

Sociodemographic, Lifestyle And Clinical Predictors Of Coronary Artery Disease In A Cross- Sectional Epidemiological Study

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Abstract

Background: Coronary artery disease (CAD) is the leading cause of morbidity and mortality worldwide, with increasing prevalence in low, middle-income countries, including India. Epidemiological factors such as age, gender, lifestyle habits, socioeconomic status, comorbidities significantly influence CAD risk and outcomes.

Aim: To investigate the association of epidemiological factors among patients with CAD and healthy controls.

Methods: A retrospective study was conducted at Visakhapatnam city using Star Pinnacle Heart Centre hospital records of angiographically confirmed CAD patients (n=200) and age, sex matched controls (n=200). Sociodemographic variables (age, gender, religion, area of living, education, occupation, and socioeconomic status), lifestyle factors (smoking, tobacco use, alcohol consumption, physical activity, sleep, stress level), family history, comorbidities, symptoms were recorded and analyzed. Statistical analysis was performed using SPSS version 20 with chi-square, odds ratio applied to determine significance (p < 0.05 considered significant).

Results: Among the socioeconomic and demographic characteristics, all parameters except gender showed a highly significant association with the study population. Regarding lifestyle habits, all parameters were statistically highly significant. In terms of physical activity, all parameters except running were statistically insignificant. Sleep duration showed a statistically significant association with the study population, whereas stress level was found to be highly significant. Among comorbidities, all parameters except obesity were statistically highly significant. With respect to symptoms in CAD patients, all parameters except chest pain were statistically insignificant.

Conclusions: Epidemiological factors play a pivotal role in the development of CAD. Identifying high-risk groups through sociodemographic, lifestyle characteristics may help in targeted prevention and early intervention strategies.

Keywords: Coronary artery disease; epidemiological factors; sociodemographic factors; lifestyle; family history; comorbidities; symptoms.

INTRODUCTION

Coronary artery disease remains to be the leading cause of death worldwide, contributing to nearly 17.9 million deaths annually, accounting for approximately one-third of all global deaths¹. The burden is disproportionately higher in low and middle income countries, where rapid urbanization, demographic transition, lifestyle changes have driven a sharp rise in incidence and mortality². Epidemiological evidence consistently demonstrates that sociodemographic factors, lifestyle behaviours and comorbidities substantially shape CAD risk profiles across populations.

In India, CAD has emerged as a major public health challenge, with prevalence rates almost doubling over the past three decades³. Urban populations have been reported to have higher prevalence (10-12%) compared to rural areas (4 - 6%)⁴. South Asians, including Indians are known to experience CAD at younger ages, with more severe disease and higher case fatality than many Western populations⁵. The INTERHEART study further established that modifiable epidemiological risk factors-smoking, diabetes, hypertension, dyslipidemia, abdominal obesity, psychosocial stress and low physical activity-account for over 90% of myocardial infarction risk in South Asians⁶.

At the regional level, the India state level disease burden initiative reported that ischemic heart disease is the leading cause of years of life lost in Andhra Pradesh, reflecting the growing impact of CAD on premature mortality in this state⁷. Hospital-based studies in Visakhapatnam and Coastal Andhra have highlighted a clustering of risk factors including smoking, alcohol consumption, physical inactivity,

psychosocial stress, alongside significant urban-rural variation^{8,9}. These findings emphasize the importance of understanding epidemiological determinants of CAD at the community level, since cultural, occupational and socioeconomic factors differ widely across regions.

Given the high and rising prevalence of CAD in Visakhapatnam, it is important to evaluate the contribution of epidemiological parameters-including age, gender, religion, area of residence, education, occupation, socioeconomic status, family history, lifestyle behaviours and comorbidities to disease occurrence. Such studies are essential to identify high risk groups, inform targeted preventive strategies and contribute to the growing body of region-specific CAD epidemiology.

In this context, the present study was aimed to investigate the epidemiological determinants of CAD in a hospital-based population from Visakhapatnam, Andhra Pradesh, with special emphasis on sociodemographic, lifestyle and comorbid risk factors.

Research gap

Coronary artery disease is influenced by a wide range of factors, but in most research from this region, emphasis has been placed on biochemical and clinical findings rather than epidemiological determinants. The interconnection between sociodemographic characteristics (age, sex, religion, residence, education, occupation, socioeconomic status, family history), lifestyle practices (smoking, alcohol consumption, tobacco chewing, physical activity, sleep duration, stress levels) and CAD has not been systematically examined in the Visakhapatnam population. This leaves an important gap in understanding how epidemiological variables collectively contribute to CAD occurrence and progression in this setting.

Rationale

The present study was designed to bridge this gap by comprehensively assessing epidemiological parameters among CAD patients. By linking sociodemographic profiles, lifestyle behaviors, family history and comorbidities with CAD burden, the study provides region-specific insights. Such evidence can help to identify high-risk groups, support targeted preventive strategies and strengthen the epidemiological understanding of CAD in Visakhapatnam, complementing clinical and diagnostic findings.

Objectives

1. To evaluate the association of sociodemographic factors and to assess the influence of family history in study population.
2. To determine the role of lifestyle factors and comorbidities in study population.
3. To analyze the clinical symptoms in CAD patients.

MATERIALS AND METHODS

Study Design and Setting: A retrospective cross-sectional study was conducted at Star pinnacle heart center hospital in Visakhapatnam, Andhra Pradesh.

