ORIGINAL RESEARCH ARTICLE

One-Year Trajectories of Diabetes, Hypertension, and Dyslipidemia after Coronary Artery Disease: A Sex-Stratified Retrospective Cohort Study from Kerala, India

Ambili P V[1], Punitha VC[2*], Sreelal TP[3], Shankar K[4], Dinesh Roy D[5], Jiju JS[6], Arun William[7], Jayapal V[8], Abhijith MS[9], Meena Beegam M[10]

[1,10]Department of Community Medicine, Meenakshi Academy of Higher Education and Research (MAHER - Deemed to be University), Chennai, Tamil Nadu, India
[2,4]Department of Community Medicine, Meenakshi Medical College and Research Institute (MMCHRI), Kanchipuram, Tamil Nadu, India
[3]Department of Community Medicine, Dr. Moopens Medical College, Wayanad, Kerala, India
[5]Department of Cytogenetics, Genetika, Centre for Advanced Genetic Studies, Thiruvananthapuram, Kerala, India
[6]Division of Pathology, Regional Cancer Centre (RCC), Thiruvananthapuram, Kerala, India
[7]Department of Biochemistry, Mount Zion Medical College, Adoor, Kerala, India
[8]Department of Cardiology, Hridayalaya Heart and Robotic Research Centre Pvt. Ltd., Thiruvananthapuram, Kerala, India
[9]Security Business Group, Cisco Systems India Pvt. Ltd., Bangalore, Karnataka, India

DOI: 10.55489/njcm.170420266252

ABSTRACT

Background: Cardiovascular diseases remain the leading cause of global mortality, with post-CAD cardiometabolic risk factor control frequently suboptimal, particularly among women. This study analysed sex-specific trajectories of diabetes, hypertension, and dyslipidemia one year after CAD in Kerala, India, and exploratorily examined associations with environmental and lifestyle factors.

Methods: A cross-sectional study with retrospective baseline ascertainment was conducted among 420 CAD survivors. McNemar's test compared paired pre- and post-comorbidity status within each sex group, while chi-square test assessed sex differences in trajectory distributions.

Results: Substantial resolution of baseline risk factors was observed at one year, with diabetes, hypertension, and dyslipidemia resolving in 36.7%, 39.3%, and 36.7% of participants respectively. New-onset comorbidities were equally frequent at 24%, 21.9%, and 18.8% respectively. Exploratory analyses suggested associations between self-reported environmental exposures and specific diabetes trajectories.

Conclusions: One-year post-CAD, considerable proportions experienced resolution of cardiometabolic conditions, while comparable subsets developed new or persistent comorbidities. Women showed higher persistent dyslipidemia. Environmental and lifestyle associations identified warrant prospective investigation.

Keywords: Coronary artery disease, Cardiometabolic risk, Sex-specific trajectories, Post-CAD comorbidities

ARTICLE INFO

Financial Support: None declared
Conflict of Interest: The authors have declared that no conflict of interest exists.
Received: 28-11-2025, **Accepted**: 06-03-2026, **Published**: 01-04-2026
***Correspondence:** Punitha VC (Email: drpunitha77@yahoo.co.in)

How to cite this article: Ambili PV, Punitha VC, Sreelal TP, Shankar K, Roy DD, Jiju JS, William A, Jayapal V, Abhijith MS, Beegam MM. One-Year Trajectories of Diabetes, Hypertension, and Dyslipidemia after Coronary Artery Disease: A Sex-Stratified Retrospective Cohort Study from Kerala, India. Natl J Community Med 2026;17(4):284-292. DOI: 10.55489/njcm.170420266252

www.njcmindia.com | pISSN: 0976-3325 | eISSN: 2229-6816 | Published by Medsci Publications

INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of death worldwide, accounting for roughly 18 million deaths annually. The vast majority of this burden falls on low- and middle-income countries; about 80% of CVD deaths occur in developing nations.[1] In India, CAD rates have risen dramatically in recent decades. In fact, CAD now causes over a quarter of India's medically certified deaths, and Indians experience myocardial infarction on average nearly 10 years earlier than Western populations.[2] Kerala exemplifies this trend: a recent community survey found the prevalence of definite CAD has nearly tripled since 1993, accompanied by very high rates of cardiometabolic risk factors (59% overweight/obesity, 28% hypertension, 15% diabetes, 52% hypercholesterolemia).[3]

Women with CAD tend to present later in life but with a heavier risk factor burden than men.[4] They are on average older and have more co-morbid conditions at first presentation.[5] For example, in a large North Indian cohort (n = 3660), hypertension and diabetes were observed in 65.4% and 60.7% of women with CAD, respectively, compared with 52.9% and 42.5% of men.[4] Women also generally fare worse after CAD; one study noted higher mortality in women despite their older age at presentation.[5] Diabetes in particular is especially deleterious in females: asymptomatic women with type 2 diabetes were found to have nearly twice the prevalence of early coronary microvascular damage (a precursor to CAD) than diabetic men.[6] These sex-specific disparities underscore the need for tailored secondary prevention since women with CAD often require more aggressive risk-factor management.

