Journal of Cardiovascular Disease Research
ISSN: 0975-3583, 0976-2833 VOL13, ISSUE 08, 2022

EVALUATING RISK FACTORS OF PREMATURE CORONARY ARTERY DISEASES IN SOUTH INDIAN PATIENTS

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Type of study: Original Research Paper

Conflicts of interest: nil

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ABSTRACT

Background: Heart attacks and cardiac deaths have become so common nowadays that when an elderly individual succumbs, it gets little attention as it seems natural and an inevitable. As in all populations, conventional risk factors are significantly associated with the risk of coronary artery disease in Indians.

Objectives: this study was conducted to evaluate conventional risk factors with premature coronary artery disease by analyzing the levels of HbA1c, lipoproteins and triglycerides in premature coronary artery disease.

Methods: We studied 90 patients of either gender who underwent angiography. study population was analyzed in three stages namely; their demographical representation in accordance with risk factors, presence and type of angiographic findings, case control type analysis to prove independent significance of risk factors with relation to CAD and analysis of risk factors as per the angiographic finding in terms of severity. Weight, BMI, HbA1c, systolic blood pressure, diastolic blood pressure, pulse, LDL, VLDL, HDL, total cholesterol, and triglyceride were calculated.

Result: Mean age in cases was 50±7.3 years and of controls was 51±6.3 years with Male preponderance [3.5:1]. Most frequent conventional risk factors present in total study sample were hypertension (53%), diabetes (50%), dyslipidemia(41%), family history (32%), alcohol

use(43%), tobacco use(38%); while in cases of CAD they were hypertension (73%), diabetes (75%), dyslipidemia(62%), family history(49%), alcohol use(60%), tobacco use(53%). Statistically significant risk factors were BMI, HbA1c, triglycerides, LDL, and total cholesterol. dyslipidemia was correlated 20% to CAD progression as well as severity.

Conclusion: Our study also proves multi factorial causation of CAD with lesser number of risk factors present in controls and higher number of risk factors in cases. 8 risk factors were present in 2% whereas 31 % cases showed presence of 6.

Keywords- Angiographic Severity of Disease, Conventional Risk Factors, Premature coronary artery disease [CAD]

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INTRODUCTION

Heart attacks and cardiac deaths have become so common nowadays that when an elderly individual succumbs, it gets little attention as it seems natural and an inevitable. But when young individuals in prime of their health are afflicted, it may lead to grave consequences. Like elderly, atherosclerosis is the leading cause of coronary artery disease (CAD) in the young, and accounts for 80% of the heart attacks; 60% mainly due to one coronary artery disease. In young adults, inborn abnormalities of the coronary artery anatomy cause only 4% of the heart attacks.[1]

Especially in India there is an epidemic of coronary heart disease. The Registrar General of India reported that CAD led to 17% of total deaths and 26% of adult deaths in 2001-2003, which increased by 6% of total and adult deaths in 2010-2013 compared to previous years. WHO India, after reviewing studies has reported increasing CAD prevalence over the last 60 years, from 1% to 10% in urban population and from less than 1% to 6% in rural population.[2]

The overall prevalence rate of CAD is 11.0% (age standardized, 9.0%). Major risk factors for high rate of CAD are impaired glucose tolerance and diabetes. Prevalence of CAD increases with an increase in total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides (TG) and total cholesterol/high-density lipoprotein (HDL) ratio.[3]

It is a well-established fact that atherosclerosis starts early in the childhood and progresses silently for decades. Only 1/3rd of the individuals with heart disease develops advanced warning sign in the form of angina. Otherwise, most of the time it manifests as a heart attack or sudden death without any warning signs.[4]

Framingham Heart Study going on continuously since last 50 years has proved the concept of cardiovascular risk factors.[5] These advances in the medical research have refined our understanding of these risk factors which are now classified as traditional and non-traditional risk factors. As the number or the severity of risk factors increases, it gets more likely that one will develop cardiovascular disease.[6]

Smoking, alcohol consumption, hypertension, hyperlipidemia, diabetes, obesity, mental stress, sedentary lifestyle, and family history are the conventional risk factors. Healthy diet, regular physical activity and avoidance of tobacco use can avoid at least 80% of CAD. Today, the hallmark of preventive practice is an early identification and reduction of risk in the high-risk adult population.[7]

