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Original Research Article

Carotid Intima–Media Thickness as an Independent Predictor of Coronary Artery Disease

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Abstract

Introduction: Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide. Early identification of individuals at increased risk is essential for timely intervention. Carotid intima–media thickness (CIMT), measured by high-resolution B-mode ultrasonography, reflects early atherosclerotic changes and has emerged as a reliable, non-invasive marker of systemic atherosclerosis and cardiovascular risk.

Aims: To evaluate carotid intima–media thickness as an independent predictor of coronary artery disease and to assess its association with cardiovascular risk factors.

Materials and Methods: This hospital-based observational case–control study was conducted in the Department of Radiology, BKL Walawalkar rural medical college, Chiplun, Ratnagiri. The study included 52 angiographically confirmed CAD cases and 26 age- and gender-matched controls without CAD. All subjects underwent clinical evaluation, biochemical investigations, and carotid ultrasonography for measurement of CIMT, carotid plaque, and degree of carotid stenosis. Statistical analysis was performed to determine the association between CIMT and CAD.

Results: Mean CIMT was significantly higher in CAD cases compared to controls. Carotid plaque was present in 65.38% of cases versus 23% of controls ($p < 0.05$). CAD cases showed significantly higher total cholesterol, LDL-C, triglycerides, post-prandial blood sugar, and body mass index, with lower HDL-C levels. Moderate to severe carotid artery stenosis was observed exclusively among cases, supporting the role of increased CIMT as an independent predictor of CAD.

Keywords: Carotid intima–media thickness; Coronary artery disease; Atherosclerosis; Carotid plaque; Carotid Doppler and Ultrasonography.

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Introduction

Nature has for centuries been conducting gigantic experiments as to the effects of climate, of type of work, of diet and of social or worldwide diseases on men women and children of different races that are spread out before our vary eyes for us to record and analyze, quite readily yielding information that might never be obtainable by our own experiments on man P.D. White[1].

Obstruction of coronary artery or any of its large branches has been regarded as a serious accident. Several events contributed towards the prevalence of the view that this condition was almost always suddenly fatal. But there are reasons to believe that even large branches of the coronary arteries may be occluded-at times acutely occluded-without resulting death, at least without death in immediate future. [2] Coronary artery disease is characterized by presence of atherosclerosis in the epicardial coronary arteries and is the most common form of heart disease. Presence of atherosclerotic plaque is the hallmark of the disease which causes progressive narrowing of coronary artery lumen. Coronary Artery Disease (CAD) is the leading cause of death worldwide.

The prevalence of cardiovascular disease goes on increasing from 5% at age of 20 to 75% at 75yrs of age[3]. It is said that by the year 2020 cardiovascular disease will be the major cause of death worldwide [4]. Previously considered a disease of the affluent, the past three decades have witnessed a significant decline in incidence and prevalence of atherosclerotic coronary artery disease in the industrialized western world, whereas at the same time it is assuming a epidemic proportion in the developing world [5].The Asian Indians have much higher incidence of CAD as

compared to all ethnic groups. CAD among Asian Indians has been found to be more severe, diffuse & associated with serious complications & increasing mortality in young age. An underlying genetic susceptibility associated with modest abnormality in lipid & lifestyle factors makes C.A.D. assume a malignant course in Asian Indians[5]. Atherosclerosis lies at the root of C.A.D. and because atherosclerosis is considered a generalized disease, manifested in the entire vasculature, an association between coronary and peripheral vascular disease has been well established. The important relationship between carotid artery disease and coronary artery disease (CAD) is best expressed by the high incidence of myocardial infarction following carotid endarterectomy and the devastating effects of neurological injury occurring occasionally after routine coronary artery by pass[6].