Study Population: All clinically confirmed CAD patients diagnosed through standard cardiac investigations (2D ECHO, coronary angiogram, TMT and clinical evaluation) were included.

Data Collection: The informed consent was obtained from each and every participant and assured the confidentiality of all information about them. The personal information regarding age group, gender, religion, area of living, education level, occupation, socioeconomic status, family history of CAD, lifestyle factors, physical activity, sleep duration, stress levels, comorbidities and symptoms of CAD patients and controls were obtained in a predesigned questionnaire at the time of interview.

Statistical analysis

Data was analyzed using SPSS version 20. Descriptive statistics were expressed as percentages. Associations between categorical variables and CAD were evaluated using the chi-square test, odds ratio and significance was set at $p < 0.05$.

RESULTS

Table - 1: Socioeconomic and Demographic Characteristics

Socioeconomic and Demographic Characteristics	CAD patients N= 200 (%)	Controls N=200 (%)	Total N=400(%)	χ^2	P-Value
Age group					

28-38	4 (2)	50 (25)	54 (13.5)	50.9886	<0.001
39-49	64 (32)	68 (34)	132 (33)		
50-60	132 (66)	82 (41)	214 (53.5)		
Gender					
Male	137 (68.5)	120 (60)	257 (64.25)	3.1456	0.0761
Female	63 (31.5)	80 (40)	143 (35.75)		
Religion					
Hindu	175 (87.5)	155 (77.5)	330 (82.5)	8.355	0.0038
Muslim	7 (3.5)	7 (3.5)	14 (3.5)		
Christian	18 (9)	38 (19)	56 (14)		
Area of living					
Urban	157 (78.5)	89 (44.5)	246 (61.5)	48.8228	<0.001
Rural	43 (21.5)	111 (55.5)	154 (38.5)		
Education level					
Illiterate	20 (10)	8 (4)	28 (7)	15.1082	0.0001
SSC	33 (16.5)	28 (14)	61 (15.25)		
Intermediate	24 (12)	13 (6.5)	37 (9.25)		
Graduation	49 (24.5)	44 (22)	93 (23.25)		
Post-Graduation	74 (37)	107 (53.5)	181 (45.25)		
Occupation					
Unskilled labor	7 (3.5)	2 (1)	9 (2.25)	19.0784	<0.001
Farmer	12 (6)	20 (10)	32 (8)		
Business	53 (26.5)	62 (31)	115 (28.75)		
Employee	105 (52.5)	112 (56)	217 (54.25)		
Housebound	23 (11.5)	4 (2)	27 (6.75)		
Economic status					
LIG	21 (10.5)	16 (8)	37 (9.25)	25.4772	<0.001
MIG	82 (41)	39 (19.5)	121 (30.25)		
HIG	97 (48.5)	145 (72.5)	242 (60.5)		
Family history					
Negative	70 (35)	104 (52)	174 (43.5)	11.7586	0.0006
Positive	130 (65)	96 (48)	226 (56.5)		

Table 1 represents, the distribution of CAD patients and controls according to age group, gender, religion, area of living, education level, occupation, economic status and family history. The highest frequencies of CAD patients (66%) and controls (41%) were found in 50-60 age group, whereas the lowest frequencies of CAD patients (2%) and controls (25%) were found in 28-38 age group. The chi square p value was highly significant with age group of study population. The frequencies of male CAD patients were (68.5%) and controls (60%), whereas the female frequencies of CAD patients were (31.5%) and controls (40%). The highest frequencies was found in males than females. The chi square p value was insignificant with gender of study population. The highest frequencies of CAD patients (87.5%) and controls (77.5%) were observed in Hindu religion category, whereas the lowest frequencies of CAD patients (3.5%) and controls (3.5%) were found in Muslim religion category. The chi square p value was highly significant with religion of study population. The frequencies of CAD patients and controls in urban area were (78.5%) and (44.5%) respectively, whereas, the frequencies of CAD patients and controls were (21.5%) and (55.5%) in rural area. The chi square p value was highly significant with area of living of study population. The highest frequencies of CAD patients (37%) and controls (53.5%) were found in post-graduation category, whereas the lowest frequencies of CAD patients (10%) and controls (4%) were found in illiterate category. The chi square p value was highly significant with education level of study population. The highest frequencies of CAD patients (52.5%) and controls (56%) were found in employee category. The lowest frequencies of CAD patients (3.5%) and controls (1%) were found in unskilled labor category. The chi square p value was highly significant with occupation of study population. The highest frequencies of

CAD patients (48.5%) and controls (72.5%) were found in HIG category. The lowest frequencies of CAD patients (10.5%) and controls (8%) were found in LIG category. The chi square p value was highly significant with economic status of study population. The frequencies of negative family history in CAD patients and controls were (35%) and (52%) respectively, and positive family history frequencies of CAD patients and controls were (65%) and (48%) respectively. The chi square p value was highly significant with family history of study population.

