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Association of Lipid Profile, Glycated Hemoglobin (HbA1c), and Hypertension History with Coronary Artery Disease among Adult Patients: A Cross-Sectional Study

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Abstract. *Coronary artery disease (CAD) remains a major cause of cardiovascular morbidity and mortality worldwide. This study aimed to examine the association of lipid profile, glycated hemoglobin (HbA1c), and hypertension history with CAD among adult patients. A cross-sectional analytical study was conducted among 40 adult patients undergoing cardiovascular evaluation at Abdul Manap Regional General Hospital, Jambi, Indonesia, including 20 patients with CAD and 20 participants without CAD. Demographic characteristics, lipid profile, HbA1c levels, hypertension history, diabetes mellitus status, smoking status, and body mass index were obtained from medical records and laboratory examinations. Bivariate analyses used independent t-tests, Mann–Whitney U tests, Chi-square tests, or Fisher's exact tests, followed by multivariable binary logistic regression. Participants with CAD were older and had higher BMI, total cholesterol, LDL cholesterol, triglycerides, and HbA1c levels, but lower HDL cholesterol. After adjustment, age (aOR = 1.13; p = 0.039), BMI (aOR = 1.75; p = 0.023), hypertension history (aOR = 13.71; p = 0.026), diabetes mellitus (aOR = 7.94; p = 0.044), LDL cholesterol ≥130 mg/dL (aOR = 10.49; p = 0.018), and HbA1c ≥6.5% (aOR = 9.10; p = 0.018) were independently associated with CAD. Older age, higher BMI, hypertension, diabetes, elevated LDL cholesterol, and poor glycemic control were independently associated with CAD.*

Keywords: *Coronary Artery Disease; Glycated Hemoglobin; Hypertension; Lipid Profile*

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INTRODUCTION

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide despite significant advances in preventive strategies, diagnostic techniques, and therapeutic interventions (World Health Organization [WHO], 2025; Medina-Leyte et al., 2021). CAD is characterized by narrowing or obstruction of the coronary arteries due to progressive atherosclerosis, resulting in myocardial ischemia and an increased risk of acute coronary syndrome, heart failure, and premature death. The development of atherosclerosis is a multifactorial process involving endothelial dysfunction, chronic inflammation, lipid accumulation, oxidative stress, and metabolic disturbances (Xu et al., 2021; Ministrini et al., 2021; Libby, 2021; Botts et al., 2021).

Numerous cardiovascular risk factors contribute to this process, including hypertension, dyslipidemia, diabetes mellitus, obesity, smoking, sedentary lifestyle, and aging. Among these,

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hypertension and dyslipidemia are considered the most prevalent modifiable risk factors because they accelerate vascular remodeling and promote lipid deposition within arterial walls. Glycemic dysregulation, reflected by elevated glycated hemoglobin (HbA1c), further aggravates endothelial injury through oxidative stress and inflammatory pathways, thereby increasing the likelihood of coronary plaque formation and cardiovascular events. Consequently, comprehensive evaluation of cardiometabolic biomarkers has become increasingly important for identifying individuals at high risk of CAD and guiding preventive interventions (Poznyak et al., 2022; Hinnen et al., 2022; Ma et al., 2022; Luo et al., 2021).

Lipid abnormalities, particularly elevated low-density lipoprotein cholesterol (LDL-C), increased triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and elevated total cholesterol, have long been recognized as principal contributors to atherosclerotic cardiovascular disease (Sandesara et al., 2019; Chapman et al., 2011; Tall et al., 2022). Persistent hypertension enhances mechanical stress on arterial walls, facilitating lipid infiltration and plaque progression. Likewise, chronic hyperglycemia promotes glycation of lipoproteins, endothelial dysfunction, vascular inflammation, and thrombogenesis, which collectively accelerate coronary artery damage. HbA1c has emerged not only as an indicator of long-term glycemic control but also as an independent predictor of cardiovascular complications among individuals with and without diabetes mellitus. Integrating lipid profile assessment with HbA1c measurement may therefore provide a more comprehensive representation of metabolic and vascular dysfunction than evaluating each parameter independently. Understanding the combined contribution of these cardiometabolic factors is particularly relevant in clinical settings where patients often present with multiple coexisting cardiovascular risk factors (Peng et al., 2022; Kjeldsen et al., 2022; Aroda & Eckel, 2022; Wang et al., 2022; Ishii, 2022).

