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Original Research Article

Association between Body Mass Index and Coronary Artery Disease Risk in Young Adults

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Abstract

Background: Coronary artery disease (CAD) has increasingly been recognized as a significant health concern among young adults, with obesity emerging as a major modifiable risk factor. However, the precise nature and magnitude of the association between body mass index (BMI) and angiographically confirmed CAD in young adults remain insufficiently characterized. This study aimed to evaluate the association between BMI categories and the prevalence, severity, and pattern of coronary artery disease in young adults undergoing coronary angiography.

Methods: A cross-sectional analytical study was conducted at a tertiary care center. A total of 468 patients aged 18–45 years who underwent coronary angiography for suspected CAD were included. Participants were categorized into four BMI groups: normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), class I obesity (30.0–34.9 kg/m²), and class II–III obesity (≥35.0 kg/m²). Angiographic findings, cardiovascular risk factors, lipid profiles, inflammatory markers, and metabolic parameters were systematically compared across BMI categories.

Results: The mean age was 38.4 ± 5.6 years, with 71.8% male predominance. The prevalence of angiographically significant CAD (≥50% stenosis) increased progressively across BMI categories: 28.4% in normal weight, 42.7% in overweight, 56.3% in class I obesity, and 68.9% in class II–III obesity (p-trend < 0.001). Multivessel disease was significantly more prevalent in obese patients (34.2% vs. 12.8% in normal weight, $p < 0.001$). After multivariable adjustment, class II–III obesity remained independently associated with significant CAD (adjusted odds ratio [aOR] 3.24, 95% CI 1.72–6.11, $p < 0.001$). Higher BMI was significantly associated with elevated high-sensitivity C-reactive protein (hs-CRP), insulin resistance (HOMA-IR), triglycerides, and lower HDL cholesterol levels.

Conclusion: Elevated BMI demonstrates a strong, graded, and independent association with coronary artery disease prevalence and severity in young adults. Obesity-mediated metabolic derangements and chronic systemic inflammation likely constitute the principal pathophysiological mechanisms underlying this relationship. Aggressive weight management strategies should be prioritized in young adult cardiovascular risk reduction programs.

Keywords: Body Mass Index; Coronary Artery Disease; Young Adults; Obesity; Cardiovascular Risk; Coronary Angiography; Metabolic Syndrome.

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Introduction

Coronary artery disease has historically been regarded as a disease of middle-aged and elderly populations. However, mounting epidemiological evidence over the past two decades has revealed an alarming increase in the incidence of CAD among young adults, with individuals below 45 years of age now accounting for 5–10% of all acute myocardial infarction cases in contemporary registries [1]. This epidemiological shift carries profound implications for public health, as premature CAD in young adults is associated with substantial loss of productive life years, significant psychological burden, and considerable long-term healthcare expenditure [2]. The global obesity epidemic has evolved in parallel with this rise in premature CAD, and the temporal association between these two trends is unlikely to be coincidental. The worldwide prevalence of obesity has nearly tripled since 1975, and current estimates suggest that over 650 million adults are obese, with the highest rates of increase observed among younger age groups [3]. Body mass index, while an

imperfect measure of adiposity, remains the most widely utilized anthropometric parameter for classifying weight status in clinical and epidemiological research and has been consistently associated with cardiovascular morbidity and mortality in large prospective cohort studies [4].

The pathophysiological mechanisms linking obesity to coronary atherosclerosis are multifaceted and involve a complex interplay of metabolic, inflammatory, hemodynamic, and neurohumoral pathways. Excess adiposity, particularly visceral adiposity, promotes insulin resistance, atherogenic dyslipidemia (characterized by elevated triglycerides, reduced HDL cholesterol, and increased small dense LDL particles), chronic low-grade systemic inflammation, endothelial dysfunction, oxidative stress, and activation of the renin-angiotensin-aldosterone system [5]. Additionally, obesity is strongly associated with the clustering of traditional cardiovascular risk factors—hypertension, diabetes mellitus, and metabolic syndrome—further amplifying coronary risk [6].

