point method. For triglyceride (TG) estimation GPO-PAP method was used. Direct enzyme clearance method was used for estimation of serum high-density lipoprotein cholesterol (HDL-c) and low-density lipoprotein cholesterol (LDL-c). Very low-density lipoprotein cholesterol (VLDL-c) levels were derived by a formula based on the Friedewald's report,¹² i.e. $VLDL-c = 0.20 (TG)$.

Lipid levels reported from both laboratories (using different methodologies) were tested to reveal a non-significant statistical difference and therefore, were clubbed together.

Statistical methods and sample size

For computation of minimum sample size it was assumed that the mean total cholesterol (TC) values in YG would be 180 ± 55 mg/dL and in NSYG 160 ± 55 mg/dL. For two independent sample *t*-test at *P* value of ≤ 0.05 with 90% power, the minimum sample size was estimated was 160 subjects per group. Assuming a 25% non-response rate the sample size was enhanced to 200/group.

The data was computed using SPSS 16.0. Data quality assurance and exploratory data analysis was performed before final analysis. Pearson's χ^2 test, student's *t*-test and univariate logistic regression analysis were used as applicable. A *P* value ≤ 0.05 was considered significant. All hypothesis testing was done assuming a two-tailed test.

Results

Of the 453 patients included in the final analysis, 218 were in YG (≤ 45 years) and 235 were in NSYG (≥ 55 years). A total of 394 (87%) patients enrolled were males including 193 (88.5%) in YG and 201 (85.5%) in NSYG. For the whole cohort mean values of demographics were: height 163.6 ± 0.8 cm, body weight 65 ± 10.5 kg, body mass index (BMI) 24.3 ± 3.3 kg/m² and abdominal girth 90.23 ± 9.4 cm.

On comparison of anthropometric measurements between the two groups, younger patients had higher body weight

Table 1
Baseline characteristics of study subjects.

Baseline characteristics	Young Group (≤ 45 yr) <i>n</i> =218	Not so Young Group (≥ 55 yr) <i>n</i> =235	<i>P</i> value
Mean age at inclusion (years $\pm$ SD)	40.61 $\pm$ 5.26	64.33 $\pm$ 6.58	NA
Sex			
Male, <i>n</i> (%)	193 (88.5)	201 (85.5)	0.40
Female, <i>n</i> (%)	25 (11.5)	34 (14.5)	
Mean height (cm)	164.46 $\pm$ 7.77	162.85 $\pm$ 8.53	0.03*
Mean body weight (kg)	66.41 $\pm$ 10.66	63.74 $\pm$ 10.34	<0.01*
Mean body mass index (kg/m ²)	24.52 $\pm$ 3.37	24 $\pm$ 3.28	0.105
Mean abdominal girth (cm)	90.53 $\pm$ 8.56	89.96 $\pm$ 10.12	0.513
Diagnosis			
Acute myocardial infarction, <i>n</i> (%)	160 (73.4)	170 (72.3)	0.884
Unstable angina, <i>n</i> (%)	28 (12.8)	39 (16.6)	0.322
Stable angina, <i>n</i> (%)	21 (9.6)	24 (10.2)	0.961
Asymptomatic known CAD, <i>n</i> (%)	9 (4.1)	2 (0.9)	0.050

**P* value <0.05 was considered to be significant. CAD: coronary artery disease, NA: not applicable, SD: standard deviation.

Table 2
Prevalence of known risk factors.

Risk factors	Young Group (≤ 45 yr) <i>n</i> =218	Not so Young Group (≥ 55 yr) <i>n</i> =235	<i>P</i> value
Smoking ¹ , <i>n</i> (%)	98 (45)	102 (43)	0.77
Smokeless tobacco users, <i>n</i> (%)	85 (39)	72 (30.6)	0.075
Diabetes ² , <i>n</i> (%)	33 (15.1)	55 (23.4)	0.032*
Family history of premature CAD ³ , <i>n</i> (%)	70 (32.1)	52 (22.6)	0.026*
Hypertension ⁴ , <i>n</i> (%)	46 (21.1)	77 (32.8)	<0.01*
Alcohol consumers, <i>n</i> (%)	78 (35.8)	53 (22.6)	<0.01*

**P* value <0.05 was considered to be significant. ¹'Smoking' was defined in our study as regular smoking of cigarettes/beedies (a local way of smoking tobacco) by the patient. ²'Diabetes mellitus' was diagnosed on the basis of fasting blood glucose levels of >126 mg/dL on two separate occasions or if a patient is already on anti-diabetic medications. ³A positive family history of premature CAD' was defined as any first degree relative of the subject that had documented CAD below the age of 55 years in males or 65 years in females. ⁴Patients were classified as 'hypertensive', if they were already on antihypertensive treatment or if their blood pressure on two separate occasions was recorded as ≥ 140 mmHg systolic and/or ≥ 90 mmHg diastolic. CAD: coronary artery disease.