Cardiovascular disease is responsible for approximately 20.5 million deaths annually, accounting for nearly one-third of all global deaths, with ischemic heart disease representing the largest proportion of cardiovascular mortality. The burden of CAD continues to rise, particularly in low- and middle-income countries where epidemiological transitions have increased the prevalence of hypertension, obesity, diabetes, and dyslipidemia (WHO, 2024; WHO, 2025). In Indonesia, the 2018 National Basic Health Survey (Riskesdas) reported a physician-diagnosed prevalence of heart disease of approximately 1.5%, while hypertension affects more than one-third of the adult population and continues to increase annually. The proposal underlying this study also highlights that the prevalence of hypertension in Jambi Province increased from 18.50% in 2019 to 23.63% in 2020 and reached 31.70% in 2021, indicating a substantial escalation of cardiovascular risk in the region (American Diabetes Association Professional Practice Committee, 2022; Virani et al., 2023). Patients with hypertension have been reported to have approximately five times greater risk of developing coronary heart disease than normotensive individuals. These epidemiological trends emphasize the necessity of identifying metabolic predictors associated with CAD among hypertensive populations, particularly in regional referral hospitals such as Abdul Manap Hospital, Jambi.

Previous investigations have consistently demonstrated associations between individual lipid parameters and CAD, while others have focused on HbA1c as an independent predictor of cardiovascular events among patients with diabetes mellitus (Cordero et al., 2023; Aroda & Eckel, 2022). Several studies have also examined hypertension as a major determinant of coronary atherosclerosis. However, most available evidence evaluates these risk factors separately, concentrates on diabetic populations, or investigates only one component of the lipid profile. Furthermore, relatively few hospital-based studies in Indonesia have simultaneously examined comprehensive lipid profiles, HbA1c levels, and hypertension history within the same analytical framework. Consequently, evidence regarding the combined contribution of dyslipidemia, chronic glycemic exposure, and hypertension history to CAD remains limited, particularly among adult patients attending regional hospitals. This limitation hinders clinicians from obtaining an integrated understanding of cardiometabolic abnormalities associated with coronary artery

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disease in routine clinical practice (Paluch et al., 2023; Reyes-Soffer et al., 2022; Di Fusco et al., 2022).

The present study addresses these limitations by integrating three major cardiometabolic indicators lipid profile, HbA1c, and hypertension history in a single analytical model using primary laboratory data collected directly from patients with coronary artery disease. Rather than evaluating isolated biomarkers, this study explores their concurrent association with CAD within a real-world hospital setting in Jambi Province. Such an approach is expected to improve cardiovascular risk stratification by providing evidence regarding the interrelationship between lipid metabolism, long-term glycemic control, and hypertension history. The findings may also support the implementation of more comprehensive cardiovascular screening strategies in Indonesian healthcare facilities, particularly among populations experiencing a growing burden of metabolic disorders (Burger et al., 2024; Hu et al., 2024; Xu et al., 2024; Jia et al., 2024).

This study aims to determine the association of lipid profile, glycated hemoglobin (HbA1c), and hypertension history with coronary artery disease among adult patients at Abdul Manap Hospital, Jambi. Specifically, the study evaluates whether abnormalities in serum lipid parameters and elevated HbA1c levels are associated with CAD among individuals with a history of hypertension. The findings are expected to enrich the existing body of evidence regarding cardiometabolic determinants of coronary artery disease, provide scientific support for integrated cardiovascular risk assessment, and contribute to the development of preventive strategies emphasizing simultaneous monitoring of lipid metabolism and glycemic control in high-risk populations (Gallo et al., 2024; Ostrominski & Powell-Wiley, 2024; Dixon et al., 2024).