1. Lipid disorders [7]: Abnormality in plasma lipoproteins and derangements in lipid metabolism rank as the most firmly established and best understood risk factor for atherosclerosis. Patient with familial hyperlipidaemia have a high incidence of premature CAD and many epidemiological studies have demonstrated positive correlation between mean population, plasma cholesterol concentration and morbidity and death from coronary disease.
2. Hypertension [7]: Incidence of CAD increases as blood pressure rises and the excess risk is related to both systolic and diastolic blood pressure.
3. Smoking [8]: Probably the most important avoidable cause of CAD. There is strong, consistent and dose linked relationship between cigarette smoking and CAD. Incidence of CAD is 3 to 5 times higher in smoker who smoke 20 cigarettes per day compared to non – smokers.
4. Diabetes [7]: DM is “CHD risk equivalent”. Most patients of diabetes die of atherosclerosis and its complications.
5. Alcohol [9]: Heavy drinking defined as 75 gms or more per day is an independent risk factor for coronary heart disease, hypertension and all cardiovascular events.
6. Obesity and increased body mass index [8]: Obesity, particularly if central or truncal is an independent risk factor. Same is true for BMI more than 25kg/m^2.
7. Physical activity [8]: Regular exercise (Brisk walking, cycling or swimming for 20 minutes 2 or 3 times a week) appears to have protective effect which may be related to its ability to increase HDL, lower BP, reduced blood clotting and promote collateral vessel development.

Apart from these conventional cardiovascular risk factors, the newer emerging risk factors for CAD are hyperhomocystinemia, hyperfibrinogenemia, small dense LDL phenotype, elevated lipoprotein (a) and inflammatory and infectious agents.

In ethnic Asian Indians the insulin resistance syndrome, metabolic syndrome, lipoprotein(a), atherogenicdyslipidemic phenotype & some newer emerging risk factors (homocysteine, tPA, PAI-1, fibrinogen, factor VII, infection & inflammations) may be more relevant [10].

Materials and Methods

This hospital based, observational case control study was carried out in the Department of Radiology, BKL Walawalkar rural medical college, Chiplun, Ratnagiriduring the period of August 2023 to August 2025.

Total 52 consecutive cases of angiographically confirmed coronary artery disease and 26 age and gender matched controls without coronary artery diseases were enrolled in the present study. All the subjects were examined and investigated according to proforma that was predesigned and pretested.

This study was approved by the ethical committee of Department of Radiology, BKL Walawalkar rural medical college, Chiplun, Ratnagiri. Informed consent was obtained from all subjects enrolled in the study.

Methodology

Study Design: Cross-Sectional Observational Analytical Case Control Study.

Study Setting: Department of Radiology, BKL Walawalkar rural medical college, Chiplun, Ratnagiri.

Sample Size: A total of 52 consecutive angiographically confirmed coronary artery disease cases and 26 age- and gender-matched controls without coronary artery disease were enrolled in the study based on a predetermined sample size.

Study Variables

Inclusion Criteria for Cases: Adults above 35 yrs of age having angiographically demonstrated significant coronary artery disease as mentioned above are included in study.

Exclusion Criteria for Cases: Patients with acute myocardial infarction, acute cerebrovascular episode, hepatic, renal failure, cases with neck pathologies likely to produce changes in carotid Doppler study were excluded.

Statistical Analysis: Data were entered and analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies

and percentages. The prevalence of refractive errors was calculated, and associations with screen time and other risk factors were assessed using chi-square tests for categorical variables and independent t-tests or ANOVA for continuous variables. Correlation between screen time and severity of refractive errors was evaluated using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

Result

Table 1(a): Age and sex distribution in cases

Age (yrs)	Male (%)	Female (%)	Total (%)
35-45	5(55.5)	4(44.4)	9(17.3)
46-55	10(58.8)	7(41.1)	17(32.6)
56-65	10(62.5)	6(37.5)	16(30.7)
66 & above	6(60)	4(40)	10(19.2)
Total	31	21	52

Table 1(b): Age and sex distribution in controls

Age (yrs)	Male (%)	Female (%)	Total (%)
35-45	1(33.3%)	2(66.6%)	3(11.5%)
46-55	5(62.5%)	3(37.5%)	8(30.7%)
56-65	6(66.6%)	3(33.3%)	9(34.6%)
66 & above	4(66.6%)	2(33.3%)	6(23.06%)
Total	16	10	26