Table - 2: Distribution of CAD patients and Controls according to Life style habits

Life Style Habits	CAD patients N= 200 (%)	Controls N=200(%)	Total N=400(%)	χ^2	P-Value
Smoking					
Non-smokers	105 (52.5)	168 (84)	273 (68.25)	45.7904	<0.001
Smokers	95 (47.5)	32 (16)	127 (31.75)		
Tobacco Chewing					
Non-chewers	168 (84)	190 (95)	358 (89.5)	12.8758	0.0003
Chewers	32 (16)	10 (5)	42 (10.5)		
Alcohol Consumption					
Non-Alcoholics	122 (61)	147 (73.5)	269 (67.25)	7.0944	0.0077
Alcoholics	78 (39)	53 (26.5)	131 (32.75)		

Table 2 represents, the distribution of CAD patients and controls according to smoking status, tobacco chewing and alcohol consumption. The frequencies of non-smokers in CAD patients and controls were (52.5%) and (84%) respectively, whereas the smoker frequencies in CAD patients and controls were (47.5%) and (16%) respectively. The highly significant chi square p value shows association with smoking status of study population. The frequencies of non-chewers in CAD patients and controls were (84%) and (95%) respectively, whereas the chewers frequencies in CAD patients and controls were (16%) and (5%) respectively. The highly significant chi square p value shows association with tobacco chewing of study population. The frequencies of CAD patients and controls who do not consume alcohol were (61%) and (73.5%) respectively, whereas the frequencies were (39%) and (26.5%) in CAD patients and controls who consume alcohol. The highly significant chi square p value shows association with alcohol consumption of study population.

Table - 3: Distribution of CAD patients and Controls according to Physical Activity

Physical Activity	CAD patients N= 200 (%)	Controls N=200(%)	Total N=400(%)	Odds Ratio	95% CI	P-Value
Running						
No	146 (73)	119 (59.5)	265 (66.25)	0.4954	0.2982 -0.8232	0.0067
30 minutes	31 (15.5)	51 (25.5)	82 (20.5)	1	~	~
1 hour	23 (11.5)	30 (15)	53 (13.25)	0.7928	0.3925 -1.6015	0.5175
Chi-square - (8.5536); P-Value - (0.0034)						
Walking						
No	129 (64.5)	132 (66)	261 (65.25)	0.7674	0.3494 -1.6856	0.5097
30 minutes	59 (29.5)	52 (26)	111 (27.75)	0.6610	0.2865 -1.5253	0.3319
1 hour	12 (6)	16 (8)	28 (7)	1	~	~
Chi-square - (1.0472); P -Value - (0.3062)						
Exercise						
No	134 (67)	144 (72)	278 (69.5)	1.2665	0.8265 -1.9407	0.2779
30 minutes	66 (33)	56 (28)	122 (30.5)	1	~	~

Chi-Square - (1.1794); P-Value - (0.2775)						
Yoga						
No	134 (67)	131 (65.5)	265 (66.25)	1.0400	0.6530 - 1.6563	0.8688
30 minutes	50 (25)	47 (23.5)	97 (24.25)	1	~~	~~
1 hour	16 (8)	22 (11)	38 (9.5)	1.4628	0.6860 - 3.1189	0.3249
Chi-Square - (1.0742); P-Value - (0.3000)						

Table 3 represents, the distribution of CAD patients and controls according to physical activity. The chi square p value was highly significant with running and the odds ratio p value was insignificant, whereas the chi square p value and odds ratio p value were insignificant with walking, exercise and yoga.

Table - 4: Distribution of CAD patients and Controls according to sleep duration

Sleep	CAD patients N= 200 (%)	Controls N=200(%)	Total N=400(%)	Odds Ratio	95% CI	P-Value
4 hours	24 (12)	16 (8)	40 (10)	0.5194	0.2557-1.0550	0.0700
5 hours	57 (28.5)	44 (22)	101 (25.25)	0.6014	0.3623-0.9982	0.0492
6 hours	52 (26)	54 (27)	106 (26.5)	0.8090	0.4922-1.3299	0.4033
7 hours	67 (33.5)	86 (43)	153 (38.25)	1	~~	~~
Chi-Square - (5.6704); P-Value - (0.0173)						

Table 4 represents, the distribution of CAD patients and controls according to sleep duration. The chi square p value was significant with sleep duration, whereas the odds ratio p value was insignificant.

Table - 5: Distribution of CAD patients and Controls according to Stress levels

Stress levels	CAD patients N=200(%)	Controls N=200(%)	Total N=400(%)	Odds Ratio	95% CI	P-Value
Low	28 (14)	83 (41.5)	111 (27.75)	1	~~	~~
Moderate	85 (42.5)	58 (29)	143 (35.75)	0.2302	0.1337-0.3962	0.0001
High	87 (43.5)	59 (29.5)	146 (36.5)	0.2288	0.1332-0.3930	0.0001
Chi-Square - (37.72); P-Value - (<0.001)						

Table 5 represents, the distribution of CAD patients and controls according to stress levels. The chi square p value and odds ratio p value were highly significant with stress levels.

Table - 6: Distribution of CAD patients and Controls according to comorbidities

Comorbidities		CAD patients N=200 (%)	Controls N=200(%)	Total N=400(%)	χ^2	P-Value
Obesity	Yes	20 (10)	16 (8)	36 (9)	0.4884	0.4846
	No	180 (90)	184 (92)	364 (91)		
Hypertension	Yes	122 (61)	45 (22.5)	167 (41.75)	60.9494	<0.001
	No	78 (39)	155 (77.5)	233 (58.25)		

Diabetes	Yes	78 (39)	39 (19.5)	117 (29.25)	18.3746	<0.001
	No	122 (61)	161 (80.5)	283 (70.75)		
Triglycerides	Yes	144 (72)	72 (36)	216 (54)	52.174	<0.001
	No	56 (28)	128 (64)	184 (46)		
HDL-C	Yes	142 (71)	94 (47)	236 (59)	23.8116	<0.001
	No	58 (29)	106 (53)	164 (41)		

Table 6 represents, the distribution of CAD patients and controls according to comorbidities. The chi square p value was highly significant with hypertension, diabetes, triglycerides and HDL-C, whereas insignificant with obesity.