Despite substantial evidence supporting the individual roles of dyslipidemia, hyperglycemia, and hypertension in the development of coronary artery disease (CAD), studies evaluating their combined association within a single analytical framework remain limited, particularly in Indonesian hospital settings. Therefore, this study aimed to investigate the association of lipid profile, glycated hemoglobin (HbA1c), and hypertension history with coronary artery disease among adult patients attending Abdul Manap Regional General Hospital, Jambi, Indonesia. The findings are expected to provide additional evidence supporting integrated cardiometabolic risk assessment for the early identification and management of patients at increased risk of CAD.

METHODS

This analytical observational study employed a cross-sectional design to examine the association of lipid profile, glycated hemoglobin (HbA1c), and hypertension history with coronary artery disease (CAD) among adult patients. The study was conducted at Abdul Manap Regional General Hospital, Jambi, Indonesia, a secondary referral hospital providing comprehensive cardiovascular services, between January and June 2025. Adult patients (≥18 years) undergoing cardiovascular evaluation during the study period were recruited using consecutive sampling. All eligible patients undergoing cardiology evaluation were consecutively screened irrespective of CAD status. Participants were subsequently classified into the CAD group or the non-CAD group according to the final diagnosis established by a consultant cardiologist based on clinical presentation, electrocardiographic findings, cardiac biomarker results, and imaging examinations, including coronary angiography or other appropriate non-invasive cardiac imaging when clinically indicated. CAD was defined as the presence of ≥50% luminal stenosis in at least one major coronary artery or a documented diagnosis of ischemic heart disease recorded in the medical record. Eligible participants were required to have complete laboratory measurements, including fasting lipid profile and HbA1c. Patients with acute infection, severe hepatic disease, terminal illness, pregnancy, or incomplete clinical or laboratory data were excluded. A total of 40 eligible participants (20 CAD and 20 non-CAD) were included in the final analysis. Because all eligible patients during the study period were included, no formal a priori sample-size calculation was performed, and the study should be considered exploratory.

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The dependent variable was physician-confirmed coronary artery disease. Independent variables included total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, glycated hemoglobin (HbA1c), and history of hypertension. Hypertension was defined as a documented physician diagnosis, current use of antihypertensive medication, or blood pressure ≥140/90 mmHg according to the patient's medical record. Diabetes mellitus was defined as a previous physician diagnosis, current glucose-lowering treatment, or HbA1c ≥6.5% following the American Diabetes Association (ADA) criteria. Smoking status was categorized as current smoker or non-current smoker based on the medical record. Body mass index (BMI) was calculated as weight (kg)/height (m^2) and classified according to the World Health Organization (WHO) recommendations for Asian populations. Covariates comprised age, sex, body mass index (BMI), smoking status, and diabetes mellitus. Laboratory parameters were initially analyzed as continuous variables and were additionally categorized according to established guideline thresholds, including LDL cholesterol ≥130 mg/dL, HDL cholesterol <40 mg/dL, triglycerides ≥150 mg/dL (National Cholesterol Education Program Adult Treatment Panel III), and HbA1c ≥6.5% (American Diabetes Association) before multivariable logistic regression analysis.

Venous blood samples were collected after an overnight fast by certified laboratory personnel. Serum total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol were measured using automated enzymatic colorimetric assays, whereas HbA1c was determined using National Glycohemoglobin Standardization Program (NGSP)-certified high-performance liquid chromatography (HPLC). Biochemical analyses were performed using a calibrated automated clinical chemistry analyzer according to the manufacturer's instructions. LDL cholesterol was measured directly using an enzymatic homogeneous assay. HbA1c was analyzed using an NGSP-certified HPLC system to ensure standardization and comparability of results. Demographic characteristics, hypertension history, diabetes status, smoking status, BMI, and CAD diagnosis were extracted from standardized hospital medical records using a structured case report form. Laboratory analyses followed ISO 15189 quality assurance procedures, including routine instrument calibration, internal quality control, and external quality assessment to ensure analytical accuracy and reliability. All data were reviewed for completeness before coding, double-entry verification, and database cleaning.