Table 2: Risk Factors in Cases and Controls

Parameters	Cases (N=52)	Controls (N=26)	P Value
Male Gender	31(59.6%)	16(61.5%)	0.39, NS
Dyslipidemia	24(48%)	7(6.9%)	0.22, NS
Hypertension	20(38.4%)	7(6.9%)	2.17, NS
Diabetes	17(32.6%)	8(30.7%)	4.5, S
BMI>25kg/M^2	10(19.2%)	2(7.6%)	42.9, S
Smoking	10(19.2%)	3(11.5%)	36.9, S
Physical Inactivity	47(90.3%)	22(84.6%)	2.62, NS
Alcohol	4(7.6%)	0	-

Table 3: Distribution of Serum Lipids, Blood Sugar, Blood Pressure and BMI in cases and controls

Parameters	Cases (N=52)	Controls (N=26)	P Value
Total Cholesterol	190±48.1mg%	170±32.6mg%	0.04
Low Density Lipoprotein-C	118.2±48.1mg%	96.3±31.9mg%	0.02
High Density Lipoprotein –C	42.9±6mg%	47.7±5.4mg%	0.0007
Triglyceride	145.5±39.2mg%	128.3±25.8mg%	0.024
Fasting Blood Sugar	93.4±25.3mg%	91.1±16.3mg%	0.6
Post Prandial Blood Sugar	140.6±38mg%	116.8±25.6mg%	0.0017
Systolic Blood Pressure	130.3±9.5mg%	128.1±9.7mg%	0.25
Diastolic Blood Pressure	81.2±8.2mg%	78.6±6.9mg%	0.06
Body Mass Index	23.7±2.2kg/m^2	22±2.2kg/m^2	0.001

Table 4: Number of risk factors and angiographic pattern of coronary artery involvement

No. of Risk Factors	S.V.D. N=16	D.V.D. N=16	T.V.D. N=20	Total
NO R.F.	0	1	0	1(1.9%)
ONE R.F.	2	0	2	4(7.6%)
TWO R.F.	3	2	2	7(13.4%)
>TWO R.F.	11	13	16	40(76.9%)
16	16	16	20	52(100%)

Table 5: Pattern of Coronary Involvement and Carotid Plaque

Coronary Involvement	No. Cases with Carotid Plaque	Percentage
Single Vessel Disease (n=16)	8	50%
Double Vessel Disease (n=16)	11	68%
Triple Vessel Disease (n=20)	15	75%
52	34	

Table 6(a): Carotid Doppler abnormality and number of risk factors

Intima Media Thickness			
Number of Risk Factors	**Cases (N=52)**	**Controls (N=26)**	**P Value**
No Risk Factor (n=1)	-	0.6mm	-
One Risk Factor (n=6).	1±0.1mm	-	-
Two Risk Factor (n=14)	1.06±0.2mm	0.75±0.1mm	0.009
> Two Risk Factor (n=57)	1.1±0.2mm	0.77±0.2mm	0.000….p<0.05,S

Table 6(b): Carotid Doppler abnormality and number of risk factors

Carotid Plaque			
Number of R.F.	**Cases (N=52)**	**Controls (N=26)**	**P Value**
NO R.F.	-	-	-
ONE R.F.	15±70.1%	-	-
TWO R.F.	25±11.4%	3.50%	-
>TWO R.F.	45.6 ± 16.7%	14%	(p=0.000…. p<0.05, s)