Guidelines make clear that intensive management of blood pressure, glycemia, lipids, and lifestyle after a CAD event can greatly reduce recurrent CVD events.[7] Yet real-world control of these risk factors is often poor, especially in resource-limited settings. Observational studies document large gaps between recommended and actual care. For instance, registry data from Asia show very low adherence to guideline targets.[8] In Indonesia, only 1.8% of post-MI patients met all treatment goals at one year: LDL cholesterol control was achieved in only 5.1%.[9] Similar shortcomings in risk factor control have been reported across South Asia.[8,9] In summary, although effective secondary prevention strategies exist, there are systemic barriers and implementation gaps (particularly in LMICs) that leave many post-CAD patients with suboptimal blood pressure, glucose, and lipid control.

In this context, the status of diabetes, hypertension, and dyslipidemia in Kerala patients was analysed by retrospectively comparing their status at the time of the CAD event to their status one year later. Kerala's high NCD burden and escalating CAD prevalence make it an informative setting for this study. This study was exploratory and hypothesis-generating in nature. The primary objective was to observationally profile sex-stratified comorbidity trajectories and identify associated factors, without assuming causality.

METHODOLOGY

Study Design and Setting: This study was designed as a cross-sectional study with retrospective baseline ascertainment, conducted one year after the index CAD event. The study was carried out in the state of Kerala, India. The participants were recruited from tertiary care hospitals providing follow-up care to patients with documented CAD. Laboratory analyses were performed at a centralized accredited diagnostic facility to ensure uniformity in biochemical measurements. The study period extended from June 2022 to June 2025.

Study Population and Eligibility Criteria: The source population comprised adult patients with clinically documented CAD who were attending follow-up services at participating institutions. Individuals aged 30 to 60 years who had completed one year following their index CAD event were eligible for inclusion. All participants were receiving guideline-directed secondary prevention therapy at the time of enrolment, including pharmacological treatment and lifestyle counselling.

Patients were excluded if they had a history of exposure to ionizing radiation, chemotherapy, or known mutagenic agents to avoid confounding effects on cardiometabolic outcomes from cardiotoxic therapies or radiation-induced vascular damage, or if complete medical documentation of the index CAD event was unavailable. Eligibility was verified through hospital records and discharge summaries.

The study was conducted in the Outpatient Departments and follow-up clinics of selected tertiary care facilities in Thiruvananthapuram, Kerala, primarily Hridayalaya Heart and Robotic Research Centre Pvt. Ltd. This centre was selected due to its high volume of post-CAD patients and well-maintained clinical records, which facilitated efficient recruitment during routine medication and cardiovascular monitoring visits. All subsequent clinical and biochemical analyses were performed at Genetika, Centre for Advanced Genetic Studies, Thiruvananthapuram, Kerala. Participants were recruited consecutively during their routine one-year follow-up visits. Out of 490 screened individuals, 70 were excluded due to missing baseline documentation or meeting specific exclusion criteria, resulting in a final enrolled cohort of 420 participants. Flow diagram of participants is provided in Supplementary Figure S1.

Sample Size: The sample size was determined based on standard epidemiological principles for cross-sectional assessments. In the absence of precise regional prevalence estimates for the specific comor-

bidity trajectories, an expected prevalence (p) of 50% for coronary artery disease-related risk factors was utilized to ensure a conservative, maximum sample size requirement. Assuming a 95% confidence level ($Z = 1.96$) and a 5% allowable margin of error ($d = 0.05$), the minimum required sample size was calculated as 384 participants using the standard formula $n = (Z^2 \times p \times q)/d^2$. To account for a potential 10% rate of non-response or incomplete medical records, the final target sample size was adjusted to 420 participants.

Definition and Verification of Coronary Artery Disease: Coronary artery disease was defined based on documented clinical evidence of acute myocardial infarction confirmed by cardiac biomarkers and electrocardiographic findings, angiographically demonstrated coronary stenosis of at least 50 percent in one or more major coronary vessels, prior percutaneous coronary intervention or coronary artery bypass grafting, or a hospital discharge diagnosis consistent with ischemic heart disease. Diagnostic confirmation was obtained from hospital records, angiographic reports, and cardiology consultation documentation.

Baseline Data Ascertainment: Baseline comorbidity status at the time of the index CAD event was determined retrospectively using hospital medical records, laboratory reports at admission, documented physician diagnoses, and medication history. A structured questionnaire administered at the one-year assessment was used to supplement documented information. Classification of baseline diabetes, hypertension, and dyslipidemia required documented physician diagnosis or laboratory values meeting predefined diagnostic thresholds. Self-reported information without supporting documentation was not considered sufficient for classification.

One-Year Clinical and Laboratory Assessment: At one year (with a window of +2 months) following the index CAD event, participants underwent standardized clinical and biochemical evaluation. Participants were instructed to refrain from tobacco, caffeine, and heavy physical activity for at least 30 minutes prior to measurement. Blood pressure was measured on the right arm using a calibrated automated sphygmomanometer after at least five minutes of seated rest. A three-reading protocol was followed, with the first reading discarded and the average of the subsequent two readings recorded. Heart rate, height, weight, and abdominal circumference were measured using standardized procedures. Body mass index was calculated as weight in kilograms divided by height in meters squared.

After an overnight fast of eight to ten hours, venous blood samples were collected under aseptic conditions. Biochemical analyses included fasting plasma glucose, serum triglycerides, total cholesterol, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. All assays were performed using automated analysers in accordance with manufacturer protocols.