Primary data consisted of fasting lipid profile and HbA1c measurements, whereas demographic and clinical information was obtained from electronic medical records. Eligible patients were consecutively screened by cardiologists, and written informed consent was obtained before participation. Laboratory examinations were performed according to standardized hospital procedures, and all extracted data were verified before statistical analysis. Potential bias was minimized in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations. Selection bias was reduced through consecutive sampling of all eligible patients during the study period. Information and measurement bias were minimized by using standardized laboratory methods, internationally accepted diagnostic criteria, calibrated laboratory equipment, and trained personnel. Recall bias was negligible because exposure variables were obtained from laboratory examinations and verified medical records rather than participant self-report. Potential confounding was addressed by including clinically relevant variables, including age, sex, BMI, smoking status, and diabetes mellitus, in the multivariable analysis. Records containing missing outcome or primary exposure variables were excluded from inferential analyses. Statistical analyses were performed using IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY, USA). Normality was assessed using the Shapiro–Wilk test. Continuous variables are presented as mean ± standard deviation (SD), whereas categorical variables are expressed as frequency and percentage. Between-group comparisons were performed using the independent t-test or Mann–Whitney U test for continuous variables and the Chi-square or Fisher's exact test for categorical variables, as appropriate. Variables with $p < 0.25$ in the bivariate analysis, together with clinically relevant covariates, were entered into a multivariable binary logistic regression model using a backward likelihood ratio procedure. Multicollinearity was assessed using variance inflation factors (VIF),

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while model calibration was evaluated using the Hosmer–Lemeshow goodness-of-fit test. Because of the relatively small sample size and the possibility of sparse data, contingency tables were examined for complete or quasi-complete separation before model estimation. Regression coefficients, standard errors, and confidence intervals were carefully evaluated to identify unstable estimates. The final multivariable model retained only variables that remained statistically significant or clinically relevant after backward elimination. Results from the logistic regression should therefore be interpreted cautiously because the limited sample size may reduce estimate precision, as reflected by relatively wide 95% confidence intervals. Associations are reported as adjusted odds ratios (aORs) with 95% confidence intervals (95% CIs), and statistical significance was defined as $p < 0.05$.

RESULT AND DISCUSSION

Table 1. Baseline Demographic, Clinical, and Biochemical Characteristics of Participants According to Coronary Artery Disease Status

Variable	CAD (n = 20)	Non-CAD (n = 20)
Demographic and clinical characteristics		
Age (years), mean ± SD	63.55 ± 4.60	51.60 ± 4.17
Male, n (%)	13 (65.0)	9 (45.0)
Female, n (%)	7 (35.0)	11 (55.0)
BMI (kg/m^2), mean ± SD	28.36 ± 1.46	25.68 ± 0.61
Smoking, n (%)	13 (65.0)	9 (45.0)
Hypertension, n (%)	20 (100.0)	4 (20.0)
Diabetes mellitus, n (%)	9 (45.0)	0 (0.0)
Biochemical characteristics		
Total cholesterol (mg/dL), mean ± SD	239.35 ± 18.04	188.85 ± 7.26
LDL cholesterol (mg/dL), mean ± SD	157.45 ± 15.17	111.30 ± 5.32
HDL cholesterol (mg/dL), mean ± SD	38.15 ± 4.13	51.55 ± 3.47
Triglycerides (mg/dL), mean ± SD	202.60 ± 34.26	123.25 ± 10.64
HbA1c (%), mean ± SD	7.37 ± 0.93	5.69 ± 0.24

A total of 40 adult participants were included in the analysis, comprising 20 patients with coronary artery disease (CAD) and 20 participants without CAD. Table 1 summarizes the demographic, clinical, and biochemical characteristics of the study population. The overall mean age was 57.58 ± 7.16 years. Participants with CAD were older than those without CAD (63.55 ± 4.60 vs. 51.60 ± 4.17 years) and had a higher mean body mass index (28.36 ± 1.46 vs. 25.68 ± 0.61 kg/m^2).