(66.4 $\pm$ 10.7 kg vs. 63.7 $\pm$ 10.3 kg, *P*≤0.01) and were taller (height 164.5 $\pm$ 7.8 vs. 162.9 $\pm$ 8.5 cm, *P*=0.03), however their BMIs and abdominal girths were comparable (Table 1).

Most of the patients enrolled had acute coronary syndrome as their presenting diagnosis (87.6% in whole cohort). However, there was no significant difference between YG and NSYG for 'Diagnosis at Presentation': AMI (160 [73.4%] vs. 170 [72.3%], *P*=0.88), unstable angina (28 [12.8%] vs. 39 [16.6%], *P*=0.32), stable angina (21 [9.6%] vs. 24 [10.2%], *P*=0.96), and asymptomatic known CAD (9 [4.1%] vs. 2 [0.9%], *P*=0.5) (Table 1).

On comparison of conventional risk factors for CAD, more of younger patients had positive family history of premature CAD (70 [32.1%] vs. 52 [22.6%], *P*=0.026). However, there was no significant difference in use of tobacco in young patients, whether in form of smoking (98 [45%] vs. 102 [43%], *P*=0.77), or smokeless tobacco (85 [39%] vs. 72 [30.6%], *P*=0.075) on compared to NSYG. Presence of diabetes and hypertension was more common in NSYG as compared to YG (diabetics 55 [23.5%] vs. 33 [15.1%], *P*=0.032 and hypertensives 77 [32.8%] vs. 46 [21.1%], *P*≤0.01, respectively). Alcohol consumption was more common in younger patients (78 [35.8%] vs. 53 [22.6%], *P*≤0.01) when compared to NSYG (Table 2).

Dyslipidaemia was more common in younger patients as evident by higher TC levels (186.4 $\pm$ 60.5 mg/dL vs. 166.4 $\pm$ 44.7 mg/dL, *P*≤0.01), higher TG levels (175.4 $\pm$ 116.8 mg/dL vs. 135.5 $\pm$ 66.7 mg/dL, *P*≤0.01), higher LDL levels (112.4 $\pm$ 55.5 mg/dL vs. 96.6 $\pm$ 35.9 mg/dL, *P*≤0.01), and higher VLDL levels (33.5 $\pm$ 19.3 mg/dL vs. 26.9 $\pm$ 13.2 mg/dL, *P*≤0.01) as opposed to NSYG respectively. However, HDL-c levels were found to be similar in both groups (YG vs. NSYG: 40.9 $\pm$ 11.2 mg/dL vs. 42.9 $\pm$ 10.7 mg/dL, *P*=0.59) (Table 3).

On comparison of TC/HDL ratio and LDL/HDL ratio between two groups, younger patients had higher TC/HDL ratio (4.8 $\pm$ 1.8 vs. 4 $\pm$ 1.1, *P*≤0.01) and higher LDL/HDL ratio (2.9 $\pm$ 1.6 vs. 2.3 $\pm$ 0.9, *P*≤0.01) when compared to NSYG respectively (Table 3).

Taking ATP III guidelines¹³ as criteria for dyslipidaemia (TC $\geq$ 200, LDL $\geq$ 100, HDL $\leq$ 40—one or more among them), we found more of YG patients having dyslipidaemia (189 [86.7%] vs. 174 [74%], *P*≤0.01), compared to NSYG patients. We also classified the patients of both groups according to different National Cholesterol Education Program (NCEP) categories. Number of patients having desirable TC (<200 mg/dL), borderline high TC (200–239 mg/dL), and high TC (>240 mg/dL) were compared in both the groups. The results were statistically

Table 3

Lipids in both study groups.