Increased appreciation of the effects of long-term exposure to risk factors and concern about the epidemic of CAD in the young has prompted a new sense of urgency for primordial and primary prevention. Now there is increased investigation into novel patho-physiological factors contributing to CVD. These factors of CVD are potential markers to enhance clinical risk strategy by helping in the prediction, identification, and assessment of atherosclerotic disease.[12]

As in all populations, conventional risk factors are significantly associated with the risk of CAD in Asian Indians.[13]When compared with Caucasians, Asian Indians have lower prevalence of hypercholesterolemia, hypertension, and smoking, but a higher prevalence of low HDL, high TG, central obesity, and glucose intolerance.[8]

Finally glycated hemoglobin (HbA1c), lipoproteins and triglycerides will be studied in view of various risk factors present concurrently with severity of disease.[9] Because when it comes to more severe disease diabetes and dyslipidemia need be analyzed as major risk factors in addition to other conventional risk factors.

It will help to infer that early interventions are required to control the level of triglycerides and lipids with tighter glycemic control especially when there is presence of other risk factors. Furthermore, the current study aims to establish the need of multi factorial analysis to correlate with the impact of various combinations of risk factors present at a time.

RESULTS

27 % cases were in age group of 36-45 years while controls were 13% in the similar age group. In age group of 46-55 years cases were 58% while controls were 67%. Mean age of cases was50 years with standard deviation (SD) of 7.3 years. Mean age of controls was51 years with SD of 6.3 years. [**Table 1**]

In this third part of results, we analyzed presence of various risk factors in different angiographically diagnosed categories of CAD is significant in causation. Weight, BMI, HbA1c and various components of lipid profile eg; triglyceride, HDL, LDL, VLDL and total cholesterol were statistically tested by applying ANOVA and calculating p value to establish statistical significance. [**Table 2**]

HbA1c, Triglycerides and LDL were found to be highly significant ($p<0.01$). HDL and VLDL were as risk factor when statistically studied in five angiographic categories for significance it was not found to be significant ($p>0.05$). Total cholesterol is gradually increasing in trend with increasing severity of disease. As per statistical analysis it was statistically highly significant, p value <0.0.1. [**Table 3]**

DISCUSSION

In this study data collection was done in one multi-specialty center in the department of Cardiology over a period of 6 months from April 2017 to October 2017. Samples were chosen based on random selection from the pool of eligible samples. Patient whose both HbA1c and lipid profiles were done were eligible for random selection. Angiographically confirmed cases were divided in two groups. Patients whose angiography showed more than 50% stenosis in single coronary vessel, double coronary vessels or triple coronary vessels were taken as cases, while controls were assumed to be patients with normal coronaries or mild coronary artery disease who showed coronary stenosis less than 50% on angiography.

Equal number of cases and controls were chosen for the purpose of study. This study included patients coming to Ramesh hospitals, Vijaywada which is a corporate tertiary care cardiac center in capital region of Andhra Pradesh so maximum patients reporting here were from affluent class of society.

On demographic profiling of study population, it was observed that age-wise distribution showed that 62% of samples belonged to an age group of 46-55 years while only 2% were less than 35 years. In these 27% cases and 13% controls were in an age group of 36-45 years. In an age group of 46-55 years cases were 58% while controls were 67% as per distribution. Mean age of cases was 50 Years with standard deviation of 7.3 Years. Mean age of controls was 51 Years with standard deviation of 6.3 Years. This is consistent with the study by **Gajanan D. al**[3] section of age 55.39 years in older age group. In **Bhattacharyya et al**[6] study mean age was 57.3 years with standard deviation of 8.7 years as they included all age groups. In the study conducted by **Sriharibabu et al**[13] manage was 54.5 years mean which is in consistency with our study. On equating age distribution as per our age criteria their study showed that 1.87%wasbelow 35 years of age, 20.78% between 35 and 45 years and 48.9% from 45 to 55 years. These findings are almost similar in graphical representation of age distribution of our study. This observation was in accordance with published literature that CAD and CAD related mortality occurs at a younger age in Indians.[14] **Iyanger et al**[19] also observed mean age in their study to be 49 years which is consistent with our study.