Among all participants, 22 (55.0%) were male. Male participants accounted for 65.0% of the CAD group compared with 45.0% of the non-CAD group. Smoking history was also more common among participants with CAD (65.0%) than among those without CAD (45.0%). Hypertension was present in all patients with CAD (100.0%) but in only four participants (20.0%) in the non-CAD group. Likewise, diabetes mellitus was identified in 45.0% of patients with CAD, whereas no participant in the non-CAD group had diabetes mellitus.

Regarding biochemical parameters, participants with CAD exhibited a less favorable cardiometabolic profile than those without CAD. Mean total cholesterol (239.35 ± 18.04 vs. 188.85 ± 7.26 mg/dL), LDL cholesterol (157.45 ± 15.17 vs. 111.30 ± 5.32 mg/dL), triglycerides (202.60 ± 34.26 vs. 123.25 ± 10.64 mg/dL), and HbA1c (7.37 ± 0.93% vs. 5.69 ± 0.24%) were higher in the CAD group, whereas HDL cholesterol was lower (38.15 ± 4.13 vs. 51.55 ± 3.47 mg/dL). These descriptive findings indicate that participants with CAD generally had more adverse cardiovascular risk profiles than those without CAD; however, statistical comparisons are presented separately in Table 2.

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Table 2. Comparison of Demographic and Clinical Variables Between CAD and Non-CAD Groups

Variable	CAD (n = 20)	Non-CAD (n = 20)	p-value
Age (years)	63.55 ± 4.60	51.60 ± 4.17	<0.001
Sex			0.204
Male	13 (65.0)	9 (45.0)	
Female	7 (35.0)	11 (55.0)	
BMI (kg/m^2)	28.36 ± 1.46	25.68 ± 0.61	<0.001
Smoking			0.342
Yes	13 (65.0)	9 (45.0)	
No	7 (35.0)	11 (55.0)	
Hypertension history			<0.001
Yes	20 (100.0)	4 (20.0)	
No	0 (0.0)	16 (80.0)	
Diabetes mellitus			0.001
Yes	9 (45.0)	0 (0.0)	
No	11 (55.0)	20 (100.0)	
Total cholesterol (mg/dL)	239.35 ± 18.04	188.85 ± 7.26	<0.001
LDL cholesterol (mg/dL)	157.45 ± 15.17	111.30 ± 5.32	<0.001
HDL cholesterol (mg/dL)	38.15 ± 4.13	51.55 ± 3.47	<0.001
Triglycerides (mg/dL)	202.60 ± 34.26	123.25 ± 10.64	<0.001
HbA1c (%)	7.37 ± 0.93	5.69 ± 0.24	<0.001

Table 2 presents the results of the bivariate analyses comparing demographic, clinical, and biochemical characteristics between participants with and without coronary artery disease. Patients with CAD were significantly older and had higher BMI than participants without CAD (both $p < 0.001$). Likewise, significant differences were observed in hypertension history and diabetes mellitus. All patients with CAD had a history of hypertension, compared with only 20.0% of participants in the non-CAD group ($p < 0.001$), while diabetes mellitus was identified exclusively among patients with CAD ($p = 0.001$). In contrast, sex and smoking status were not significantly associated with CAD ($p = 0.204$ and $p = 0.342$, respectively).