Several large-scale epidemiological studies have established the association between elevated BMI and incident cardiovascular events. The Framingham Heart Study demonstrated a continuous positive relationship between BMI and coronary heart disease risk over long-term follow-up [7]. More recently, the Global BMI Mortality Collaboration, pooling data from over 10 million individuals, confirmed that both overweight and obesity were associated with significantly increased all-cause and cardiovascular mortality [8]. Importantly, Khan et al. (2018) demonstrated that the lifetime risk of cardiovascular disease varied substantially by BMI status even among young adults, with obese individuals exhibiting markedly higher cumulative incidence of heart failure and CAD [9].

Despite this compelling epidemiological evidence, several gaps persist in the current literature. First, most studies examining the BMI-CAD relationship have relied on clinical endpoints (myocardial infarction, cardiovascular death) rather than angiographic documentation of coronary atherosclerosis, limiting the ability to characterize the pattern, extent, and severity of coronary involvement [10].

Second, studies specifically focused on young adult populations are relatively few, and the independent contribution of BMI to angiographic CAD after adjustment for obesity-associated metabolic mediators (dyslipidemia, diabetes, and hypertension) remains incompletely elucidated [11]. Third, the relationship between BMI and inflammatory biomarkers in the specific context of premature CAD deserves further investigation, as inflammation has been increasingly recognized as a central driver of atherosclerotic plaque development and instability [12]. Fourth, regional and ethnic variations in the BMI-CAD relationship have been documented, with South Asian populations demonstrating increased cardiovascular risk at lower BMI thresholds compared to Western populations, highlighting the need for population-specific data [13].

The aim of this study was to evaluate the association between BMI categories and the prevalence, severity, and pattern of angiographically documented coronary artery disease in young adults aged 18–45 years, and to assess whether BMI constitutes an independent predictor of CAD after adjusting for traditional cardiovascular risk factors and metabolic mediators.

Materials and Methods

Study Design and Setting: This was a cross-sectional analytical study conducted at a tertiary care and interventional center.

Study Population: Consecutive patients aged 18–45 years who underwent elective or emergency coronary angiography for clinical indications (acute coronary syndrome, positive non-invasive stress testing, anginal symptoms with high pretest probability, or evaluation of cardiomyopathy) during the study period were screened for eligibility.

Inclusion Criteria: Patients were included if they were: (1) aged 18–45 years; (2) undergoing coronary angiography with adequate visualization of all three major epicardial coronary territories; and (3) had complete anthropometric, laboratory, and clinical data available.

Exclusion Criteria: The following patients were excluded: (1) prior coronary revascularization (percutaneous coronary intervention or coronary artery bypass grafting); (2) known congenital heart disease or coronary anomalies; (3) severe valvular heart disease as the primary indication for catheterization; (4) active malignancy or chronic inflammatory conditions (systemic lupus erythematosus, rheumatoid arthritis, inflammatory bowel disease); (5) chronic kidney disease with eGFR <30 mL/min/1.73 m²; (6) pregnancy; (7) chronic corticosteroid or immunosuppressive therapy use; and (8) BMI <18.5 kg/m² (underweight), as the study focused on the association between overweight/obesity and CAD.

Anthropometric and Clinical Assessment: All patients underwent standardized anthropometric measurements prior to catheterization. Height was measured to the nearest 0.1 cm using a wall-

mounted stadiometer, and weight was measured to the nearest 0.1 kg using a calibrated digital scale with the patient in light clothing without shoes. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²). Waist circumference was measured at the midpoint between the lowest rib and the iliac crest using a non-stretchable measuring tape. Blood pressure was recorded as the average of two measurements taken in the seated position after five minutes of rest using a validated oscillometric device.

Patients were classified into four BMI groups according to WHO criteria: normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), class I obesity (30.0–34.9 kg/m²), and class II–III obesity (≥35.0 kg/m²).

Laboratory Investigations: Fasting venous blood samples were obtained within 24 hours of coronary angiography. The following parameters were measured: complete lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides), fasting blood glucose, glycated hemoglobin (HbA1c), fasting insulin, high-sensitivity C-reactive protein (hs-CRP), serum creatinine, and complete blood count.