Table - 7: Distribution of CAD patients according to symptoms

Symptoms		Male N=137(%)	Female N=63(%)	Total N=200(%)	χ^2	P-value
Chest pain	Yes	130 (94.89)	54 (85.7)	184 (92)	4.9372	0.0263
	No	7 (5.10)	9 (14.2)	16 (8)		
Dyspnea	Yes	136 (99.2)	63 (100)	199 (99.5)	0.4622	0.4966
	No	1 (0.72)	0 (0)	1 (0.5)		
Palpitations	Yes	44 (32.1)	29 (46)	73 (36.5)	3.6052	0.0576
	No	93 (67.8)	34 (53.9)	127 (63.5)		
Syncope	Yes	18 (13.1)	15 (23.8)	33 (16.5)	3.5666	0.0590
	No	119 (86.8)	48 (76.1)	167 (83.5)		
Cardiogenic shock	Yes	11 (8.02)	8 (12.6)	19 (9.5)	1.0943	0.2955
	No	126 (91.9)	55 (87.3)	181 (90.5)		
Sudden cardiac arrest	Yes	2 (1.45)	0 (0)	2 (1)	0.929	0.3351
	No	135 (98.5)	63 (100)	198 (99)		
Acute LVF	Yes	37 (27)	25 (39.6)	62 (31)	3.2413	0.0718
	No	100 (72.9)	38 (60.3)	138 (69)		

Table 7 represents, the distribution of CAD patients according to symptoms. The chi square p value was significant with chest pain, whereas insignificant with dyspnea, palpitation, syncope, cardiogenic shock, sudden cardiac arrest and acute LVF.

DISCUSSION

This study investigated the prevalence of several socioeconomic and demographic factors were found to influence the occurrence of coronary artery disease, reflecting broader epidemiological trends. Participants were of both sexes and aged 28-60 years CAD patients along with controls.

Age emerged as a major determinant, with the majority of CAD patients belonging to the 50-60 years group (66%), compared to (41%) in controls, while younger age groups showed significantly lower CAD prevalence. Advancing age is a well-established risk factor for CAD due to cumulative vascular changes and higher burden of comorbidities. This aligns with the INTERHEART study (Yusuf et al., 2004)⁶, which reported that CAD incidence increases sharply after the age of 40, with men presenting at an earlier age than women. Similarly, (Ralapanawa & Sivakanesan, 2021)¹⁰ confirmed age as a strong predictor of CAD

in South Asian populations. This finding aligns with earlier reports that advancing age is one of the strongest non-modifiable risk factors for CAD due to cumulative vascular injury, endothelial dysfunction and atherosclerotic progression¹¹.

Although, male gender was more frequent among CAD cases (68.5%) compared to controls (60%), the association was not statistically significant ($p = 0.076$). Prior research has consistently shown that men have higher CAD risk due to differences in sex hormones, lifestyle behaviors and earlier manifestation of atherosclerosis, highlighted higher CAD prevalence in Indian men compared to women. However, increasing CAD burden among women, especially postmenopausal, has also been reported, suggesting that gender differences may be narrowing¹².

Religion showed a significant association, with Hindus forming the majority of CAD patients (87.5%) compared to controls (77.5%), while Christians were relatively more common in controls (19%) than CAD patients (9%). Although, religion itself is not a direct biological determinant, cultural practices, diet and lifestyle linked to religious background may influence CAD risk. (Panigrahi, 2024)¹³ similarly observed variation in CAD prevalence across sociocultural groups, reflecting differences in lifestyle behaviors.

In the current analysis, Urban residence was strongly associated with CAD (78.5% of cases vs. 44.5% of controls; $p < 0.001$). A higher proportion of CAD cases were from urban settings compared to controls, consistent with international research. Urbanization is widely recognized as a risk amplifier for cardiovascular disease due to increased exposure to processed foods, sedentary lifestyles and psychosocial stressors. (Wei et al., 2024)¹⁴ observed a similar pattern, attributing higher CAD prevalence in urban populations to rapid lifestyle transitions and higher prevalence of modifiable risk factors.

Illiteracy was more frequent in CAD patients (10% vs. 4% controls), while higher education (post-graduation) was more common in controls (53.5% vs. 37%). Education reflects awareness, access to healthcare and ability to adopt preventive strategies. Education was inversely associated with CAD risk in our data, with a higher prevalence of lower educational attainment among cases. This relationship is well-established individuals with less education generally have limited access to healthcare resources, poorer health literacy and are less likely to engage in preventive behaviors, all of which elevate cardiovascular risk. The umbrella review by (Marques et al., 2023)¹⁵ and a recent mediation analysis (PLOS Medicine, 2024)¹⁶ both confirm that educational inequalities are strongly linked to ischemic heart disease (IHD) mortality, with behavioral risk factors explaining a substantial portion of these disparities.

Occupational status was significantly associated with CAD. A greater proportion of CAD patients were housebound (11.5% vs. 2%), while business and employee categories were common in both groups. Occupation reflects physical activity levels, stress, and socioeconomic status. (Ralapanawa & Sivakanesan, 2021)¹⁰ observed that sedentary employment and high job stress were linked to increased CAD prevalence in South Asia.