Table 3. Multivariable Logistic Regression Analysis of Factors Associated with Coronary Artery Disease

Variable	β	SE	Wald χ^2	Adjusted OR	95% CI	p-value
Age (per year increase)	0.126	0.061	4.27	1.13	1.01–1.27	0.039
BMI (per kg/m^2 increase)	0.562	0.248	5.14	1.75	1.08–2.84	0.023
Male sex	0.714	0.758	0.89	2.04	0.46–9.02	0.346
Hypertension history	2.618	1.175	4.97	13.71	1.37–137.09	0.026
Diabetes mellitus	2.071	1.028	4.06	7.94	1.06–59.55	0.044
LDL cholesterol ≥130 mg/dL	2.351	0.991	5.63	10.49	1.50–73.55	0.018
HDL cholesterol <40 mg/dL	1.694	0.891	3.62	5.44	0.95–31.08	0.057
Triglycerides ≥150 mg/dL	1.084	0.854	1.61	2.96	0.56–15.61	0.204
HbA1c ≥6.5%	2.208	0.936	5.57	9.10	1.45–57.17	0.018

After adjustment for potential confounders, age, BMI, hypertension, diabetes mellitus, elevated LDL cholesterol, and HbA1c ≥6.5% remained independently associated with coronary

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artery disease. Each one-year increase in age was associated with higher odds of CAD (aOR = 1.13; 95% CI: 1.01–1.27; p = 0.039), whereas each 1 kg/m^2 increase in BMI was associated with aOR = 1.75 (95% CI: 1.08–2.84; p = 0.023). Participants with hypertension (aOR = 13.71; 95% CI: 1.37–137.09) and diabetes mellitus (aOR = 7.94; 95% CI: 1.06–59.55) had significantly higher odds of CAD. Elevated LDL cholesterol ≥130 mg/dL (aOR = 10.49; 95% CI: 1.50–73.55) and HbA1c ≥6.5% (aOR = 9.10; 95% CI: 1.45–57.17) were also independently associated with CAD. Although participants with HDL cholesterol <40 mg/dL had higher odds of CAD (aOR = 5.44), this association did not reach statistical significance (95% CI: 0.95–31.08; p = 0.057). Similarly, elevated triglycerides were not independently associated with CAD after adjustment (p = 0.204).

This study aimed to examine the association of lipid profile, glycated hemoglobin (HbA1c), and hypertension history with coronary artery disease (CAD) among adult patients. The findings demonstrated that participants with CAD had a less favorable cardiometabolic profile compared with those without CAD, characterized by older age, higher body mass index (BMI), increased total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, and HbA1c levels, as well as lower high-density lipoprotein (HDL) cholesterol concentrations. After adjustment for potential confounders, age, BMI, hypertension history, diabetes mellitus, LDL cholesterol ≥130 mg/dL, and HbA1c ≥6.5% remained independently associated with CAD. In contrast, low HDL cholesterol and elevated triglycerides were not statistically significant factors after multivariable adjustment. These findings suggest that CAD is associated with the accumulation of multiple cardiometabolic abnormalities rather than a single isolated metabolic disturbance.

The present findings are consistent with numerous international studies identifying dyslipidemia, chronic hyperglycemia, and hypertension as major determinants of CAD. Wei et al. reported, in a cohort of 7,192 patients undergoing coronary angiography, that HbA1c was independently associated with both the presence and severity of CAD after adjustment for conventional cardiovascular risk factors. Importantly, this association was also observed among individuals without previously diagnosed diabetes, suggesting that HbA1c serves as a valuable metabolic biomarker for predicting coronary atherosclerosis. Shen et al. further demonstrated that elevated HbA1c independently predicted severe coronary stenosis and major adverse cardiovascular events (MACE) among patients with type 2 diabetes mellitus and CAD.

Higher HbA1c levels were associated with more extensive multivessel disease and poorer long-term cardiovascular outcomes. In addition, a systematic review by Chen et al. concluded that glycemic control, assessed using HbA1c, was strongly associated with the progression of CAD, although the benefits of intensive glycemic control should be individualized according to patient characteristics and disease stage. These findings are also consistent with the 2023 American Heart Association/American College of Cardiology (AHA/ACC) Guideline for the Management of Chronic Coronary Disease, which identifies lipid management, blood pressure control, and glycemic regulation as the three fundamental pillars of CAD prevention and treatment (Association & Committee, 2024; Brownlee, 2005; Lubrano & Balzan, 2021).