Operational Definitions of Comorbidities: Classification of the comorbidities was based on nationally accepted guidelines that provide uniform cut-off values widely utilized in Indian clinical and epidemiological research. Diagnostic criteria were defined using established national guidelines. Diabetes was identified based on a fasting plasma glucose of ≥126 mg/dL, in accordance with the ICMR Guidelines for Management of Type 2 Diabetes (2018).[10] Hypertension was diagnosed when systolic blood pressure reached ≥140 mmHg or diastolic blood pressure ≥90 mmHg, as per the MoHFW Standard Treatment Guidelines for Hypertension.[11] Dyslipidemia was defined using the CSI Dyslipidemia Guidelines (2024),[12] with cut-off values of total cholesterol ≥200 mg/dL, LDL cholesterol ≥100 mg/dL, HDL cholesterol <40 mg/dL in men and <50 mg/dL in women, triglycerides ≥150 mg/dL, and non-HDL cholesterol ≥130 mg/dL.

Dyslipidemia was classified using an LDL-cholesterol cut-off of ≥100 mg/dL as the primary threshold. This cut-off aligns with the prevalence marker explicitly highlighted in the Cardiological Society of India (CSI) 2024 guidelines and is consistent with operational definitions used in recent Indian studies on patients with acute coronary syndrome and statin-treated cohorts.[12,13,14] Although more stringent targets (LDL-C <55 mg/dL) are recommended for very-high-risk secondary prevention in post-CAD patients, the ≥100 mg/dL threshold was deliberately selected to maintain consistency with retrospective baseline diagnoses from hospital records (which routinely apply general population or screening cut-offs) and to enable straightforward binary trajectory categorisation (present/absent) across both time points in this real-world treated cohort.

Definition of Comorbidity Trajectories: Based on the comparison of the subject's status at these two time points, subjects were categorized into one of four trajectory groups (Table 1). These categories allowed to compare the pre-CAD and current (after one year of CAD) status of each comorbidity, reflecting progression or improvement during the one-year period.

It is important to note that the 'Resolution' category likely reflects effective pharmacological normalization of clinical parameters rather than true biological disease reversal, as all participants were on secondary prevention pharmacotherapy.

Treatment Exposure: At the time of one-year assessment, all participants were receiving guideline-based secondary prevention therapy, including antiplatelet agents, beta-blockers, statins, and renin-angiotensin system inhibitors where indicated. Medication use was verified through prescription records and patient-held treatment documentation.

Table 1: Trajectory groups

Category	Condition		Interpretation
	Before CAD	After One Year	
Resolution	Present	Absent	The condition was resolved or well-controlled post-treatment.
Consistently Healthy	Absent	Absent	No occurrence of the condition in either period.
New-onset	Absent	Present	The condition was diagnosed during the one-year period.
Persistent	Present	Present	The condition persisted despite treatment or remained poorly controlled.

Data Collection: Socio-demographic characteristics, lifestyle behaviours, environmental exposures, and psychosocial variables were collected using a structured and pre-tested questionnaire. Clinical measurements were recorded using standardized case-report forms. Socioeconomic status was assessed using the modified Kuppuswamy socioeconomic status scale (updated for 2021).[15] Physical activity levels were assessed using the validated International Physical Activity Questionnaire (IPAQ).[16] Other lifestyle variables, including dietary patterns and daily water intake, were evaluated using the structured, pre-tested questionnaire based on self-reported recall, which did not undergo formal test-retest reliability validation. Self-reported environmental exposures (high air pollution, environmental toxins, household pesticides) were recorded as dichotomous variables based on participant perception of residential or occupational exposure in the preceding year; no objective measurement or validated exposure questionnaire was used.

Handling of Missing Data: Participants with incomplete baseline documentation for a specific comorbidity were excluded from trajectory analysis for that condition. Laboratory variables missing at the one-year assessment were handled using complete-case analysis. The overall proportion of missing data for primary variables was low and did not exceed five percent. No imputation procedures were performed.

Statistical Analysis: All statistical analyses were conducted using Stata software. Categorical variables were summarized as frequencies and percentages. Continuous variables were expressed as mean with standard deviation or median with interquartile range, as appropriate. Normality of continuous variables was assessed using the Shapiro-Wilk test.

Changes in comorbidity status between baseline and at one-year were evaluated using McNemar's test for paired proportions, performed separately within women and men. Sex-based differences in the distribution of trajectory categories were assessed using the chi-square test. Fisher's exact test was applied where expected cell counts were small.

Comparisons of continuous variables between women and men were conducted using the Mann-Whitney U test for non-normally distributed variables. For comparisons of continuous cardiometabolic parameters across more than two trajectory groups, the Kruskal-Wallis test was used, with post hoc pairwise comparisons performed using Dunn's test where the overall test was statistically significant.

All statistical tests were two-tailed, and a p-value < 0.05 was considered statistically significant. Given the exploratory and observational nature of the study, formal corrections for multiple comparisons were not applied. No sensitivity analyses were conducted. Borderline statistically significant p-values should be interpreted with caution.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee (Ref. No. 07/2023/IECG, Dated: 06/04/2023). Written informed consent was obtained from all participants prior to enrolment. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments.