Insulin resistance was estimated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) = [fasting glucose (mg/dL) × fasting insulin (μU/mL)] / 405. Metabolic syndrome was defined according to the harmonized Joint Interim Statement criteria.

Coronary Angiography and Assessment: Coronary angiography was performed via the radial or femoral approach using standard Judkins technique. Angiograms were reviewed by two experienced interventional cardiologists blinded to patient BMI status, with discrepancies resolved by consensus. Significant CAD was defined as ≥50% luminal diameter stenosis in at least one major epicardial coronary artery or its major branches. Patients were further classified as having single-vessel disease (SVD), double-vessel disease (DVD), or triple-vessel disease (TVD)/left main disease. Angiographic severity was additionally quantified using the Gensini scoring system, which assigns weighted scores based on stenosis severity and lesion location.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation and compared across BMI groups using one-way ANOVA with Bonferroni post hoc correction. Categorical variables were expressed as frequencies and percentages and compared using chi-square tests for trend. Multivariable binary logistic regression was performed to evaluate the independent association between BMI categories and significant CAD, adjusting for age, sex, and smoking status, family history of premature CAD, hypertension, diabetes mellitus, LDL cholesterol, and hs-CRP. Pearson and Spearman correlation coefficients were calculated as appropriate. All analyses were performed using SPSS version 28.0 (IBM Corporation, Armonk, NY, USA), with a two-tailed p-value <0.05 considered statistically significant.

Results

Baseline Characteristics: A total of 468 patients met inclusion criteria and were enrolled. The mean age was 38.4 ± 5.6 years, and 336 patients (71.8%) were male. The distribution across BMI categories was: normal weight 109 (23.3%), overweight 164 (35.0%), class I obesity 135 (28.8%), and class II–III obesity 60 (12.8%). Baseline demographic, clinical, and metabolic characteristics stratified by BMI group are presented in Table 1.

The prevalence of hypertension, diabetes mellitus, metabolic syndrome, and family history of premature CAD increased significantly across ascending BMI categories (all p-trend < 0.01). Fasting glucose, HbA1c, HOMA-IR, total cholesterol, LDL cholesterol, triglycerides, and hs-CRP all demonstrated significant progressive elevation across BMI groups, while HDL cholesterol decreased significantly. Waist circumference increased correspondingly with BMI (p < 0.001).

Table 1: Baseline Demographic, Clinical, and Metabolic Characteristics by BMI Category

Variable	Normal Weight (n=109)	Overweight (n=164)	Class I Obesity (n=135)	Class II–III Obesity (n=60)	p-trend
Age (years), mean ± SD	37.6 ± 5.8	38.2 ± 5.4	39.1 ± 5.7	38.9 ± 5.3	0.126
Male sex, n (%)	74 (67.9)	118 (72.0)	100 (74.1)	44 (73.3)	0.384
BMI (kg/m²), mean ± SD	22.8 ± 1.4	27.3 ± 1.3	32.1 ± 1.4	38.6 ± 3.2	<0.001
Waist circumference (cm), mean ± SD	82.4 ± 6.8	94.6 ± 7.2	104.3 ± 8.1	116.8 ± 10.4	<0.001
Smoking, n (%)	28 (25.7)	48 (29.3)	42 (31.1)	18 (30.0)	0.436
Hypertension, n (%)	22 (20.2)	52 (31.7)	62 (45.9)	36 (60.0)	<0.001
Diabetes mellitus, n (%)	14 (12.8)	32 (19.5)	38 (28.1)	24 (40.0)	<0.001
Metabolic syndrome, n (%)	16 (14.7)	48 (29.3)	68 (50.4)	42 (70.0)	<0.001