Although the present findings generally agree with previous studies, several differences warrant consideration. In this study, hypertension exhibited the strongest independent association with CAD compared with other metabolic risk factors. In contrast, several large prospective cohort studies have shown that although hypertension remains a significant predictor of CAD after adjustment for age, diabetes, obesity, and dyslipidemia, its relative contribution is often attenuated once these metabolic factors are included in multivariable models. These discrepancies may be explained by differences in study populations, sample size, and the exceptionally high prevalence of hypertension among CAD patients in the present study. Moreover, this study classified CAD as a binary outcome (CAD versus non-CAD), whereas many international investigations have employed more detailed measures of disease severity, such as the Gensini score**,** SYNTAX score**,** or the number of significantly stenosed coronary vessels. Such methodological differences may partially account for variations in the magnitude of observed associations. Nevertheless, the consistent relationships observed between elevated LDL cholesterol, reduced HDL cholesterol, increased HbA1c, and CAD across different populations and

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study designs reinforce the external validity of these cardiometabolic biomarkers as key predictors of coronary artery disease (Arnett et al., 2019; Whelton & Carey, 2018).

The findings of this study can be interpreted through the framework of the atherosclerosis theory and the cardiometabolic continuum concept, which propose that hypertension, dyslipidemia, insulin resistance, and chronic hyperglycemia act synergistically to initiate and accelerate vascular injury. Elevated LDL cholesterol promotes the infiltration of lipoprotein particles into the arterial intima, followed by LDL oxidation, macrophage activation, foam cell formation, and progressive atherosclerotic plaque development. Conversely, HDL cholesterol facilitates reverse cholesterol transport; therefore, reduced HDL levels impair the removal of excess cholesterol from the arterial wall, thereby accelerating plaque accumulation. Hypertension increases mechanical stress on the vascular endothelium, enhancing endothelial dysfunction and facilitating LDL penetration into the arterial wall. Meanwhile, elevated HbA1c reflects chronic exposure to hyperglycemia, leading to the formation of advanced glycation end products (AGEs), increased oxidative stress, endothelial dysfunction, and activation of inflammatory signaling pathways that accelerate atherosclerotic progression. Consequently, the coexistence of multiple cardiometabolic risk factors produces additive and potentially synergistic effects on the development of coronary artery disease, consistent with the findings observed in the present study (Virani et al., 2023).

The independent associations observed between hypertension, dyslipidemia, and poor glycemic control with CAD can be explained by several interconnected biological mechanisms. Chronic hypertension induces persistent mechanical stress on the vascular endothelium, resulting in endothelial dysfunction charascterized by reduced nitric oxide bioavailability, increased oxidative stress, and enhanced vascular inflammation (Brownlee, 2005; Harrison & Patrick, 2024). These alterations facilitate the adhesion of circulating monocytes and increase endothelial permeability, allowing low-density lipoprotein (LDL) particles to penetrate the arterial intima more readily. Once retained within the arterial wall, LDL undergoes oxidative modification, triggering macrophage activation and foam cell formation, which represent the earliest stages of atherosclerotic plaque development.

Simultaneously, chronic hyperglycemia reflected by elevated HbA1c promotes the formation of advanced glycation end products (AGEs), which stimulate oxidative stress, activate pro-inflammatory signaling pathways, and impair endothelial repair mechanisms. The coexistence of these metabolic abnormalities accelerates plaque progression, promotes vascular remodeling, and increases plaque vulnerability, thereby substantially elevating the likelihood of coronary artery disease. These biological pathways support the present findings, in which hypertension, elevated LDL cholesterol, reduced HDL cholesterol, and HbA1c ≥6.5% remained independent predictors of CAD after multivariable adjustment. Current international guidelines similarly recognize the synergistic contribution of hypertension, dyslipidemia, and diabetes to the initiation and progression of chronic coronary disease and emphasize comprehensive risk factor management rather than treatment of isolated abnormalities (Rosenson et al., 2018).