On gender-wise distribution, analysis showed that male gender is statistically highly significant risk factor ($p<0.01$). In cases 78% were male compared to 51% in controls while females were only 22% in cases and 49% in controls. In cases M:F ratio was 3.5:1. In total sample selected 64% were male and M:F ratio in this study was 1.81:1. It is in consistency with findings of **Gajanan D. al**[3]. In their study males 67.6% and M: F ratio was 2.09:1. It was 1.91:1 in a large-scale study done by **Sriharibabu et al**[13] in Andhra Pradesh.

One of the best documented and the most consistent risk factor for coronary atherosclerosis was proved to be the male gender. The protective effects of estrogens in preventing atherosclerosis have been clearly demonstrated in earlier epidemiologic studies.[2] In another study[3], a profile of acute MI in young patients showed a male: female ratio of 20:1, where as in **Sricharan et al** study,[1] it was 9:1. But trend remains the same. **Iyanger et al**[19] observed M:F ratio to be 2.12:1 in their study which clearly demonstrates male preponderance as in our study.

Presence of hypertension as a risk factor was statistically highly significant ($p<0.01$). 73% cases had known hypertension and all were on treatment. **Sriharibabu et al**[13] also showed hypertension as the most prevalent risk factor along with diabetes and smoking in all CAD cases. Similar findings were found in **Gajanan D.** et al[3] study. The prevalence rate for hypertension in India varies between 20% and 40% in urban areas and 12% to 17% in rural areas.[25] The population attributed risk due to hypertension in India is 28.9% for acute myocardial infarction.[16] **Bhasin et al** also showed 33% study population was suffering from hypertension which is almost half the proportion detected in our study. It can be explained by patients coming to our hospital are for hypertension follow-up.

On statistical significance testing weight, HDL, and VLDL were not found to be significant while BMI, HbA1c, LDL, triglyceride and total cholesterol were found to be significantly associated with CAD causation.

Diabetes mellitus as a risk factor was statistically highly significant ($p<0.01$). 50% of total study subjects had diabetes. 76% cases had diabetes while only 24% controls were with diabetes. This shows that diabetes is an independent factor in causation of CAD. In other studies, also diabetes is significant risk factor for CAD causation.[4,23,26,32,29] This is in consistent with our study. **Iyanger et al[19]** and **Gajanan D. et al[3]** showed prevalence of diabetes as 44% and 48.5% respectively. In the **Chennai Urban population study** (CUPS) the prevalence rates of coronary artery disease were 9.1% in normal subjects and 21.4% in those with type 2 diabetes. The attributable risk due to diabetes for myocardial infarction was 20.5% in the Inter heart study.[16]

HbA1c as risk factor was statistically highly significant ($p<0.01$) in our study. HbA1cwas deranged in 69% cases against 20% of controls with mean of 7.58. It is deranged based on test values more than 6.5. 44% of total sample were observed having deranged HbA1c levels. These finding are consistent with **Bhasinet al**[17] study. HbA1c as risk factor when statistically studied in five angiographic categories for significance it was found to be highly significant ($p<0.01$). **Ravipati**[8] **et al** showed that the HbA1c level increased significantly with the number of arteries involved with CAD in diabetics.

Berry et al[5] found that fasting blood glucose, HbA1c, and presence of diabetes were associated with the severity and progression of coronary atherosclerosis. They concluded that better glycemic control favorably influences CAD in patients with abnormal glucose tolerance or diabetes. Findings of **Bhattacharya et al**[6] are consistent with the present study which also demarcates HbA1c as an independent factor for severity of CAD. **Gong**[9] studied the association of glycemic variability and the presence and severity of CAD.

Our findings suggest that in patients of coronary artery disease when conventional risk factors were evaluated, diabetes emerged as an independent predictor of the extent of obstructive CAD. Prior studies of angiographic progression of CAD using serial quantitative angiography to define predictors of severity or progression of disease burden including **CASS registry,**[10] have also consistently identified diabetes as an independent predictor of severity or disease progression[11]. Atherosclerosis affects the coronary arteries of diabetic patients more severely and diffusely than those of non-diabetics.[9]

On comparing lipid profile of our study to **Bhasin et al**,[17] and **Gajanan et al**,[3] found that triglycerides, LDL, and total cholesterol were very much higher like our study while HDL and VLDL are variable in comparison with other studies. But all these studies along with our study show consistent trends and statistical significance between dyslipidemia with lower level of HDL, higher total cholesterol, higher LDL and higher triglycerides. However, inter study variation in mean values was noted which was highly variable. Lipid profile was deranged in 62.7% samples while 41% were known dyslipidemias. **Bhasinet al**[17] also showed similar findings with 70.9% having dyslipidemia.