Economic differences were marked, with more patients belonging to middle-income groups (41% vs. 19.5%), whereas controls had a higher representation in high-income groups (72.5% vs. 48.5%). Lower economic status is associated with reduced access to quality healthcare, poor diet and higher exposure to risk factors. These findings are consistent with (Gupta et al., 2019)¹⁷ who demonstrated socioeconomic disparities as key drivers of CAD burden in India.

Positive family history was significantly associated with CAD (65% cases vs. 48% controls, $p = 0.0006$). This underscores the role of genetic predisposition and shared lifestyle patterns. The INTERHEART study (Yusuf et al., 2004)⁶ found family history to be an independent risk factor, while (Panigrahi, 2024)¹³ similarly confirmed that individuals with a positive family history face significantly higher CAD risk. Our study highlights that sociodemographic determinants, including age, education, occupation, residence and family history, strongly predict CAD risk. These findings align with global and regional evidence, confirming the need for targeted preventive strategies addressing urban populations, lower socioeconomic groups and individuals with positive family history. Taken together, these findings demonstrate that socioeconomic and demographic characteristics are integral epidemiological determinants of CAD. They not only shape exposure to traditional risk factors but also influence access to healthcare and preventive strategies. Integrating these factors into CAD risk assessment may improve early identification of high-risk groups and guide the design of targeted interventions.

In the present study, smoking was significantly more common among CAD patients (47.5%) compared to controls (16%), indicating a strong association ($p < 0.001$). Cigarette smoking is one of the most established modifiable risk factors for CAD, accelerating atherosclerosis through endothelial damage, oxidative stress and pro-thrombotic effects. The INTERHEART study (Yusuf et al., 2004)⁶ demonstrated

that smoking nearly triples the risk of myocardial infarction worldwide. Similarly, this aligns with findings from the study by coronary heart disease and risk factors in India (Krishnan et al., 2012)¹⁸, which reported that tobacco smoking remains among the top modifiable risk factors for CAD in Indian settings.

Chewing tobacco was also significantly associated with CAD in our population (16% of patients than in 5% of controls and this difference was statistically significant ($p = 0.0003$) has been linked with increased cardiovascular risk due to nicotine-induced sympathetic activation and endothelial dysfunction. Further, The Bhubaneswar slum population study by (Panigrahi et al., 2024)¹³ identified smokeless tobacco as a key behavioral determinant of CAD among Indian adults. This underscores the importance of including tobacco chewing in CAD prevention strategies.

Alcohol use was more frequent in CAD patients (39%) compared to controls (26.5%), showing a significant association ($p = 0.0077$). The relationship between alcohol and CAD is complex, with light- to-moderate intake sometimes considered protective, but heavy or chronic use being harmful. In our population, alcohol consumption emerged as a clear risk factor. Additionally, (Ralapanawa & Sivakanesan, 2021)¹⁰ described alcohol as a dose-dependent risk factor for CAD, particularly in Asian populations where binge-drinking patterns are common. Many past studies including prevalence of risk factors for coronary artery disease in government employees across India (Sekhri et al., 2014)¹⁹ have similarly observed harmful alcohol use as a contributor to cardiovascular risk, especially when combined with other lifestyle risk behaviors.

The present study demonstrates that smoking, tobacco chewing and alcohol consumption are significant lifestyle predictors of CAD. These findings confirm the established evidence that behavioral factors strongly influence cardiovascular risk, particularly in low- and middle-income countries where tobacco use in multiple forms and harmful drinking patterns are prevalent. Public health interventions focused on smoking cessation, elimination of smokeless tobacco and reducing harmful alcohol use remain critical strategies for reducing CAD burden.

In the present study, physical activity showed a significant association with CAD, particularly for running activity ($\chi^2 = 8.55$, $P = 0.0034$). CAD patients reported lower participation in regular running (15.5% for 30 minutes, 11.5% for 1 hour) compared to controls (25.5% and 15%, respectively). This aligns with earlier reports demonstrating that regular aerobic exercise substantially reduces the risk of CAD by improving vascular function, enhancing lipid metabolism and lowering blood pressure. Sedentary lifestyle has been consistently identified as a major modifiable risk factor for coronary artery disease, contributing to obesity, hypertension, insulin resistance and dyslipidemia. Our findings are consistent with the INTERHEART study, which demonstrated that lack of physical activity significantly increased the risk of myocardial infarction across global populations (Yusuf et al., 2004)⁶. Similarly, (Sekhri et al., 2014)¹⁹, in a large nationwide Indian study, found that insufficient physical activity was highly prevalent among government employees and significantly contributed to the overall burden of CAD risk factors.

Walking habits did not differ significantly between CAD patients and controls. While walking has been recognized as beneficial, studies indicate that moderate-to-vigorous physical activity has a stronger cardioprotective effect compared to low-intensity walking. In a meta-analysis, reported that individuals engaging in higher-intensity physical activity had up to a 30% lower risk of CAD compared with sedentary individuals. This may explain the lack of strong association in our study, as walking patterns may not have achieved sufficient intensity or duration to yield significant benefits²⁰.

For general exercise, our results showed no statistically significant differences between cases and controls, though controls reported slightly higher participation in structured physical activity. This aligns with the findings of (Krishnan et al., 2012)¹⁸, who observed that while awareness of exercise benefits has increased in India, actual adoption of sustained physical activity remains low, limiting its protective impact.