RESULTS

A total of 420 participants [204 women (48.6%) and 216 men (51.4%)] were included (Table 2). Women were slightly younger than men (40.2 ± 7.3 vs 41.5 ± 6.4 years; p = 0.016). Men were more commonly from urban areas (56.0% vs 48.0%), while rural residence was more frequent among women (18.3% vs 9.7%; p = 0.033). Educational attainment was comparable between sexes (p = 0.584), and occupational distribution was similar, with approximately half in sedentary roles. Socioeconomic status differed significantly (p < 0.01), with men more often in the average-income category (69.9%) and women more frequently in the high-income category (34.3%).

No women reported smoking, tobacco use, or alcohol consumption, compared with 21.8%, 11.1%, and 28.2% of men, respectively (all p < 0.01). Men exercised more frequently (42.6% vs 28.4%; p = 0.002) and for longer durations (42.8 ± 17.9 vs 34.9 ± 16.2 minutes; p = 0.011).

Men were taller and heavier, but women had a higher mean BMI (27.4 ± 5.1 vs 25.9 ± 4.1 kg/m^2; p = 0.005) and greater prevalence of obesity (25.5% vs 17.1%; p = 0.036). Abdominal circumference was similar between sexes (p = 0.940).

Sex-Specific Trajectories of Comorbidities

At one year post-CAD, diabetes resolution occurred in 36.7% of the cohort, 23.1% remained consistently non-diabetic, 24.0% developed new-onset diabetes, and 16.2% had persistent diabetes (Table 3). Proportions were broadly similar between sexes. Within-

sex changes were significant in both women (McNemar's $\chi^2 = 5.83$, p = 0.020) and men ($\chi^2 = 5.24$, p = 0.027), though between-sex distribution did not differ significantly ($\chi^2 = 2.532$, p = 0.469).

Table 2: Baseline characteristics of study participants

Characteristic	Women (n = 204)(%)	Men (n = 216) (%)	p-value
Age (years), mean ± SD	40.2 ± 7.3	41.5 ± 6.4	0.016
Residence, n (%)			0.033
Urban	97 (48.0)	121 (56.0)	
Coastal	68 (33.7)	74 (34.3)	
Rural	37 (18.3)	21 (9.7)	
Educational level, n (%)			0.584
≤ SSLC	87 (42.6)	78 (36.1)	
Plus Two	67 (32.8)	79 (36.6)	
≥ Degree	50 (24.5)	59 (27.3)	
Occupational type, n (%)			0.554
Sedentary	106 (52.0)	106 (49.1)	
Non-sedentary	98 (48.0)	110 (50.9)	
Socioeconomic status, n (%)			<0.01
Low	34 (16.7)	28 (13.0)	
Average	100 (49.0)	151 (69.9)	
High	70 (34.3)	37 (17.1)	
Smoking, n (%)	0 (0.0)	47 (21.8)	<0.01
Chewing tobacco, n (%)	0 (0.0)	24 (11.1)	<0.01
Alcohol consumption, n (%)	0 (0.0)	61 (28.2)	<0.01
Regular exercise, n (%)	58 (28.4)	92 (42.6)	0.002
Duration of exercise (minutes), mean ± SD*	34.9 ± 16.2	42.8 ± 17.9	0.011
Height (cm), mean ± SD	152.5 ± 8.5	163.6 ± 9.7	<0.01
Weight (kg), mean ± SD	63.4 ± 11.7	69.5 ± 13.4	<0.01
Body mass index (kg/m^2), mean ± SD	27.4 ± 5.1	25.9 ± 4.1	0.005
Abdominal circumference (cm), mean ± SD	94.1 ± 11.5	94.0 ± 11.4	0.94
Obesity, n (%)	52 (25.5)	37 (17.1)	0.036

Data are presented as mean ± standard deviation (SD) for continuous variables and n (%) for categorical variables. *Duration of exercise calculated only among participants reporting regular exercise (women n = 58, men n = 92). SSLC = Secondary School Leaving Certificate. p-values were calculated using Mann-Whitney U test for continuous variables and chi-square test (or Fisher's exact test where appropriate) for categorical variables.

Table 3: Diabetes status before and after CAD treatment – stratified by sex

Diabetes Status	Female n (%)	Male n (%)	Total n (%)
Resolution	71 (34.8)	83 (38.4)	154 (36.7)
Consistently healthy	52 (25.5)	45 (20.8)	97 (23.1)
New-onset	45 (22.1)	56 (25.9)	101 (24.0)
Persistent	36 (17.6)	32 (14.8)	68 (16.2)
Total	204 (100.0)	216 (100.0)	420 (100.0)
McNemar's test results	$\chi^2 = 5.83$ (p = 0.020)	$\chi^2 = 5.24$ ($p = 0.027$)	

Table 4: Hypertension status before and after CAD treatment – Stratified by sex

Hypertension Status	Female n (%)	Male n (%)	Total n (%)
Resolution	84 (41.2)	81 (37.5)	165 (39.3)
Consistently healthy	46 (22.5)	50 (23.1)	96 (22.9)
New-onset	42 (20.6)	50 (23.1)	92 (21.9)
Persistent	32 (15.7)	35 (16.2)	67 (16.0)
Total	204 (100.0)	216 (100.0)	420 (100.0)
McNemar's test results	$\chi^2 = 14.00$ ($p = 0.0002$)	$\chi^2 = 7.34$ ($p = 0.0085$)	

