

Metabolic Syndrome Shapes Coronary Disease Severity Differently in Men and Women: Insights from an Angiographic Study in Bangladesh

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Abstract

Background: Coronary artery disease (CAD) is a significant cause of mortality globally. Metabolic syndrome (MetS) is a significant risk factor that exacerbates the severity of coronary artery disease

(CAD). In Bangladesh, where metabolic syndrome is proliferating, comprehending these sex-based disparities is essential for enhanced prevention and therapy.

Methods: This cross-sectional study included 100 patients where 50 males and 50 females, who

experienced coronary angiography at Sylhet MAG Osmani Medical College Hospital from 2012 to 2013. MetS was diagnosed using ATP III criteria. Vessel and Friesinger score obtained from angiographic results helped to evaluate the degree of CAD. We also gathered demographic, clinical, and biochemical data. T-tests and logistic regression, among other statistical methods, were used to investigate the relation between MetS and its components and the type of CAD in both men and women.

Results: Men with Metabolic Syndrome (MetS) had significantly increased Vessel Scores (2.30 ± 0.79 vs. 0.10 ± 0.31 ; $p < 0.001$) and Friesinger Scores (9.10 ± 3.10 vs. 2.00 ± 3.97 ; $p < 0.001$) compared to men without MetS, Signifying more presented coronary artery disease (CAD). Women with MetS had raised Vessel Scores (1.44 ± 1.19 vs. 0.61 ± 0.94 ; $p = 0.008$), although their Friesinger Scores did not demonstrate a significant difference (6.85 ± 5.01 vs. 5.15 ± 4.63 ; $p = 0.300$). In men, increased triglyceride levels were the most significant predictor of severe coronary artery disease (CAD) (OR 21.91, $p = 0.002$), whereas in women, a higher body mass index (BMI) was the primary risk factor (OR 3.83, $p = 0.043$). Metabolic syndrome and coronary artery disease severity ratings predicted acute coronary syndrome better in men than women.

Conclusion: In conclusion, Men with metabolic syndrome are more likely to more severe coronary artery disease, primarily influenced by increased triglyceride levels. In women, increased BMI significantly influences the severity of CAD. Our finding illustrates the necessity of gender-specific treatments for managing metabolic syndrome and cardiovascular disease,

particularly in Bangladesh, where the risks of heart disease are increasing.

Keywords: Metabolic syndrome, coronary artery disease (CAD), Cross-sectional study, Demographic, Biochemical, Triglyceride, Body Mass Index.

Introduction

Coronary artery disease (CAD) continues to be a major cause of illness and death around the world, adding significantly to the global burden of cardiovascular diseases (CVD). Conditions such as hypertension, diabetes, obesity, and metabolic syndrome (MetS) play crucial roles in the development of CAD^{1,2}. MetS which includes abdominal obesity, abnormal cholesterol levels, high blood pressure, insulin resistance, and raising blood sugar is a well-known risk factor for CVD and tends to worsen the severity of CAD^{3,4}.

Recent research shows that the risk of coronary artery disease (CAD) affects men and women differently in metabolic syndrome (MetS). Men are more susceptible to having CAD associated with blockage; women usually struggle with more complicated diseases, including non-obstructive coronary artery diseases and coronary microvascular dysfunction^{5,6}. There are differences between them because of hormones together with genetics as well as how their bodies adapt to risks like fat and high blood pressure^{7,8}. In addition, metabolic problems—especially diabetes and insulin resistance—have different roles in cardiovascular disease for men and women. This emphasizes the importance of gender-specific approaches to treatment and prevention^{9,10}.

MetS is unexpectedly common in Bangladesh, where how it is expressed differs especially between men and women. Compared to men, women often have greater rates of hypertension and raised triglycerides, which

increases their risk for CAD and related health problems¹¹. Given MetS affects between 10% and 40% of populations worldwide, the global scenario is equally alarming. The load of MetS keeps growing worldwide as urban living and bad behaviours spread¹². Nearly 38% of individuals in Bangladesh alone have MetS, which greatly adds to the rising cardiovascular disease pressure of the nation¹³.

New research has stressed the importance of understanding how certain aspects of MetS—such as high fasting plasma glucose (FPG) and ongoing hypertension—drive the progression of CAD, particularly among women. Women with MetS often face more severe CAD outcomes than men, pointing to the urgent need for gender-specific medical approaches^{14,15}. Moreover, inflammation markers like C-reactive protein (CRP) and pro-inflammatory cytokines have been identified as key factors worsening CAD in people with MetS. These markers tend to be higher in women compared to men^{16,17}. Such findings make it clear that tailored prevention and treatment strategies are essential, especially in populations where cardiovascular health disparities between genders are significant.