Parameters	Young Group (≤ 45 yr) $n=218$, mean $\pm$ SD (SE)	Not so Young Group (≥ 55 yr) $n=235$, mean $\pm$ SD (SE)	95% CI of mean difference	P value
TC (mg/dL)	186.42 $\pm$ 60.46 (4.1)	166.36 $\pm$ 44.73 (2.9)	10.17–29.94	<0.01*
TG (mg/dL)	175.38 $\pm$ 116.85 (7.9)	135.49 $\pm$ 66.75 (4.4)	22.12–57.65	<0.01*
LDL-c (mg/dL)	112.43 $\pm$ 55.51 (3.8)	96.64 $\pm$ 35.85 (2.3)	7.03–24.56	<0.01*
HDL-c (mg/dL)	40.92 $\pm$ 11.16 (0.8)	42.86 $\pm$ 10.67 (0.7)	–3.96–0.08	0.59
VLDL-c (mg/dL)	33.46 $\pm$ 19.26 (1.3)	26.94 $\pm$ 13.22 (0.9)	3.42–9.62	<0.01*
TC/HDL-c ratio	4.78 $\pm$ 1.79 (0.12)	4.01 $\pm$ 1.14 (0.1)	0.49–1.05	<0.01*
LDL-c/HDL-c ratio	2.88 $\pm$ 1.56 (0.1)	2.34 $\pm$ 0.91 (0.1)	0.30–0.78	<0.01*

*P value <0.05 considered to be significant. CI: confidence interval, HDL-c: high-density lipoprotein cholesterol, LDL-c: low-density lipoprotein cholesterol, SD: standard deviation, SE: standard error, TC: total cholesterol, TG: triglycerides, VLDL-c: very low-density lipoprotein cholesterol.

Table 4

Classification of subjects according to different National Cholesterol Education Program categories.

Range	Young Group (≤ 45 yr) $n=218$ (%)	Not So Young Group (≥ 55 yr) $n=235$ (%)	P value
Total cholesterol (mg/dL)			
Desirable (<200)	149 (68.3)	181 (77)	<0.01*
Borderline high (200–239)	37 (17)	43 (18.3)	
High (>240)	32 (14.7)	11 (4.7)	
HDL-c (mg/dL)			
Low (<40)	109 (50)	93 (39.6)	0.07*
Normal (40–60)	96 (44)	128 (54.5)	
High (>60)	13 (6)	14 (6)	
LDL-c (mg/dL)			
Optimal (<100)	102 (46.8)	132 (56.2)	<0.01*
Above optimal (100–129)	56 (25.7)	68 (28.9)	
Borderline high (130–159)	25 (11.5)	26 (11.1)	
High (160–189)	14 (6.4)	5 (2.1)	
Very high (>190)	12 (5.5)	4 (1.7)	
Un-estimated (If TG >400)	9 (4.1)	0	
TG (mg/dL)			
Normal (<150)	116 (53.2)	158 (67.2)	<0.01*
Borderline high (150–199)	47 (21.6)	40 (17)	
High (200–499)	49 (22.5)	37 (15.7)	
Very high (>500)	6 (2.8)	0	

*P value <0.05 considered to be significant. HDL-c: high-density lipoprotein cholesterol, LDL-c: low-density lipoprotein cholesterol, TG: triglycerides.

different ($P\leq 0.01$) with younger patients having more percentage of hyperlipidaemics (on the basis of TC levels only) when compared with NSYG patients (YG: NSYG; 31.7% vs. 23%, respectively). Classification of patients of both groups on the basis of LDL and TG levels also revealed statistically significant difference in distribution among YG and NSYG ($P\leq 0.01$). Deranged LDL and TG were found in 53.2% and 46.8% in YG vs. 43.8% and 32.8% in NSYG respectively (Table 4).

However, no significant difference in distribution was observed between YG and NSYG when the patients were classified according to HDL values (Table 4).

When we investigated common determinants of dyslipidaemia in younger population inflicted with CAD using univariate logistic regression (Table 5), we found 10 fold increased risk of dyslipidaemia in young diabetics with proven CAD in young vs. 2.9 fold in NSYG patients, eight fold increased risk of dyslipidaemia in young hypertensives with CAD vs. 3.8 fold in NSYG patients, fivefold increased risk of dyslipidaemia in young males inflicted with CAD as compared to 2.3 fold in elderly,

four fold risk of dyslipidaemia was found in CAD patients who are young urban dwellers as opposed to 1.7 fold risk in the elderly, and four fold risk of dyslipidaemia was found in young CAD patients who have positive family history of premature CAD against 2.3 fold in older NSYG patients (Table 5).