Interestingly, yoga participation was similar in both groups, with no significant protective effect observed. However, yoga has been reported to improve cardiovascular risk markers in previous trials. (Patel et al., 2017)²¹ demonstrated in a randomized controlled trial that yoga practice reduced blood pressure and improved lipid profiles among CAD patients. The lack of association in our study may be due to lower overall participation rates or variability in practice intensity.

Overall, inadequate physical activity has been consistently identified as a global risk factor for CAD. Together, our findings reinforce the evidence that structured physical activity, particularly aerobic exercise of moderate to vigorous intensity, is critical for reducing CAD risk. This supports the need for public health initiatives encouraging regular, moderate-to-vigorous physical activity as part of CAD prevention strategies in India.

In the present study, sleep duration was significantly associated with coronary artery disease ($\chi^2=5.67$, $P =$

0.0173). Short sleep duration (<6 hours) was more common among CAD patients (12%) compared to controls (8%), whereas optimal sleep (7 hours) was more frequent in controls (43%) than CAD patients (33.5%). These findings support the growing evidence that inadequate sleep is an independent cardiovascular risk factor.

Short sleep and CAD risk link to our results are consistent with evidence from the PURE study, which found that individuals sleeping <6 hours had significantly higher risk of cardiovascular disease and mortality compared to those with 7–8 hours of sleep (Wang et al., 2019)²². Similarly, Sleep restriction has been shown to elevate sympathetic activity, increase cortisol release and promote systemic inflammation, all of which accelerate atherosclerosis and endothelial dysfunction. (St-Onge et al., 2016)²³, highlighted that short sleep contributes to cardiometabolic risk through hormonal dysregulation, increased appetite, impaired glucose metabolism and hypertension. These mechanisms may explain the higher prevalence of CAD among short sleepers in our population. In our study, 7 hours of sleep was most common among controls, supporting the hypothesis that 7-8 hours represents the “protective” range for cardiovascular health. (Cappuccio et al., 2010)²⁴ in a meta-analysis also reported that both short (<6 h) and long (>9 h) sleep durations are associated with increased coronary heart disease risk.

In the present study, stress levels were significantly associated with study population ($\chi^2 = 37.72$, $p < 0.001$). The stress levels were markedly higher in CAD patients than controls. These findings highlight psychosocial stress as an important and independent predictor of CAD.

In the global evidence INTERHEART study (Yusuf et al., 2004)⁶ provided large-scale evidence that psychosocial stress contributes substantially to myocardial infarction risk, independent of traditional risk factors, across diverse populations worldwide. Our findings reinforce this global perspective within an Indian population. (Panigrahi, 2024)¹³, in his study also found that stress was a significant behavioral determinant of CAD among Indian adults. This supports the applicability of our findings in regional epidemiology. In Mechanistic pathways psychological stress influences CAD risk through multiple biological pathways including sympathetic activation, elevated catecholamines, increased cortisol secretion, endothelial dysfunction and pro-inflammatory states. (Cohen et al., 2007)²⁵ further emphasized that chronic stress impairs immune and metabolic regulation, thereby predisposing individuals to cardiovascular disease.

Taken together, our findings demonstrate that stress is a critical non-traditional risk factor for CAD. Both moderate and high stress levels were significantly overrepresented among CAD patients compared to controls, suggesting that stress management strategies such as lifestyle modification, counseling and stress reduction therapies.

In the present study, the frequency of obesity was less in both CAD patients and controls, with an insignificant p value ($p = 0.4846$). While obesity is recognized as a major risk factor for CAD, its impact in this study may have been masked by other stronger metabolic factors. Nevertheless, epidemiological studies, including the INTERHEART study, have shown central obesity to be a powerful predictor of myocardial infarction⁶.

In the present study, the frequency of hypertension was more in CAD patients (61%) compared with controls (22.5%), showing a strong association ($p < 0.001$). These findings establish hypertension as a strong comorbidity and predictor of CAD in our study population. Hypertension is one of the most important and modifiable risk factors for CAD. It accelerates atherosclerosis by increasing endothelial dysfunction, oxidative stress and arterial stiffness. The INTERHEART study demonstrated that hypertension contributed significantly to myocardial infarction risk across all global regions, including South Asia (Yusuf et al., 2004)⁶.

The frequency of diabetes mellitus was more in CAD patients (39%) compared with controls (19.5%), showing a strong association ($p < 0.001$). Diabetes promotes CAD through hyperglycaemia-induced oxidative stress, chronic inflammation and dyslipidemia, leading to accelerated plaque formation. Previous studies confirm that diabetic individuals have a two to four fold higher risk of CAD²⁶.

Elevated triglycerides level was present in 72% of CAD patients compared with 36% of controls ($p < 0.001$). Elevated triglycerides contribute to atherogenesis by promoting small dense LDL particles and lowering HDL, consistent with prior studies linking hypertriglyceridemia with increased cardiovascular risk²⁷.

Similarly, low HDL-C levels were more common in CAD patients (71%) than in controls (47%) ($p < 0.001$). Low HDL has long been considered an independent risk factor for CAD due to its role in reverse cholesterol transport and anti-inflammatory properties²⁸.

Collectively, these findings reaffirm that hypertension, diabetes and dyslipidemia (high triglycerides and

low HDL) are significant epidemiological determinants of CAD. Addressing these comorbidities through early screening, lifestyle modification and pharmacological interventions remains central to reducing CAD burden.