Family history of premature CAD, n (%)	18 (16.5)	34 (20.7)	36 (26.7)	20 (33.3)	0.008
Total cholesterol (mg/dL)	184.6 ± 32.4	198.2 ± 36.8	212.4 ± 38.6	224.8 ± 42.1	<0.001
LDL cholesterol (mg/dL)	108.4 ± 28.6	122.6 ± 32.4	136.8 ± 34.2	148.2 ± 38.6	<0.001
HDL cholesterol (mg/dL)	48.6 ± 10.2	42.4 ± 9.8	38.2 ± 8.6	34.8 ± 7.4	<0.001
Triglycerides (mg/dL)	132.4 ± 48.6	168.6 ± 62.4	198.4 ± 72.8	238.6 ± 84.2	<0.001
Fasting glucose (mg/dL)	94.2 ± 14.6	102.8 ± 22.4	112.6 ± 28.8	124.4 ± 36.2	<0.001
HbA1c (%)	5.4 ± 0.6	5.8 ± 0.9	6.2 ± 1.2	6.8 ± 1.6	<0.001
HOMA-IR	1.8 ± 0.9	2.8 ± 1.4	4.2 ± 2.1	5.8 ± 2.8	<0.001
hs-CRP (mg/L)	1.4 ± 1.2	2.6 ± 1.8	4.2 ± 2.4	6.8 ± 3.6	<0.001

Angiographic Findings: Coronary angiographic findings stratified by BMI category are presented in Table 2. The prevalence of angiographically significant CAD increased progressively from 28.4% in normal-weight patients to 42.7% in overweight, 56.3% in class I obesity, and 68.9% in class II–III obesity (p-trend < 0.001). Normal coronary arteries or non-significant atherosclerosis (<50% stenosis) were correspondingly more common in the normal-weight group. Among patients with significant CAD, multivessel disease (DVD + TVD) was significantly more prevalent in higher BMI categories: 12.8% in normal weight versus 21.3% in overweight, 34.2% in class I obesity, and 41.7% in class II–III obesity (p-trend < 0.001). The mean Gensini score increased progressively across BMI categories (12.4 ± 14.8 vs. 22.6 ± 18.4 vs. 34.8 ± 24.6 vs. 46.2 ± 28.4, p < 0.001). The left anterior descending artery was the most commonly affected vessel across all groups. Left main coronary artery involvement was rare overall but was observed more frequently in the class II–III obesity group (5.0% vs. 0.9% in normal weight, p = 0.046).

Table 2: Coronary Angiographic Findings by BMI Category

Angiographic Finding	Normal Weight (n=109)	Overweight (n=164)	Class I Obesity (n=135)	Class II–III Obesity (n=60)	p-trend
Significant CAD (≥50%), n (%)	31 (28.4)	70 (42.7)	76 (56.3)	41 (68.3)	<0.001
Normal/non-significant, n (%)	78 (71.6)	94 (57.3)	59 (43.7)	19 (31.7)	<0.001
Single-vessel disease, n (%)	17 (15.6)	35 (21.3)	30 (22.2)	16 (26.7)	0.048
Double-vessel disease, n (%)	9 (8.3)	22 (13.4)	28 (20.7)	14 (23.3)	<0.001
Triple-vessel/left main, n (%)	5 (4.6)	13 (7.9)	18 (13.3)	11 (18.3)	<0.001
Multivessel disease (DVD+TVD), n (%)	14 (12.8)	35 (21.3)	46 (34.1)	25 (41.7)	<0.001
LAD involvement, n (%)	26 (23.9)	58 (35.4)	62 (45.9)	34 (56.7)	<0.001
LCx involvement, n (%)	12 (11.0)	28 (17.1)	36 (26.7)	20 (33.3)	<0.001
RCA involvement, n (%)	14 (12.8)	32 (19.5)	40 (29.6)	22 (36.7)	<0.001
Left main involvement, n (%)	1 (0.9)	3 (1.8)	4 (3.0)	3 (5.0)	0.046
Gensini score, mean ± SD	12.4 ± 14.8	22.6 ± 18.4	34.8 ± 24.6	46.2 ± 28.4	<0.001