We found slightly higher risks (risk difference <1) of developing dyslipidaemia in young CAD patients with factors such as alcohol consumption, smoking, non-vegetarian consumption, and smokeless tobacco consumption. Little or no association of risk of dyslipidaemia in proven CAD patients was seen when we investigated other factors like height, weight, BMI, abdominal girth, gross family income, occupation, nature of work, and physical activity.

Discussion

Our study found significantly higher levels of various lipid types in the sera of 'Young' as compared to 'Not so Young'

Table 5

Risk for dyslipidaemia in proven coronary artery disease patients.*

Factor	Group	Reg. coefficient (β)	SE (β)	Exp ^b (Risk)	95% CI (β)
Diabetes	Young	2.303	0.606	10.00	3.05–32.76
	Not so Young	1.075	0.310	2.93	1.59–5.37
Hypertension	Young	2.104	0.474	8.20	3.24–20.75
	Not so Young	1.338	0.281	3.81	2.19–6.61
Male sex	Young	1.68	0.186	5.36	3.72–7.73
	Not so Young	0.845	0.129	2.33	1.80–2.99
Urban residents	Young	1.370	0.199	3.94	2.66–5.82
	Not so Young	0.578	0.121	1.78	1.40–2.25
Family history of premature CAD	Young	1.418	0.162	4.08	2.97–5.61
	Not so Young	0.833	0.122	2.30	1.81–2.92
Alcohol consumption	Young	0.991	0.130	2.69	2.08–3.47
	Not so Young	0.661	0.112	1.94	1.55–2.41
Smoking	Young	0.817	0.097	2.26	1.87–2.73
	Not so Young	0.446	0.064	1.56	1.37–1.77
Non-vegetarian consumption	Young	0.830	0.101	2.29	1.88–2.79
	Not so Young	0.502	0.077	1.65	1.42–1.92
Smokeless tobacco consumption	Young	0.261	0.032	1.30	1.22–1.38
	Not so Young	0.134	0.020	1.14	1.09–1.19

*All factors with *P* value <0.05 have been selected. CAD: coronary artery disease, CI: confidence interval, SE: standard error.

patients of CAD. This study is the first Indian, prospective, multicentric, observational study which has established a marked difference in various lipid parameters in 'Young' (≤ 45 years) vs. 'Not so young' (≥ 55 years) patients of proven CAD presenting at several different centres in northern India, employing a central, standardised method for biochemical estimations. To get a fair idea of the baseline values of lipids in our population inflicted with CAD, we only enrolled patients who were naive for any lipid lowering therapy in at least last 4 weeks. Most (72.8%) patients were enrolled with the presentation diagnosis of AMI. These AMI cases were enrolled within 24 hours of the index event to offset probability of any significant change in lipid values due to the event.

We found mean fasting glucose levels, comparable (117.49 ± 59.27 mg/dL [standard error, SE=4.02] vs. 115.03 ± 47.69 mg/dL [SE=3.11], $P=0.63$) in both the groups, and they still had marked difference in almost all lipid parameters in the two groups, negating a possible role of deranged glucose levels affecting the lipid levels, especially TGs.

Several cross-sectional and longitudinal studies have demonstrated that serum cholesterol levels among adults generally increase with age.^{14,15} On the contrary, we, in the present study, have found most lipid parameters to be significantly raised in 'Young' CAD patients when compared to the 'Not so Young' ones.

Most of the published literature from India regarding lipids in CAD patients are either derivatives of cross-sectional surveys^{4,16–18} or excerpts from retrospective hospital data.^{19–21} Since, the design of the study was unique, and first of its kind, therefore, these results cannot be compared with any of the previously published Indian studies. To the best of our knowledge, no other study from the subcontinent has systematically examined lipids in 'Young' and 'Not so Young' CAD cases in north Indian population employing a prospective design. Recent Indian studies have mainly been cross-sectional surveys showing the prevalence of risk factors for CAD among

Indians and none of those had been specifically designed like the present study to address this specific issue.

Lipids in the whole cohort in our study were found to be higher than those in the study by Tewari et al.¹⁹ which although enrolled a similar population but was a single centre, non-standardised study. Also, unlike the study by Tewari et al.¹⁹ we enrolled patients who were practically naive to any kind of lipid lowering therapy, therefore, the reported lipid levels in our study may well represent the actual baseline levels for the diseased population in our part of the world.