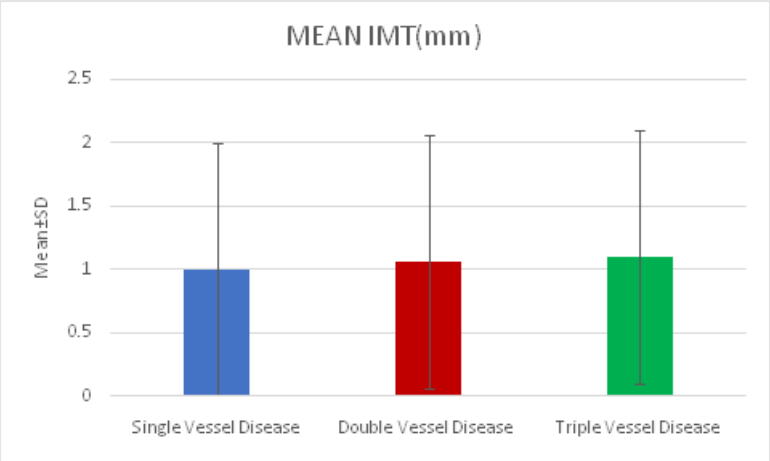

Figure 1: Relation of Carotid IMT and Pattern of Coronary Artery Involvement

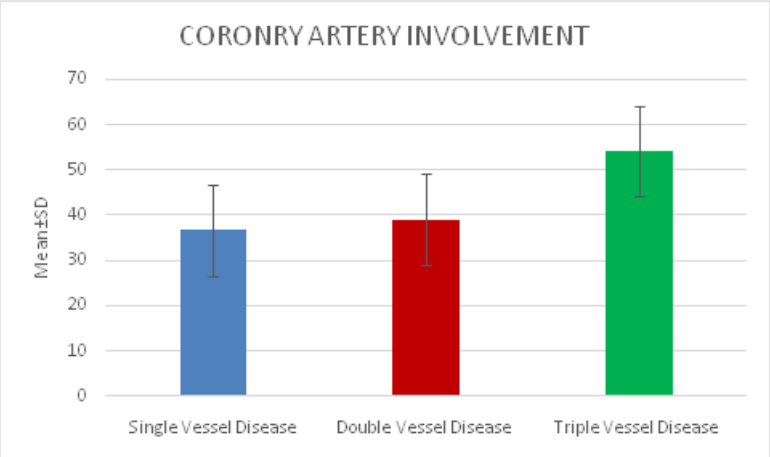

Figure 2: Relation of Carotid Artery Luminal Stenosis and Pattern of Coronary Artery Involvement

The majority of patients were in the 46–55 years age group, accounting for 17 cases (32.6%), followed by the 56–65 years group with 16 cases (30.7%). Patients aged 66 years and above

constituted 10 cases (19.2%), while the 35–45 years age group had the least number of cases (9 cases, 17.3%). Across all age groups, males predominated with 31 cases, compared to 21 female cases. Male preponderance was observed in every age group, with the highest proportion of males in the 56–65 years age group (62.5%), followed by the 46–55 years age group (58.8%). Females constituted 44.4% of cases in the 35–45 years group and 40% in those aged 66 years and above.

Among the 26 control subjects, the highest proportion belonged to the 56–65 years age group, comprising 9 cases (34.6%), followed by the 46–55 years group with 8 cases (30.7%). Individuals aged 66 years and above accounted for 6 cases (23.06%), while the 35–45 years age group constituted the smallest proportion with 3 cases (11.5%).

Overall, males predominated in the control group with 16 subjects, compared to 10 females. Male predominance was observed in all age groups except 35–45 years, where females were more common (66.6%). In the remaining age groups, males constituted 62.5% in the 46–55 years, 66.6% in the 56–65 years, and 66.6% in the 66 years and above age groups.

Male gender distribution was comparable between the two groups, with 31 cases (59.6%) and 16 controls (61.5%), showing no statistically significant difference ($p = 0.39$, NS). Dyslipidaemia was more frequently observed among cases (24; 48%) compared to controls (7; 6.9%); however, this difference was not statistically significant ($p = 0.22$, NS). Similarly, hypertension was present in 20 cases (38.4%) and 7 controls (6.9%), without a significant association ($p = 2.17$, NS).

Diabetes mellitus was noted in 17 cases (32.6%) and 8 controls (30.7%), and this difference was found to be statistically significant ($p = 4.5$, S). A BMI >25 kg/m² was significantly more common in cases (10; 19.2%) than controls (2; 7.6%), showing a statistically significant association ($p = 42.9$, S). Smoking was also higher among cases (10; 19.2%) compared to controls (3; 11.5%), and this difference was statistically significant ($p = 36.9$, S).