In the present study, the frequency of symptoms was almost similar in both male and female CAD patients. Chest pain shows significant association with study population. Chest pain remains the most common presenting complaints in CAD. Our findings are consistent with the Global Registry of Acute Coronary Events (GRACE), which documented chest pain and shortness of breath as the predominant presenting symptoms worldwide (Fox et al., 2006)²⁹. Similarly, (Krishnan, 2012)¹⁸ emphasized that typical angina is hallmark symptoms of CAD in the Indian population.

Palpitations (36.5%) and syncope (16.5%) were relatively less common in CAD patients but clinically important. Syncope was more frequent among women (23.8% vs. 13.1%), though not statistically significant. (Shen et al., 2017)³⁰ reported that syncope can occur in CAD due to arrhythmias, transient ischemia, or structural heart disease. Palpitations are often associated with ventricular ectopy and arrhythmias, which are recognized complications of CAD.

In our study, cardiogenic shock occurred in 9.5% of CAD patients, while sudden cardiac arrest was rare (1%). These findings are inconsistent with the incidence rates reported in global registries of acute coronary syndromes (Kolte et al., 2014)³¹ observed that cardiogenic shock complicates 5-10% of acute myocardial infarctions and carries high mortality. Sudden cardiac arrest, while infrequent, represents a catastrophic manifestation of CAD, as described by (Adabag et al., 2015)³².

Acute left ventricular failure (LVF) was observed in 31% of our patients, with higher prevalence in females (39.6%) compared to males (27%). This aligns with evidence that heart failure with preserved ejection fraction (HFPEF) is more common among women, while ischemic LV dysfunction is more frequent in men (Pfeffer et al., 2019)³³.

Taken together, our findings reaffirm that chest pain remain the predominant symptoms of CAD, while complications such as cardiogenic shock, LVF and arrhythmias contribute to disease severity. Gender-related differences in symptom presentation highlight the need for careful clinical evaluation, especially in women, to avoid under diagnosis.

Limitations of the Study

This study has certain limitations that should be considered when interpreting the findings. First, being a hospital-based study, the results may not represent the general population, as patients attending tertiary care centres often differ from the wider community in sociodemographic and lifestyle characteristics. Second, the cross-sectional design restricts the ability to establish causal relationships between epidemiological parameters and coronary artery disease; the associations observed should be interpreted as correlations. Third, information on lifestyle factors such as smoking, alcohol consumption, physical activity, sleep and stress was self-reported, which may introduce recall or reporting bias. Fourth, the study was conducted in Visakhapatnam and the findings may reflect regional patterns that limit their generalizability to other populations. Despite these limitations, the study provides valuable insights into how epidemiological determinants interlink with CAD and underscores the need for larger, multicentre, community-based prospective studies.

Strengths of the Study

This study has several notable strengths. It provides a comprehensive evaluation of epidemiological determinants of coronary artery disease, including sociodemographic, lifestyle, family history, symptomatology and comorbidity profiles, rather than focusing on isolated factors. By examining multiple epidemiological parameters in a single analytical framework, the study offers a holistic understanding of how these factors interlink with CAD. Conducted in Visakhapatnam, it adds region-specific evidence to the limited data available from Andhra Pradesh, thereby contributing to localized public health planning. The inclusion of diverse variables such as education, occupation, socioeconomic status and stress provides novel insights into social and behavioral influences on CAD. Furthermore, the study highlights modifiable epidemiological factors such as smoking, alcohol consumption, physical activity, sleep and stress-making the findings directly relevant for preventive strategies and community-based interventions.

Future Implications

The findings of this study highlight the importance of epidemiological determinants in the development and progression of coronary artery disease. By identifying associations between sociodemographic factors, lifestyle behaviors, family history and comorbidities with CAD, the study underscores the potential for targeted preventive strategies. Future research should focus on large-scale, community-based, prospective

studies to validate these associations and establish causal pathways. Integrating epidemiological profiling into routine risk assessment tools may improve early identification of high-risk individuals, particularly in resource-limited settings. Moreover, the evidence supports the development of region-specific public health interventions, including awareness campaigns, lifestyle modification programs and stress management initiatives, tailored to the needs of populations such as those in Visakhapatnam.

CONCLUSION

This study shows that the burden of coronary artery disease is significantly influenced by epidemiological factors. It was discovered that the prevalence and course of disease were strongly correlated with sociodemographic traits, lifestyle choices, family history and coexisting disorders. The results highlight the fact that CAD is heavily impacted by environmental, behavioural and social factors. By emphasizing these correlations in a Visakhapatnam hospital population, the study offers evidence relevant to the area that can direct public health initiatives and preventative measures. The increasing burden of CAD in comparable populations may be lessened by addressing modifiable risk factors such smoking, drinking, chewing tobacco, being physically inactive, getting little sleep and experiencing high levels of stress, as well as by identifying comorbidities early.