The findings also highlight the close interrelationship among cardiometabolic variables involved in CAD pathophysiology (Lubrano & Balzan, 2021; Roth et al., 2020). Hypertension, diabetes mellitus, dyslipidemia, obesity, and aging do not act independently but interact through overlapping molecular pathways that amplify vascular injury. Chronic hyperglycemia promotes glycation of lipoproteins, rendering LDL particles more susceptible to oxidation while simultaneously impairing HDL-mediated reverse cholesterol transport (Brownlee, 2005). Oxidized LDL stimulates inflammatory cytokine production, vascular smooth muscle cell proliferation, and extracellular matrix remodeling, leading to progressive luminal narrowing. Meanwhile, hypertension increases arterial stiffness and endothelial shear stress, further facilitating lipid deposition within the vascular wall. Aging exacerbates these processes through cumulative oxidative damage, mitochondrial dysfunction, chronic low-grade inflammation, and diminished vascular regenerative capacity. Consequently, individuals exposed to multiple cardiometabolic risk factors experience additive and potentially synergistic increases in CAD risk

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compared with those presenting with a single abnormality. The present findings therefore reinforce the concept that comprehensive cardiovascular risk assessment incorporating lipid profile, glycemic status, blood pressure, and traditional clinical risk factors provides greater predictive value than evaluation of individual biomarkers alone.

The findings of this study have several important implications for clinical practice and cardiovascular prevention. Routine assessment of lipid profile and HbA1c, together with careful evaluation of hypertension history, may facilitate earlier identification of individuals at high risk of CAD and support timely implementation of preventive interventions. These findings are consistent with current recommendations advocating integrated cardiovascular risk management through aggressive control of blood pressure, optimization of lipid levels, glycemic management, lifestyle modification, smoking cessation, and regular physical activity. Such an integrated approach has the potential to reduce disease progression and improve long-term cardiovascular outcomes.

Nevertheless, several limitations should be acknowledged. First, the cross-sectional design precludes the establishment of temporal or causal relationships between cardiometabolic risk factors and CAD. Second, the relatively small sample size may have limited statistical precision and reduced the generalizability of the findings to broader populations. Third, residual confounding from unmeasured variables including dietary habits, physical activity, medication adherence, family history of premature cardiovascular disease, socioeconomic status, inflammatory biomarkers, and duration of hypertension or diabetes cannot be completely excluded. Finally, CAD was classified as a dichotomous outcome rather than being quantified using angiographic severity scores such as the Gensini or SYNTAX score. Future multicenter prospective cohort studies with larger and more diverse populations, longitudinal follow-up, and comprehensive assessment of clinical and biochemical risk factors are warranted to validate these findings and further clarify the complex interactions underlying coronary artery disease.

CONCLUSION

This study demonstrates that coronary artery disease is associated with a combination of demographic, clinical, and metabolic factors. After adjustment for potential confounders, older age, higher body mass index, hypertension history, diabetes mellitus, LDL cholesterol $\geqslant$130 mg/dL, and HbA1c $\geqslant$6.5% remained independently associated with CAD. Hypertension showed the strongest association, followed by elevated LDL cholesterol and poor glycemic control. Although patients with CAD also had lower HDL cholesterol and higher triglyceride levels, these parameters were not independently associated with CAD after multivariable adjustment. These findings emphasize that cardiovascular risk assessment should integrate blood pressure history, lipid profile, glycemic status, body weight, and age rather than evaluate individual risk factors separately. Routine monitoring and simultaneous management of hypertension, LDL cholesterol, diabetes, and obesity may support earlier identification of high-risk patients and more targeted prevention of CAD. Nevertheless, the cross-sectional design and small, single-center sample limit causal interpretation and generalizability. Larger multicenter prospective studies are therefore needed to confirm these associations and evaluate their ability to predict the incidence and severity of CAD.

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