Physical inactivity was highly prevalent in both groups, observed in 47 cases (90.3%) and 22 controls (84.6%), with no statistically significant difference ($p = 2.62$, NS). Alcohol consumption was reported only among cases (4; 7.6%) and was absent in the control group; hence, statistical comparison was not applicable. The mean total cholesterol level was significantly higher in cases (190 ± 48.1 mg%) compared to controls (170 ± 32.6 mg%), and this difference was statistically significant ($p = 0.04$). Similarly, low-density lipoprotein cholesterol (LDL-C) was higher among cases (118.2 ± 48.1 mg%) than controls (96.3 ± 31.9 mg%), showing a statistically significant difference ($p = 0.02$). In contrast, high-density lipoprotein cholesterol (HDL-C) was significantly lower in cases (42.9 ± 6 mg%) compared to controls (47.7 ± 5.4 mg%), and this difference was highly significant ($p = 0.0007$). The mean triglyceride level was also higher in cases (145.5 ± 39.2 mg%) than controls (128.3 ± 25.8 mg%), with a statistically significant difference ($p = 0.024$). There was no statistically significant difference in fasting blood sugar between cases (93.4 ± 25.3 mg%) and controls (91.1 ± 16.3 mg%) ($p = 0.6$). However, post-prandial blood sugar levels were significantly higher in cases (140.6 ± 38 mg%) compared to controls (116.8 ± 25.6 mg%), indicating a statistically significant difference ($p = 0.0017$).

The mean systolic blood pressure was slightly higher in cases (130.3 ± 9.5 mmHg) than controls (128.1 ± 9.7 mmHg), but this difference was not statistically significant ($p = 0.25$). Similarly, diastolic blood pressure was higher in cases (81.2 ± 8.2 mmHg) compared to controls (78.6 ± 6.9 mmHg), though the difference did not reach statistical significance ($p = 0.06$).

The mean body mass index (BMI) was significantly higher among cases (23.7 ± 2.2 kg/m²) than controls (22 ± 2.2 kg/m²), and this difference was statistically significant ($p = 0.001$).

The distribution of risk factors among patients with different patterns of vessel disease is shown in the table. Only one patient (1.9%) had no risk factors, and this was observed in the double vessel disease (DVD) group. Patients with one risk factor constituted 4 cases (7.6%), equally distributed between single vessel disease (SVD) and triple vessel disease (TVD).

Those with two risk factors accounted for 7 cases (13.4%), with 3 cases in SVD, 2 cases in DVD, and 2 cases in TVD. A large majority of patients had more than two risk factors, comprising 40 cases (76.9%). This group was predominantly represented in triple vessel disease (16 cases) and double vessel disease (13 cases), followed by single vessel disease (11 cases).

The mean carotid intima–media thickness (IMT) increased progressively with the extent of coronary artery involvement. Patients with single vessel disease (SVD) had a mean IMT of 1.0 ± 0.1 mm. Those with double vessel disease (DVD) showed a higher mean IMT of 1.06 ± 0.2 mm, while the highest mean IMT was observed in patients with triple vessel disease (TVD) at 1.1 ± 0.2 mm. However, the differences in mean IMT among patients with single, double, and triple vessel disease were not statistically significant, as reflected by the p-values ($p = 0.39$, $p = 0.55$, and p

= 0.12, respectively). Despite the lack of statistical significance, a trend toward increasing IMT with greater coronary artery involvement was observed. The mean percentage of coronary artery stenosis increased with the severity of vessel involvement. Patients with single vessel disease (SVD) had a mean percentage stenosis of 36.7 ± 16.6%, and this was not statistically significant (p = 0.74). In patients with double vessel disease (DVD), the mean percentage stenosis was 39.1 ± 12.8%, showing a statistically significant association (p = 0.007).

The highest mean percentage stenosis was observed in patients with triple vessel disease (TVD), with a value of 54.3 ± 13.6%, which was also statistically significant (p = 0.02). Overall, the findings demonstrate that mean coronary artery stenosis increases significantly with the number of vessels involved, particularly in patients with double and triple vessel disease.

Carotid plaque was detected in 34 out of 52 cases. Among patients with single vessel disease (SVD), 8 of 16 cases (50%) showed evidence of carotid plaque. In the double vessel disease (DVD) group, carotid plaque was present in 11 of 16 cases (68%).

The highest prevalence of carotid plaque was observed in patients with triple vessel disease (TVD), where 15 of 20 cases (75%) demonstrated plaque formation. Overall, the presence of carotid plaque increased with the severity of coronary artery involvement, indicating a higher burden of subclinical atherosclerosis in patients with more extensive coronary disease.