Table 5: Dyslipidemia status before and after CAD treatment – Stratified by sex

Dyslipidemia Status	Female n (%)	Male n (%)	Total n (%)
Resolution	73 (35.8)	81 (37.5)	154 (36.7)
Consistently healthy	53 (26.0)	63 (29.2)	116 (27.6)
New-onset	39 (19.1)	40 (18.5)	79 (18.8)
Persistent	39 (19.1)	32 (14.8)	71 (16.9)
Total	204 (100.0)	216 (100.0)	420 (100.0)
McNemar's test results	$\chi^2 = 10.32$ ($p = 0.0017$)	$\chi^2 = 13.89$ ($p = 0.0002$)	

For hypertension, 39.3% showed resolution, 22.9% remained normotensive, 21.9% developed new-onset, and 16.0% had persistent hypertension (Table 4). Within-sex changes were significant in women (χ^2 = 14.00, p = 0.0002) and men (χ^2 = 7.34, p = 0.0085), with no significant between-sex difference (χ^2 = 0.710, p = 0.871).

For dyslipidemia, 36.7% demonstrated resolution, 27.6% remained consistently free, 18.8% were new-onset, and 16.9% had persistent dyslipidemia (Table 5). Persistent dyslipidemia was numerically higher among women. Within-sex changes were significant in both women (χ^2 = 10.32, p = 0.0017) and men (χ^2 = 13.89, p = 0.0002), with no significant between-sex difference (χ^2 = 1.638, p = 0.651).

Sociodemographic Associations

Most sociodemographic variables showed no significant associations with comorbidity trajectories. Educational attainment was significantly associated with dyslipidemia progression (p = 0.013); secondary-level educated participants had higher resolution (42.4%) and lower persistent dyslipidemia (13.3%), while those with higher secondary education showed more persistence (21.9%). Sex was not significantly associated with trajectory distribution for any comorbidity.

Environmental and Clinical Factors

High air pollution, environmental toxin exposure, and household pesticide use were each significantly associated with higher rates of new-onset and persistent diabetes (all p < 0.01). No consistent associations were found between environmental exposures and hypertension or dyslipidemia trajectories. Obesity was not significantly associated with any trajectory, though persistent hypertension was numerically higher among obese participants.

Lifestyle Factors

Regular exercise and higher self-reported physical activity were associated with lower persistent diabetes (p < 0.01). Adequate daily water intake was associated with lower persistent hypertension (p = 0.002). Smoking, alcohol, and dietary factors showed no consistent significant associations with any trajectory category.

Discussion

The analysis provides a detailed look at how three critical cardiovascular comorbidities: diabetes, hypertension, and dyslipidemia differed between the time of the CAD event and one year later, with attention to differences between women and men.

Effectiveness of Secondary Prevention: A substantial proportion of patients showed improvement in risk factors identified at baseline. Blood pressure and cholesterol control were particularly notable, with around 40% of hypertensive subjects and 37% of dyslipidemic subjects normalizing their readings after one year. These improvements may partly reflect the effects of medications (antihypertensives and statins), dietary modifications, and cardiac rehabilitation programs, although the observational design precludes attribution to any specific intervention.[17] Approximately one-fourth of the subjects presented without these comorbidities at both baseline and follow-up. This sustained absence of conventional metabolic risk factors in an established CAD cohort suggests the potential involvement of unmeasured risk variables, inherent metabolic resilience, or historical underdiagnosis.[18] However, 15-17% of patients exhibited persistent uncontrolled comorbidities despite guideline-based therapy, warranting investigation in future studies.

High Incidence of New-Onset Comorbidities and Environmental Determinants: Approximately 24% of initially non-diabetic patients developed diabetes, and a comparable percentage became hypertensive during the one-year assessment. This new-onset diabetes rate exceeds global averages of approximately 16% reported in recent meta-analyses[19], possibly reflecting India's genetic susceptibility and dietary patterns[20]. The high proportion of 'new-onset' cases may also partially represent pre-existing undiagnosed diabetes detected during intensive post-CAD screening.[21]

An exploratory finding was the observed association between self-reported environmental exposures and diabetes trajectories. Participants reporting high air pollution, environmental toxins, or pesticide exposure demonstrated higher proportions of new-onset and persistent diabetes. Large epidemiological studies have similarly associated PM2.5 exposure with increased diabetes risk through systemic inflammation[22], and pesticide exposure with glucose dysregulation[23], mechanisms potentially relevant in post-CAD patients. Because exposures were assessed via self-report without objective measurement, confounding cannot be excluded. The high new-onset rates nevertheless highlight the potential value of proactive measures: regular glucose and blood pressure screening, dietary counselling, weight management support, and early pharmacological intervention when indicated.[24]

Sex Differences in Risk Factor Trajectories: Women exhibited higher BMI and obesity prevalence, while men demonstrated substantially higher behavioral risk factors (smoking, alcohol, and tobacco), a pattern consistent with South Asian sociocultural norms.[25] Despite these baseline disparities, comorbidity trajectories over one year were largely comparable between sexes. While consistent with the possibility that systematic guideline-based care attenuates sex-based differences, this could also reflect unmeasured confounders.[26]

However, women showed subtly higher persistent dyslipidemia and lower diabetes resolution rates. These patterns align with literature documenting

that women with CAD often achieve lower LDL-cholesterol goal attainment[27] emphasizing the need for sex-sensitive secondary prevention strategies.