Multivariable Analysis: The results of multivariable logistic regression analysis examining the independent association between BMI categories and significant CAD are presented in Table 3. In the unadjusted model, all BMI categories above normal weight were significantly associated with increased odds of significant CAD. In Model 1, adjusting for age, sex, smoking, and family history of premature CAD, the associations remained significant for all elevated BMI categories. In Model 2, which additionally adjusted for hypertension, diabetes mellitus, LDL cholesterol, and hs-CRP, the associations were attenuated but remained statistically significant for class I obesity (aOR 1.94, 95% CI 1.08–3.49, p = 0.027) and class II–III obesity (aOR 3.24, 95% CI 1.72–6.11, p < 0.001). The overweight category showed a trend toward significance in the fully adjusted model (aOR 1.48, 95% CI 0.87–2.52, p = 0.148). Among other covariates, diabetes mellitus (aOR 2.12, 95% CI 1.28–3.51, p = 0.004), smoking (aOR 1.86, 95% CI 1.18–2.93, p = 0.007), hs-CRP >3.0 mg/L (aOR 1.92, 95% CI 1.24–2.97, p = 0.003), and LDL cholesterol >130 mg/dL (aOR 1.74, 95% CI 1.14–2.66, p = 0.010) were independently associated with significant CAD.

Table 3: Multivariable Logistic Regression Analysis for Significant CAD

BMI Category	Unadjusted OR (95% CI)	p-value	Model 1 aOR (95% CI)	p-value	Model 2 aOR (95% CI)	p-value
Normal weight	**Reference**	**—**	**Reference**	**—**	**Reference**	**—**
Overweight	1.88 (1.12–3.15)	0.017	1.76 (1.04–2.98)	0.036	1.48 (0.87–2.52)	0.148
Class I obesity	3.24 (1.90–5.53)	<0.001	2.86 (1.64–4.98)	<0.001	1.94 (1.08–3.49)	0.027
Class II–III obesity	5.52 (2.84–10.73)	<0.001	4.86 (2.44–9.68)	<0.001	3.24 (1.72–6.11)	<0.001
Model 2 covariates						
Diabetes mellitus	—	—	—	—	2.12 (1.28–3.51)	0.004
Smoking	—	—	—	—	1.86 (1.18–2.93)	0.007
Hypertension	—	—	—	—	1.54 (0.98–2.42)	0.062
LDL >130 mg/dL	—	—	—	—	1.74 (1.14–2.66)	0.010
hs-CRP >3.0 mg/L	—	—	—	—	1.92 (1.24–2.97)	0.003

Model 1: Adjusted for age, sex, smoking, family history of premature CAD. Model 2: Model 1 + hypertension, diabetes mellitus, LDL cholesterol, hs-CRP.

Discussion

This cross-sectional study demonstrates a strong, graded, and independent association between BMI and angiographically confirmed coronary artery disease in young adults below 45 years of age. The prevalence of significant CAD increased more than twofold from normal-weight to class II–III obese individuals, and this association persisted after comprehensive multivariable adjustment for both traditional cardiovascular risk factors and inflammatory markers. These findings carry important clinical and public health implications given the escalating prevalence of obesity among young adults worldwide.

The observed dose-response relationship between increasing BMI and CAD prevalence aligns with data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, which demonstrated that elevated BMI in young adulthood was associated with accelerated coronary artery calcification over 25 years of follow-up [14]. Similarly, Yusuf et al. (2004), in the landmark INTERHEART study, identified abdominal obesity as one of the nine modifiable risk factors accounting for over 90% of population-attributable risk for acute myocardial infarction globally, with the association being particularly strong in younger age groups [15]. Our finding that the association between BMI and CAD was partially but not completely attenuated after adjustment for metabolic mediators (diabetes, dyslipidemia, hypertension) suggests that obesity exerts both indirect effects through traditional risk factor clustering and direct pathophysiological effects on the coronary vasculature [16]. The progressive elevation of hs-CRP across BMI categories observed in our study supports the central role of chronic low-grade systemic inflammation in obesity-mediated atherosclerosis. Adipose tissue, particularly visceral adipose tissue, functions as an active endocrine organ secreting pro-inflammatory adipocytokines including interleukin-6, tumor necrosis factor-alpha, leptin, and resistin, while producing reduced quantities of the anti-inflammatory and cardioprotective adiponectin [17]. The finding that hs-CRP >3.0 mg/L was independently associated with significant CAD (aOR 1.92) in our multivariable model is consistent with the results of the JUPITER trial and subsequent meta-analyses establishing inflammatory biomarkers as independent cardiovascular risk predictors [18].