REFERENCES

1. World Health Organization. Cardiovascular diseases (CVDs). WHO Fact Sheet. 2021.
2. Johnson CO, Mensah GA, Roth GA, et al., Global burden of cardiovascular diseases and risk factors, 1990-2019: GBD 2019 Study. *Journal of the American College of cardiology* 2020;76(25):2982- 3021.
3. Jeemon P, Prabhakaran D, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. *Circulation*. 2016;133(16):1605-20.
4. Gupta R, Mohan I, Narula J. Trends in coronary heart disease epidemiology in India. *Annals of Global Health*. 2016;82(2):307-15.
5. Anand S,ounpuu S, Reddy S, Yusuf S, Global burden of cardiovascular diseases: Part II. *Circulation*. 2001;104(23):2855-64.
6. Hawken S, ounpuu S, Yusuf S, et al., Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (INTERHEART study): case-control study. *Lancet*. 2004;364(9438):937-52.
7. India State-Level Disease Burden Initiative CVD Collaborators. The changing patterns of cardiovascular diseases and their risk factors in the states of India: 1990-2016. *Lancet Glob Health*. 2018;6(12): e1339-51.
8. Devi P, Rao M, Xavier D, et al., Prevalence, treatments and outcomes of coronary artery disease in Indians: results from the PURE study. *Indian Heart J*. 2015;67(4):302-9.
9. Jeemon P, Prabhakaran D, Reddy KS, et al., educational status and cardiovascular risk profile in Indians. *Proceedings of the National Academy of Science of the U S A*. 2007;104(41):16263-8.
10. Ralapanawa U, Sivakanesan R. Epidemiology and the magnitude of coronary artery disease and acute coronary syndrome: A narrative review. *J Epidemiol Glob Health*. 2021;11(3):169-177.
11. Benjamin EJ, et al., heart disease and stroke statistics-2019 update. *Circulation*. 2019;139: e56-e528.
12. Gupta R, Xavier D. Emerging trends in cardiovascular disease in India. *Indian Heart J*. 2019;71(2):98-106.
13. Panigrahi A. Coronary artery disease, its socio-demographic and behavioral correlates: A cross- sectional study. *J Clin Epidemiol Glob Health*. 2024;12(1):34 - 40.
14. Wei, Y., et al., (2024). Influence of Socioeconomic Status on the Presence of Obstructive Coronary Artery Disease and Cardiovascular Outcomes in Patients Undergoing Invasive Coronary Angiography. *Healthcare*, 12(2),228. <https://www.mdpi.com/2227-9032/12/2/228>.
15. Marques, A. H., et al., (2023). The Role of Social Determinants of Health in Cardiovascular Diseases: An Umbrella Review. *International Journal of Environmental Research and Public Health*, 20(13), 56094.
16. PLOS Medicine Editors. (2024). Behavioral risk factors and socioeconomic inequalities in ischemic heart disease mortality in the United States: A causal mediation analysis using record linkage data. *Plos Med* 21(8): e1004455. <https://dx.plos.org/10.1371/journal.pmed.1004455>.
17. Gupta R, Xavier D. Emerging trends in cardiovascular disease in India. *Indian Heart Journal*. 2019;71(2):98-106.
18. Krishnan MN. Coronary heart disease and risk factors in India - on the brink of an epidemic? *Indian Heart Journal*. 2012;64(4):364-367. <https://doi.org/10.1016/j.ihj.2012.07.001>.
19. Sekhri T, Kanwar RS, Wilfred R, et al., Prevalence of risk factors for coronary artery disease in government employees across India. *BMJ Open*. 2014;4(12): e005346. <https://bmjopen.bmj.com/content/4/12/e005346>.
20. Sattelmair J, Pertman J, Ding EL, Kohl HW 3rd, Haskell W, Lee IM. Dose response between physical activity and risk of coronary heart disease: a meta-analysis. *Circulation*. 2011;124(7):789- 795.
21. Patel NK, Newstead AH, Ferrer RL. The effects of yoga on physical functioning and health related quality of life in older adults: A systematic review and meta-analysis. *Plos one*. 2017;12(8): e0182848.
22. Chuangshi Wang, Bangdiwala, Shrikant I, Sumathy Rangarajan, et al., Association of estimated sleep duration and naps with mortality and cardiovascular events: a study of 116,632 people from 21 countries. *European Heart Journal* (2018) 0, 1-

10 doi:10.1093/eurheartj/ehy695.

23. Marie-Pierre St-Onge, Grandner MA, Brown D, et al., Sleep Duration and Quality: Impact on Lifestyle Behaviors and Cardiometabolic Health a scientific Statement from the American Heart Association Circulation. November 1, 2016;134: e367-e386.doi:10.1161/CIR.0000000000000444.
24. Francesco P. Cappuccio, Lanfranco D'Elia, Pasquale Strazzullo, Michelle A. Miller, Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. SLEEP 2010;33(5):585-592.
25. Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. JAMA. 2007;298(14):1685-1687.
26. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects. N Engl J Med. 1998;339(4):229-234.
27. Toth PP. Triglyceride-rich lipoproteins as a causal factor for cardiovascular disease. Vascular Health Risk Manag. 2016; 12:171-183.
28. David J. Gordon, Jeffrey L. Probstfield, Robert J. Garrison, et al. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. Circulation. 1989;79(1):8-15.
29. Fox KAA, Steg PG, Eagle KA, et al., The Global Registry of Acute Coronary Events (GRACE). Am J Med. 2006;119(10 Suppl 1):1-11.
30. Shen WK, Sheldon RS, Benditt DG, et al., 2017 ACC/AHA/HRS guideline for evaluation and management of patients with syncope. Heart Rhythm. 2017;14(8): e155-e217.
31. Kolte D, Khera S, Aronow WS, et al., Trends in incidence and outcomes of cardiogenic shock complicating ST-elevation myocardial infarction. Am J Med. 2014;127(10):949-957.
32. Adabag AS, Luepker RV, Roger VL, Gersh BJ. Sudden cardiac death: epidemiology and risk factors. Nat Rev Cardiol. 2015;12(9):508-517.
33. Pfeffer MA, Shah AM, Borlaug BA. Heart failure with preserved ejection fraction in perspective. Circulation Research. 2019;124(11):1598-1617.