Lifestyle Factors and Comorbidity Trajectories: Regular exercise and higher physical activity levels were associated with substantially lower persistent diabetes rates, consistent with evidence of exercise-associated improvements in glycemic control.[28] Yet fewer than one-third of women and less than half of men reported regular exercise. This represents a crucial intervention target, as exercise benefits extend to direct cardioprotection.[29] Adequate water intake was associated with lower persistent hypertension, likely reflecting broader health-conscious behavior patterns. The educational gradient in dyslipidemia, with higher-educated participants showing greater persistence, suggests that education alone does not guarantee control. While speculative, this pattern may reflect occupational barriers, healthcare navigation complexity, or competing priorities despite greater health literacy[30], though the current data do not permit a definitive explanation.

Public Health and Clinical Implications: The trajectory of these comorbidities post-CAD has potential implications for preventing recurrent events. Patients developing new conditions represent a newly at-risk group who may benefit from systematic follow-up. Future research should evaluate whether integrating routine risk factor surveillance into post-MI care through structured clinics or scheduled monitoring at regular intervals improves long-term outcomes. Educational initiatives emphasizing medication adherence and sustained lifestyle changes also merit further evaluation.

Suboptimal adherence to secondary prevention is prevalent, particularly in resource-limited settings.[31] Interventions, including patient navigators, family support, digital reminders, and financial assistance, may improve sustained adherence. Policymakers should consider the burden of post-CAD chronic conditions and the potential value of preventive programs targeting high-risk subgroups, informed by more robust evidence.

Strengths and Limitations

This study has several limitations. Baseline risk factor status was determined retrospectively using medical records, raising potential recall bias. Patients were enrolled over a three-year period (2022-2025), which may introduce minor temporal heterogeneity in practices. Medication adherence was not objectively measured. The study evaluated one-year survivors, introducing survival bias by excluding patients who died. Categorical trajectory classifications may have reduced statistical power. Finally, the cross-sectional design limits causal inference regarding CAD and subsequent comorbidity development; accordingly, these findings should be considered hypothesis-generating rather than definitive.

Conclusion

This sex-stratified analysis of CAD survivors in Kerala demonstrates dynamic comorbidity patterns one-year post-event. Substantial proportions showed control of baseline diabetes, hypertension, or dyslipidemia, while considerable subsets exhibited new-onset or persistent disease. Patterns were broadly parallel between sexes, though women showed higher persistent dyslipidemia, suggesting closer clinical attention may be warranted for this subgroup.

Exploratory analyses associated self-reported high air pollution, environmental toxins, and pesticide exposure with higher new-onset and persistent diabetes. Regular exercise was associated with lower persistent diabetes, while adequate water intake was associated with lower persistent hypertension.

These findings highlight comprehensive secondary prevention beyond acute event management, encompassing integrated metabolic monitoring, promotion of physical activity, and sex-specific screening particularly dyslipidemia in women. These observational findings suggest environmental and lifestyle factors may warrant consideration in post-CAD metabolic surveillance, though prospective studies with objective measurements are needed to establish causality.

Acknowledgement: The authors gratefully acknowledge the Meenakshi Academy of Higher Education and Research (MAHER - Deemed to be University), West K.K Nagar, Chennai, Tamil Nadu, India and Genetika, Centre for Advanced Genetic Studies, Thiruvananthapuram, Kerala, India, for their valuable support and provision of resources.

Individual Authors' Contributions: APV contributed to conceptualization, data curation, formal analysis, investigation, and preparation of the original draft. **PVC** and **DRD** contributed to conceptualization and provision of resources, while **DRD** also contributed to methodology. **AMS** and **MBM** were involved in data curation and visualization, with **MBM** also contributing to manuscript review and editing. **AW** participated in formal analysis and validation. **JV** contributed to investigation and validation. **STP** contributed to investigation, methodology, administration, supervision, and writing of the original draft. JJS was involved in project administration and validation. SK contributed to resources and supervision.

Availability of Data: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Declaration of Non-use of Generative AI Tools: This article was prepared without the use of generative AI tools for content creation, analysis, or data generation. All findings and interpretations are based solely on the authors' independent work and expertise.