Carotid intima–media thickness (IMT) showed a progressive increase with the rising number of cardiovascular risk factors. Among subjects with no risk factors, IMT was observed only in controls with a mean value of 0.6 mm, while no comparable case was available for analysis. In individuals with one risk factor, cases demonstrated a mean IMT of 1.0 ± 0.1 mm, whereas no control subject was present in this category, precluding statistical comparison.

In participants with two risk factors, mean IMT was significantly higher in cases (1.06 ± 0.2 mm) compared to controls (0.75 ± 0.1 mm), and this difference was statistically significant (p = 0.009). Similarly, among those with more than two risk factors, cases exhibited a markedly higher mean IMT (1.1 ± 0.2 mm) than controls (0.77 ± 0.2 mm), with a highly significant difference (p < 0.05).

The prevalence of carotid plaque increased with the number of cardiovascular risk factors. No carotid plaque was observed among subjects with no risk factors in either cases or controls. In individuals with one risk factor, carotid plaque was observed only among cases, with a prevalence of 70.1%, while no control subject showed plaque in this category, precluding statistical comparison.

Among participants with two risk factors, carotid plaque was more common in cases (25 ± 11.4%) compared to controls (3.5%); however, statistical comparison was not applicable due to the small number of controls. In subjects with more than two risk factors, cases demonstrated a markedly higher prevalence of carotid plaque (45.6 ± 16.7%) than controls (14%), and this difference was statistically highly significant (p < 0.05).

Discussion

Age and sex distribution: In present study out of 52 cases 9 (17.3%) were in age group of 35- 45yrs. & 10(19.2%) were more than 66yrs. Maximum no. of cases were in age group between 46-65 yrs. Similarly in controls out of 26, 3(11.3%) were between 35-45years, 6(23.07%) were above 66years. Here too max 17(>60%) were in age group between 45-65.

The mean age of cases was 55±9.5yrs &those of controls was 56.6±9.6yrs. Male to female ratio was 1.4:1.in cases and 1.6:1 in controls. In a similar study by Kezhu Sun et al(2000)[07] 78 subjects with C.A.D. having a mean age of 62.3±8.5yrs with a male to female ratio of 1.6:1 and 69 subjects without C.A.D. having a mean age of 60±10 yrs were included.

Among the 21 female cases 9(42.8%) and among controls 5(50%) out of 10 were postmenopausal.

The age and sex distribution in present work was similar to these studies and helped to compare the data.

B.M.I.: In present study 42(80.7%) were having normal BMI i.e. <25 kg/m2, 10(19.23%) cases were with BMI above 25 kg/m2 in control group 2(7.6%) were having BMI above 25kg/m2.

The mean BMI in cases was 23.7±2.4 kg/m2 and in controls 22.1±1.9 kg/m2.(p=0.001).

Similar study by Kezhu Sun et al (2000) [7] observed a BMI of 23.6±3.1 kg/m2 in cases with CAD and 23.6±3.7 kg/m2 in cases without CAD.

UdayJadhav et al (2001)[14] had BMI of 25.5±3.37 kg/m2 in subjects with CAD and 26.05±3.36 kg/m2 in subjects without CAD.Thus the mean BMI distribution in our study was similar to other studies with a statistically significant difference between cases and controls (p-0.001).

Blood sugar fasting and postprandial: In present work the mean FBS was 93±25.3mg% and mean PPBS was 141.4±38mg% (p=0.6,0.001 resp.).

In the control group these values were 90.9±16.3mg% and 116.5±25.6mg% respectively.

In a similar study by Jadhav et al (2001) [14] mean FBS in cases was 124.3±56.7mg% and 114.5±48.7mg% in controls.

The fasting blood sugar levels were lower in present study compared to above study. The reason being less number of diabetic cases in present study compared to above study (32.6% Vs 51.1%). Moreover 13(76.2%) cases were on antidiabetic treatment either orally or by insulin regimen.

Lipid profile: In present study 24(48%) cases were having dyslipidemia.10 (54.4%) were on oral lipid lowering drugs.

Subjects were labeled dyslipidemic according to NCEP guidelines.

The mean total cholesterol level in cases was 190.09±48.5mg% and 170.6±32.6mg% in controls (p=0.04).

Out of 24, 17(70.8%) were having mixed dyslipidemia (increased LDL-C, TG, decreased HDL-C).5(20.8%) had isolated HDL-C dyslipidemia and 2(8.33%) had isolated increased TG levels.