On comparing our study to the recently published Indian studies, which enrolled similar proven CAD population, we found significant difference in the prevalence of conventional risk factors. When we examined the whole cohort in our study, we found 19.4% of diabetics in our dataset as opposed to 13.28%,²¹ 30.5%,¹⁹ and 40.4%²⁰; 27.2% hypertensives as opposed to 18.02%,²¹ 45.7%,²⁰ and 50.6%¹⁹; 44.2% of smokers as opposed to 16.5%,²⁰ 30.9%,²¹ and 35.0%¹⁹ while 27.2% of patients had positive family history of premature CAD as opposed to 10.4%¹⁹ and 14.5%²⁰ in various recent studies conducted among north Indian population.

The low prevalence of diabetics and hypertensives in our study may be explained by the fact that a majority of the patients included in the study (~73%) were admitted to the hospital with the diagnosis of AMI and they did not have a documented history of either diabetes or hypertension. As they were included in the study (and the sample drawn) soon after they were admitted to the hospital (so as to meet the <24 hour post AMI window), there was not ample amount of time to ascertain diabetes or hypertension status of the patient. On the contrary, in a study by Kumar et al.,²⁰ almost 93% of post MI patients were enrolled but within 4 weeks of MI and subsequent angiography, which gave them ample time for diagnosing a fresh case of diabetes or hypertension in the patients, similar was the case in a study by Tewari et al.¹⁹ As far as the higher frequency of smokers and patients with positive

family history of premature CAD in our study are concerned, it could well be a consequence of 1:1 ratio inclusion in YG and NSYG in our study. Also, as these factors do not need much documentation and the patient's word is usually enough to record the same, we confer that our figures may very well represent the real scenario.

Alcohol and smokeless tobacco consumption in patients with proven CAD was studied for the first time in our study. However, its distribution may not be significantly different in both the study groups implying no or little role in premature manifestation of CAD. A limitation of this study is that from the available data, we cannot infer the role of alcohol or smokeless (chewing) tobacco in CAD, as for proving/disproving their suspected influence we would need similar prospective data for equal number of age- and sex-matched controls residing in the same geographical area.

Published data so far suggests that the first MI occurs in 4.4% of Asian women and 9.7% of men at age <40 years, which is 2–3.5 fold higher than in the west European population, and is the third highest of all regions studied worldwide.¹¹ These studies suggest that Asians in general and Indians in particular are at increased risk of MI at a younger age (<40 years), in both resident and migratory populations. Also, the mean serum cholesterol levels among Asian Indians have been shown to be similar to that of the general population in the US and lower than the levels in the UK.^{22,23} Therefore, it is evident that CAD manifests in Indians at low TC and TG levels.²⁴ The lower level of TC in Asian Indians compared with Caucasians may be misleading, if we fail to recognise that the mean levels of TC and TGs in Asian Indians with CAD are 20–40 mg/dL lower than in Caucasians with CAD.^{23,25,26} This implies that cut-off lipid values for Indians inflicted with CAD should be set by studies like ours but with a larger sample size. This will help in establishing new guidelines for lipid management, tailor made for the population from Indian subcontinent.

We found that among population inflicted with CAD, diabetics had the highest risk of developing dyslipidaemia followed by hypertension, male gender, urban lifestyle, and family history of premature CAD. A little relative risk difference was observed in young patients when compared to elderly, for factors such as alcohol consumption, smoking, non-vegetarian consumption, and smokeless tobacco consumption using univariate logistic regression. Ours is the first study which reported determinants of dyslipidaemia in CAD patients with requisite statistical proof. These results need urgent attention of healthcare professionals to look for all the risk factors in patients with CAD while selecting their lipid management strategy.

Conclusion

The results of the present study indicate that lipids levels in Indian population are generally higher in younger CAD patients.

We have successfully established in our present study that lipids levels in Indians are generally higher especially among

younger patients with CAD. The normal lipid values in Indian population also seem to be different from western standards which necessitates for setting up our own standards for lipid values, an endeavour similar to the NCEP in USA for both diseased and healthy population. This would pave our way in formulating specific guidelines for lipid management in Indians.

We also found that risk factors of CAD like diabetes and hypertension are also important in patients with younger onset CAD and that they have heightened chance of developing dyslipidaemia. This calls for aggressive management of lipids in these patients with one or more risk factors using lifestyle modification and pharmacologic intervention.

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