The significant association between increasing BMI and insulin resistance (HOMA-IR ranging from 1.8 in normal weight to 5.8 in class II–III obesity) highlights the metabolic syndrome as a critical intermediary pathway. Insulin resistance promotes atherogenesis through multiple mechanisms including hyperinsulinemia-mediated vascular smooth muscle cell proliferation, enhanced hepatic VLDL production with consequent atherogenic dyslipidemia, increased plasminogen activator inhibitor-1 expression promoting a prothrombotic state, and impaired endothelial nitric oxide bioavailability [19]. The high prevalence of metabolic syndrome in our obese young adults (70.0% in class II–III obesity) mirrors concerning trends reported in contemporary epidemiological surveys [20].

The observation that multivessel disease was substantially more common among obese young adults (41.7% in class II–III obesity versus 12.8% in normal weight) is clinically significant, as multivessel CAD confers a substantially worse prognosis compared to single-vessel disease and may necessitate more complex revascularization strategies. Patel et al. (2019) similarly reported that obese patients presenting with acute myocardial infarction before age 50 had more extensive angiographic disease burden and higher Gensini scores compared to their normal-weight counterparts [21]. The progressively increasing

Gensini scores across BMI categories in our study (12.4 to 46.2) provide further quantitative evidence of the dose-dependent relationship between adiposity and coronary atherosclerotic burden.

The observation that the association between overweight (BMI 25.0–29.9 kg/m²) and significant CAD was attenuated to borderline significance after full multivariable adjustment (aOR 1.48, p = 0.148) is noteworthy and suggests that the cardiovascular risk associated with modest excess weight may be predominantly mediated through conventional risk factors rather than representing a direct independent effect of adiposity. This finding has implications for clinical risk stratification and is consistent with the "metabolically healthy overweight" phenotype described in some epidemiological studies, although the long-term cardiovascular safety of this phenotype remains debated [22].

Our study has several limitations. First, the cross-sectional design precludes causal inference and temporal sequencing of the BMI-CAD relationship. Second, BMI does not differentiate between lean body mass and adipose tissue and does not capture body fat distribution, which may be more relevant to cardiovascular risk. Waist circumference and waist-to-hip ratio, while recorded, were not the primary exposure variable. Third, selection bias may exist, as only patients referred for coronary angiography were included, potentially enriching the sample with higher-risk individuals. Fourth, certain confounders such as physical activity level, detailed dietary intake assessment, and socioeconomic status were not comprehensively assessed. Fifth, the relatively small number of patients in the class II–III obesity group may limit the precision of effect estimates for this subgroup. Future prospective longitudinal studies incorporating advanced imaging modalities such as coronary CT angiography and intravascular ultrasound would provide more nuanced characterization of the impact of obesity on coronary plaque burden and composition in young adults [23].

Conclusion

This study demonstrates a strong, graded, and independent association between elevated body mass index and angiographically confirmed coronary artery disease in young adults aged 18–45 years. The prevalence and severity of CAD, as assessed by the proportion of significant stenoses, multivessel disease, and Gensini scores, increased progressively across ascending BMI categories. Class I and class II–III obesity remained independently associated with significant CAD after comprehensive adjustment for traditional cardiovascular risk factors and inflammatory markers, suggesting that obesity exerts direct atherogenic effects beyond its well-established mediation through conventional risk factor clustering. The concurrent progressive escalation of inflammatory markers, insulin resistance, and atherogenic dyslipidemia across BMI categories highlights the complex, multifaceted pathophysiology underlying obesity-mediated coronary atherosclerosis. These findings underscore the critical importance of prioritizing aggressive weight management interventions, including lifestyle modification, dietary counseling, and when appropriate, pharmacological or surgical weight reduction strategies, as fundamental components of cardiovascular risk reduction programs targeting young adults. Early identification and management of obesity in this population may offer a significant opportunity to attenuate the growing burden of premature coronary artery disease.

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