The mean levels of LDL-C, HDL-C and TG were 118.2±48.7mg%,42.9±6.04mg%, 145.5±39.29mg% in cases and 96.38±31.9mg%, 47.7±3.1mg%, 128.3±25.8mg% in controls resp(p=0.02,0.0007,0.02 resp).

In a similar study by UM Jadhav et al (2004)[15] showed a mean total cholesterol 214.4 ± 41.3mg% mean LDL-C129.1±37.6mg%, mean HDL- C40.1 ± 6.8mg% in patients of CAD and 206 ± 48.9mg%, 122.1 ± 43.6mg%,42.7 ± 7.3mg% respectively in patients without CAD.

Similarly Jadhav et al (2001) [14] observed levels of TC, LDL-C, HDL- C, TG as 208.4 ± 43.4mg%,126.9 ± 39.2mg%,41.7 ± 7.7mg% in cases with CAD and 200.5 ± 40.8mg%,122.5 ± 4.4mg%, 42.9 ± 7.4mg% resp in controls without CAD.

Though biochemically demonstrable dyslipidemia in the present study is less, significant number of cases 24 (48%) had dyslipidemia. This may be explained by the fact that, some of our cases (54.15%) were on lipid lowering drugs. And the measurement of other highly atherogenic lipoprotein like Lp(a), apolipoprotein A, apolipoprotein B could not be done in the present study.

Hypertension: In present study out of 52 cases 20(38.4%) were hypertensive of these 9(45%) were having DM in addition.

In controls there were 7(26.9%) hypertensive.

The differences in our study was not statistically significant (X2 -2.17, NS).

In a similar study by Kezhu Sun et al (2000) [7] percentage of hypertensive in cases was 58 and that of in controls was 45. The difference was statistically insignificant.

Jadhavet al (2001) [14] observed 18.2% hypertensive in cases compared to 33.5% in controls.

Correlation of Hypertension with Carotid Doppler Abnormality: In present study the mean carotid artery luminal stenosis in cases with hypertension was 43.14% with a statistically significant difference when compared to controls without CAD (p=0.000; p<0.05).

The mean CIMT in cases with HT was 1.1±0.2mm and in hypertensive controls was 0.75±0.1mm. Difference was statistically significant (p=0.03).

In present study cases with more than two risk factors had a mean carotid artery luminal stenosis of 45.63±16.7%. Among the cases there was a statistically significant increase in mean luminal stenosis in subjects with more than two risk factors compared to those with two risk factors (p-0.007). The mean carotid IMT was 1.1±0.22mm in cases. There was a statistically significant difference between the CIMT in cases and controls with two risk factors and more than two risk factors (p-0.009, p=0.000……p<0.05, S resp.). However, it was insignificant when compared within the cases (p-0.85.0.97, 0.85). Rath PC et al (2001)[18] also found increased prevalence of carotid stenosis in patients with more than or equal to two risk factors (83.3%Vs68.8%).

A study by Held et al (2001)[17] concluded that although carotid IMT is most commonly used as surrogate marker for atherosclerosis, assessment of plaque in carotid artery is a better predictor of coronary events. Rath PC et al (2001)[18] has also mentioned that patients with significant carotid stenosis had severe coronary disease. Rachael E Mays (2006))[21] found that carotid plaque predict incident cardiovascular death and myocardial infarction.

Conclusion

Carotid intima–media thickness (CIMT) is a reliable, non-invasive marker of subclinical atherosclerosis and serves as an independent predictor of coronary artery disease. Increased CIMT shows a significant association with the presence and severity of coronary artery involvement and correlates with established cardiovascular risk factors such as diabetes, dyslipidemia, smoking, and increased body mass index.

Progressive thickening of the carotid intima–media reflects systemic atherosclerotic burden and parallels the extent of coronary vessel disease.

Routine assessment of CIMT can aid in early risk stratification, timely intervention, and improved preventive strategies, thereby reducing the morbidity and mortality associated with coronary artery disease.

It may be postulated that increased Carotid IMT indicate presence of atherosclerotic CAD and degree of carotid stenosis may reflect severity of the CAD disease.

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