Dyslipidemia as risk factor was statistically highly significant ($p<0.01$) which was present among 37.83 % patients in **Pathaket al**[4] study which is comparable to study done by **Kaul et al**[10] which was 36.3% and to our study i.e., 41%. These studies indicate that abnormalities in lipid metabolism play an important role in development of CAD in Indians.

Deranged lipid profile as a risk factor was statistically significant ($p<0.05$) in our study. Lipid profile tested was deranged in 78% of cases and 58 % of controls which is consistent with the observations made by **Dhadwadet al.**[11] 20% patients showed hypertriglyceridemia in the fasting state while 68%of patients show hypertriglyceridemia (TG >200mg%) in their postprandial state. **Iyanger et al**[19] reported a significant association between the magnitude of the postprandial TG rise and low fasting plasma HDL cholesterol concentration.

Triglyceride as a risk factor when statistically studied in five angiographic categories for significance it was found to be highly significant ($p<0.01$) which shows rising triglycerides are also directly related to severity of coronary artery diseases. Mean triglyceride value was 257mg%. **Axelson et al** showed 50% greater TG rise in CAD. The **ARIC study** finding of a large sample of men and women supports the interpretation of **Axelson et al** that rise in lipoprotein is of intestinal origin.[22] 20% patients showed hypertriglyceridemia in the fasting state while 68%of patients show hypertriglyceridemia (TG >200mg%) in their postprandial state in **Dharwad et al study.**[21] These findings are consistent with our study.

Low **HDL** was not found to be significant ($p>0.05$). This agrees with the inverse relation between risk of CAD and HDL level. In the **PROCAM** study 45% subjects who developed CHD had HDL cholesterol less than 30 mg %.[11.]

LDL was found to be highly significant ($p<0.01$) with mean 165 mg %, which was like Dharwad et al[21] study. **VLDL** was non-significant ($p>0.05$) with mean 33mg % which was not in consistency with **Sekhri et al**[21] study. **Hypothyroidism** was statistically non-significant ($p>0.05$). Hypothyroidism could not be proved to be associated with CAD most probably because of very few numbers of hypothyroid in study sample. So, our study is not viable enough to throw light on hypothyroidism as risk factor. **Positive family history of CAD** was statistically highly significant ($p<0.01$) which was like the results in the previous studies conducted by **Pathaket al,**[6] **Kaul et al**[17] and **Kaulet al.**[18] **Tobacco habit** was statistically highly significant ($p<0.01$) as the **INTERHEART** study also observed that smoking was a greater risk factor in younger men than in women.[16]

The risk of CAD increased incrementally with smoking. Consistency of our findings is confirmed by other epidemiological studies from India which also suggest a greater association of smoking with CAD in younger individuals[8] such as study by **Sriharibabuetal.**[13] **Krishnan et al**[20] showed that 50.3% smoked some time in life which is like our finding in cases as of 53%.

BMI as a risk factor was statistically significant ($p<0.05$). this finding was like the study by **Gajanan D. et al**[3] and **Bhattacharyya et al.**[6]

This study is neither matched nor blinded study. It involves individuals from only one hospital for enrollment. Comparison with reference population and statistical comparison with other studies was not done which increases chance of bias and association by chance or selection.

Being a private corporate setup only affluent patients reported here. Also, study did not include MI patients or with previous history of CAD or PCI done in past or CABG done in past. Only those patients were included who underwent HbA1c and lipid profile assessment.

Treatment with statins, OHA and antihypertensive drugs among the CAD group could have resulted in lower BP, HbA1c and lipids, which could be one of the reasons for the lack of association of BP, HbA1c, triglycerides and lipids with CAD.

By this study we infer that early intervention can be helpful to control levels of triglycerides and lipids with tighter glycemic control especially when there is presence of other risk factors. Regular lipid profile and HbA1c test are required in Indian population to detect deranged parameters and act early. Multi factorial analysis is required to correlate impact of various combinations of factors present at a time. Furthermore, there is a need to conduct detailed multi-centric studies representing general population in regards to angiographic severity of CAD. Prospective longitudinal follow-up studies and complex statistical analysis to assess multi factorial as well as inter-factorial analysis are required to throw light on the true risk factors for various cardiovascular end points including mortality.