References

1. World Health Organization. Cardiovascular diseases (CVDs) [Internet]. Geneva: World Health Organization; 2025 [cited 2025 Nov]. Available from: https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)
2. Kalra A, Jose AP, Prabhakaran P, Kumar A, Agrawal A, Roy A, et al. The burgeoning cardiovascular disease epidemic in Indians - perspectives on contextual factors and potential solutions. Lancet Reg Health Southeast Asia. 2023 Feb 10;12:100156. DOI: https://doi.org/10.1016/j.lansea.2023.100156 PMid:37384064 PMCid:PMC10305862
3. Krishnan MN, Zachariah G, Venugopal K, Mohanan PP, Harikrishnan S, Sanjay G, et al. Prevalence of coronary artery disease and its risk factors in Kerala, South India: a community-based cross-sectional study. BMC Cardiovasc Disord. 2016 Jan 14;16:12. DOI: https://doi.org/10.1186/s12872-016-0189-3 PMid:26769341 PMCid:PMC4712491
4. Sharma AK, Dar MI, Iqbal M, Tramboo NA. Gender based differences in coronary artery disease: a prospective observational study from a North Indian state. Heart India. 2020;8(2):85-92. DOI: https://doi.org/10.4103/heartindia.heartindia_13_20
5. Manfrini O, Yoon J, van der Schaar M, Kedev S, Vavlukis M, et al. Sex Differences in Modifiable Risk Factors and Severity of Coronary Artery Disease. J Am Heart Assoc. 2020 Oct 20;9(19):e017235. DOI: https://doi.org/10.1161/JAHA.120.017235 PMid:32981423 PMCid:PMC7792418
6. Yeo JL, Dattani A, Bilak JM, Wood AL, Athithan L, Deshpande A, et al. Sex differences and determinants of coronary microvascular function in asymptomatic adults with type 2 diabetes. J Cardiovasc Magn Reson. 2025 Summer;27(1):101132. DOI: https://doi.org/10.1016/j.jocmr.2024.101132 PMid:39647765 PMCid:PMC11761338
7. Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021 Sep 7;42(34):3227-3337. doi: 10.1093/eurheartj/ehab484. Erratum in: Eur Heart J. 2022 Nov 7;43(42):4468. DOI: https://doi.org/10.1093/eurheartj/ehab484 PMid:34458905
8. Laranjo L, Lanas F, Sun MC, Chen DA, Hynes L, Imran TF, et al. World Heart Federation Roadmap for Secondary Prevention of Cardiovascular Disease: 2023 Update. Glob Heart. 2024 Jan 22;19(1):8. DOI: https://doi.org/10.5334/gh.1278 PMid:38273995 PMCid:PMC10809857
9. Arsyad DS, Qalby N, Qanitha A, Mappangara I, Milayanti W, Wahiduddin, et al. Low Attainment of Treatment Targets for Cardiovascular Risk Factors in Indonesian Adults With Established Coronary Artery Disease. J Eval Clin Pract. 2025 Feb;31(1):e14311. DOI: https://doi.org/10.1111/jep.14311 PMid:39803729 PMCid:PMC11726404
10. Indian Council of Medical Research. ICMR Guidelines for Management of Type 2 Diabetes [Internet]. New Delhi: ICMR; 2018. [cited 2025 Nov 10]. Available from: https://www.icmr.gov.in/icmrobject/custom_data/pdf/resource-guidelines/ICMR_GuidelinesType2diabetes2018_0.pdf
11. Ministry of Health and Family Welfare. Standard Treatment Guidelines: Hypertension [Internet]. New Delhi: Government of India; 2016. [cited 2025 Nov 10]. Available from: https://nhm.gov.in/images/pdf/guidelines/nrhm-guidelines/stg/Hypertension_full.pdf
12. Sawhney JP, Ramakrishnan S, Madan K, Ray S, Jayagopal PB, Prabhakaran D, et al. CSI clinical practice guidelines for dyslipidemia management: Executive summary. Indian Heart J. 2024 Mar;76 Suppl 1(Suppl 1):S6-S19. DOI: https://doi.org/10.1016/j.ihj.2023.11.271. PMID: 38052658; PMCID: PMC11019331
13. Parigela P, Manohar B, Reddy BKK, Tirumala NS, Sanakhan PN, Kothakota SK, Punugupati M. A study on patterns of dyslipidaemia in patients with acute coronary syndrome in India - The Study patterns Dyslipedemia (STED)-Coronary Artery Disease (CAD) study. J Pract Cardiovasc Sci. 2025;11(2):139-144. DOI: https://doi.org/10.4103/jpcs.jpcs_66_24
14. Borgharkar SS, Das SS. Low-density lipoprotein cholesterol levels among individuals receiving statin therapy: real-world evidence from India. Am J Health Res. 2021;9(1):13-18. DOI: https://doi.org/10.11648/j.ajhr.20210901.13
15. Majumder S. Socioeconomic status scales: Revised Kuppuswamy, BG Prasad, and Udai Pareekh's scale updated for 2021. J Family Med Prim Care. 2021;10(11):3964-3967. DOI: https://doi.org/10.4103/jfmpc.jfmpc_600_21 PMid:35136753 PMCid:PMC8797108
16. Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003 Aug;35(8):1381-1395. DOI: https://doi.org/10.1249/01.MSS.0000078924.61453.FB PMid:12900694
17. Sigamani A, Gupta R. Revisiting secondary prevention in coronary heart disease. Indian Heart J. 2022 Nov-Dec;74(6):431-440. DOI: https://doi.org/10.1016/j.ihj.2022.11.011 PMid:36455667 PMCid:PMC9773289
18. Mazhar J, Figtree G, Vernon ST, Galougahi KK, Carlo J, Nissen SE, Nicholls SJ. Progression of coronary atherosclerosis in patients without standard modifiable risk factors. Am J Prev Cardiol. 2020 Nov 24;4:100116. DOI: https://doi.org/10.1016/j.ajpc.2020.100116 PMid:34327476 PMCid:PMC8315424
19. Douelrachad M, Thistle A, Rijal H, Ochuba A, Ke C, Lee C, Vyas MV. New diagnosis of diabetes in patients with myocardial infarction or stroke: a systematic review and meta-analysis. Eur J Prev Cardiol. 2026 Jan 6;33(1):19-29. DOI: https://doi.org/10.1093/eurjpc/zwaf286 PMid:40359383
20. Muniyappa R, Narayanappa SBK. Disentangling Dual Threats: Premature Coronary Artery Disease and Early-Onset Type 2 Diabetes Mellitus in South Asians. J Endocr Soc. 2023 Dec 26;8(1):bvad167. DOI: https://doi.org/10.1210/jendso/bvad167 PMid:38178904 PMCid:PMC10765382
21. Ostrominski JW, Vaduganathan M, Girish MP, Gupta P, Hendrickson MJ, Qamar A, et al. Missed Opportunities for Screening and Management of Dysglycemia among Patients Presenting with Acute Myocardial Infarction in North India: The Prospective NORIN STEMI Registry. Glob Heart. 2022 Aug 12;17(1):54. DOI: https://doi.org/10.5334/gh.1140 PMid:36051328 PMCid:PMC9374010
22. GBD 2019 Diabetes and Air Pollution Collaborators. Estimates, trends, and drivers of the global burden of type 2 diabetes attributable to PM2·5 air pollution, 1990-2019: an analysis of data from the Global Burden of Disease Study 2019. Lancet Planet Health. 2022 Jul;6(7):e586-e600. DOI: https://doi.org/10.1016/S2542-5196(22)00122-X PMid:35809588 PMCid:PMC9278144
23. Chung YL, Hou YC, Wang IK, Lu KC, Yen TH. Organophosphate pesticides and new-onset diabetes mellitus: From molecular mechanisms to a possible therapeutic perspective. World J Diabetes. 2021 Nov 15;12(11):1818-1831. DOI: https://doi.org/10.4239/wjd.v12.i11.1818 PMid:34888010 PMCid:PMC8613664
24. Kerkar P, Harikrishnan S, Sawhney JPS, Kapoor A, Gharat C. Home-based care post-acute coronary syndrome: A comprehensive review. Heart India. 2025;13(2):82-88. DOI: https://doi.org/10.4103/heartindia.heartindia_84_24
25. Sundaresan M, Velmurugan G, Dhivakar M, Ramakrishnan A, Cherian M, Alexander T, Swaminathan K. Gender Differential Prevalence of Overweight and Obesity, Hypertension and Diabetes in South India: A Population-Based Cross-Sectional