CONCLUSION

from this study can be drawn that CAD in terms of causation and severity is multi factorial. Most significant factors which can be attributed as independent risk factors for causation of CAD and as well attributed to severity of CAD were hypertension, diabetes, dyslipidemia, family history, high HbA1C, high LDL, high triglycerides, high total cholesterol along with tobacco and alcohol use. Dyslipidemia was prevalent in study sample for which they were unknown. Most common derangement observed in lipid profile was hypertriglyceridemia and low HDL. Our study also proves multi factorial causation of CAD with lesser number of risk factors present in controls and higher number of risk factors in cases.

HbA1c, LDL, triglycerides and total cholesterol were shown to act as independent risk factors for severity of disease.

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TABLES

Table 1: Demographic distribution of cases and control

		Group		Total
		Cases	Controls	

Age	<= 35	1(2%)	1(2%)	2
	36 – 45	12(27%)	6(13%)	18
	46 - 55	26(58%)	30(67%)	56
	56+	6(13%)	8(18%)	14
Gender	**Male**		58	64.44%
	Female		32	35.56%

Table 2: Risk factor significance analysis compiled group statistics

Risk Factor	**Cases**		**Controls**		**P Value (t-test)**
	Mean	**SD**	**Mean**	**SD**	
Weight	69.31	9.278	67.51	10.304	0.386
BMI	25.51	3.109	24.20	2.573	0.032
Pulse	78.31	10.146	83.00	11.897	0.047
SBP	128.00	17.787	126.44	13.341	0.640
DBP	82.44	10.478	82.00	8.146	0.823
Serum Creatinine	0.94	0.203	0.97	0.224	0.589
HBAIC	7.58	1.486	5.72	0.813	<0.001
Triglyceride	257.38	68.106	114.20	37.099	<0.001
HDL	35.89	10.456	39.09	7.713	0.102
LDL	164.69	83.433	84.96	27.111	<0.001
VLDL	32.87	20.677	28.62	13.359	0.251
Total Cholesterol	234.31	81.908	152.27	33.452	<0.001

Table 3: Group characteristics and risk factor significance analysis of angiographic severity

Risk Factors		Frequency	Mean	SD	P Value
Weight	Normal Coronary	16	66.13	11.407	0.543
	Mild CAD	29	68.28	9.768	
	SVD	12	66.50	9.709	
	DVD	19	69.26	9.544	
	TVD	14	71.79	8.460	
BMI Raw	Normal Coronary	16	24.38	2.156	0.286
	Mild CAD	29	24.10	2.807	
	SVD	12	25.08	3.175	
	DVD	19	25.58	3.372	
	TVD	14	25.79	2.860	
HB/IC	Normal Coronary	16	5.706250	.5397144	<0.001
	Mild CAD	29	5.734483	.9393295	
	SVD	12	6.983333	1.1983575	

	DVD	19	7.105263	1.2176816	
	TVD	14	8.735714	1.4505209	
Triglyceride	Normal Coronary	16	104.63	40.013	<0.001
	Mild CAD	29	119.48	34.982	
	SVD	12	289.42	55.801	
	DVD	19	230.79	79.167	
	TVD	14	266.00	48.229	
HDL	Normal Coronary	16	37.75	7.716	0.418
	Mild CAD	29	39.83	7.746	
	SVD	12	37.67	14.355	
	DVD	19	34.68	8.226	
	TVD	14	36.00	9.845	
LDL	Normal Coronary	16	83.19	16.558	<0.001
	Mild CAD	29	85.93	31.707	
	SVD	12	119.33	51.215	
	DVD	19	175.58	96.455	
	TVD	14	188.79	75.906	
VLDL	Normal Coronary	16	26.13	11.419	0.688
	Mild CAD	29	30.00	14.320	
	SVD	12	30.33	20.847	
	DVD	19	32.89	14.529	
	TVD	14	35.00	27.860	
Total	Normal Coronary	16	147.00	25.602	<0.001
	Mild CAD	29	155.17	37.185	
	SVD	12	188.17	42.697	
	DVD	19	244.68	99.233	
	TVD	14	259.79	68.617	