Study. Glob Heart. 2024 Sep 9;19(1):72. DOI: https://doi.org/10.5334/gh.1354 PMid:39281001 PMCid:PMC11396087

26. Virani SS, Alonso A, Aparicio HJ, Benjamin EJ, Bittencourt MS, Callaway CW, et al. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. Circulation. 2021 Feb 23;143(8):e254-e743. DOI: https://doi.org/10.1161/CIR.0000000000000950 PMid:33501848

27. Sedrakyan S, Nadeem B, Gaughran D, Sinauridze A, Georgy A, Sally H, Lam U. Bridging Gaps in LDL-C Management: Utilization of Lipid-Lowering Therapies, Gender Disparities, and the Impact of Cardiology Visits on Secondary Prevention. Am J Cardiol. 2025 Oct 15;253:1-9. DOI: https://doi.org/10.1016/j.amjcard.2025.05.029 PMid:40466966

28. Gallardo-Gómez D, Salazar-Martínez E, Alfonso-Rosa RM, Ramos-Munell J, Del Pozo-Cruz J, Del Pozo Cruz B, Álvarez-Barbosa F. Optimal Dose and Type of Physical Activity to Improve Glycemic Control in People Diagnosed With Type 2 Diabetes: A Systematic Review and Meta-analysis. Diabetes Care. 2024 Feb 1;47(2):295-303. DOI: https://doi.org/10.2337/dc23-0800 PMid:38241499

29. Quindry JC, Michalak RE. Exercise-induced cardioprotection: From endogenous to exogenous mechanisms. Sports Med Health Sci. 2025 Mar 28;7(5):366-374. DOI: https://doi.org/10.1016/j.smhs.2025.03.009 PMid:40936660 PMCid:PMC12421174

30. Marzà-Florensa A, Vaartjes I, Graham I, Klipstein-Grobusch K, Grobbee DE; Surf CHD II collaborators. A Global Perspective on Cardiovascular Risk Factors by Educational Level in CHD Patients: SURF CHD II. Glob Heart. 2024 Jul 16;19(1):60. DOI: https://doi.org/10.5334/gh.1340 PMid:39035775 PMCid:PMC11259115

31. Mohandas NV, Krishnapillai V, Sreedevi A, George N, Dinesh A, Mohandas V, Menon JC. Adherence to Secondary Prevention Strategies Among Adults with Coronary Artery Disease in Rural Aluva, South India: A Community-Based Cross-Sectional Study. Indian J Community Med. 2025 Nov-Dec;50(6):1021-1027. DOI: https://doi.org/10.4103/ijcm.ijcm_223_24 PMid:41451041 PMCid:PMC12735374