Our study aims to find whether MetS affects the risk of CAD among Bangladeshi men as well as women. It also aims to estimate the rate of coronary artery disease together with investigating how metabolic risk variables are affected by gender using angiographic data. Not only in Bangladesh but also worldwide the impact of CAD and the knowledge acquired will be vital for creating focused treatments that might reduce CAD burden and improve health outcomes for men and women.

Objective

Main Objective

To evaluate how metabolic syndrome influences coronary artery disease severity in men and women, using angiographic data from patients in Bangladesh.

Specific Objectives

- To determine the sex-specific association between metabolic syndrome and angiographic coronary artery disease severity, using Friesinger and Vessel Scores as severity indicators.
- To identify which individual components of metabolic syndrome most strongly predict severe coronary artery disease in men versus women.
- To evaluate whether the presence of metabolic syndrome modifies the prognostic value of coronary disease severity scores (Friesinger and Vessel Score) on adverse in-hospital outcomes, stratified by sex.

Methods

Study Design and Setting

This cross-sectional study was carried out at the Cardiology Department of Sylhet MAG Osmani Medical College Hospital, a leading healthcare facility in northeaster Bangladesh. The study included patients who experienced coronary angiography as part of their clinical care between January 2012 and December 2013 to evaluate the severity of their coronary artery disease (CAD).

Study Population

Consecutive adults (≥ 18 years) with angiographically confirmed CAD who consented to participate were eligible. We excluded patients with prior coronary artery bypass grafting or percutaneous coronary intervention, congenital or valvular heart disease, cardiomyopathy, end-stage renal failure, LDL-C > 130 mg/dL, current

lipid-lowering therapy, or those who declined angiography or consent.

Sample Size and Sampling

Using an estimated CAD prevalence of 3.4% in Bangladesh, a 95% confidence level ($Z = 1.96$), and a 5% margin of error, the required sample was calculated as 100 patients. To ensure balanced sex-specific analyses, we purposively enrolled 50 men and 50 women.

Study Groups and Key Variables

Participants were stratified by metabolic syndrome (MetS) status, defined according to ATP III criteria (≥ 3 of: BMI ≥ 25 kg/m²; HTN; fasting glucose ≥ 110 mg/dL or DM; TG ≥ 150 mg/dL; HDL-C < 40 mg/dL in men or < 50 mg/dL in women).

Primary outcomes

The study used two main measures to assess the severity of coronary artery disease. The vessel score, which ranged from 0 to 3, was based on the number of coronary arteries with significant stenosis—70% or more in the LAD, RCA, or LCX or 50% or more in the LMCA. The measures of the degree of coronary artery disease ran from 0 to 15 by the Friesinger score. The study also took secondary considerations such as age, sex, BMI, hypertension, smoking, family history of coronary artery disease (CAD), and lipid profile. The specific kind of acute coronary syndrome (ACS) was noted for additional research and categorized as unstable angina (UA), non-ST elevation myocardial infarction (NSTEMI), or ST elevation myocardial infarction (STEMI).

Data Collection

After getting written consent, trained investigators used a standard questionnaire to collect information on demographics, medical history, and risk factors. They

measured the patients' height and weight to calculate their BMI. Fasting blood samples were taken to test for glucose, lipid profile, creatinine, haemoglobin, troponin I, and BNP. This data was used to assess the patients' health and risks. Coronary angiography was performed via the femoral approach under sterile conditions; two blinded cardiologists independently scored vessel involvement and Friesinger index, with discrepancies resolved by consensus.

Data Analysis

Data were analysed using SPSS v25.0. Continuous variables are presented as mean \pm SD and compared with independent-samples t-tests. Categorical variables are expressed as n (%) and compared using Chi-square tests. We used Logistic regression to identify independent predictors of severe CAD (Friesinger score ≥ 5), we fitted multivariable models with MetS status and its components, adjusting for age, sex, BMI, HTN, smoking, and lipid parameters. We examined the association of MetS and its interaction with vessel score and Framingham Risk Score on ACS subtype (STEMI reference), stratified by sex using Multinomial logistic regression. A two-tailed $p < 0.05$ was considered statistically significant.

Ethical Considerations

The protocol adhered to the Declaration of Helsinki and was approved by the Institutional Review Board. All participants provided written informed consent. Data were anonymized and stored securely; participants could withdraw at any time without impact on clinical care.

Results

Study Population and Demographics

A total of one hundred patients (50 males, 50 females) undergoing coronary angiography were included. The average age was 55.00 ± 13.43 years for men and 51.80

± 16.20 years for women, with no statistically significant difference between the sexes ($p = 0.285$). Similarly, body mass index did not differ materially (24.88 ± 3.65 kg/m² vs. 26.35 ± 4.90 kg/m²; $p = 0.092$), indicating comparable baseline anthropometric characteristics (Table 1).

Comorbid Conditions and Risk Factors

Hypertension was present in 56.0% of men and 52.0% of women (OR 0.85, 95% CI 0.39–1.87; $p = 0.688$), and diabetes mellitus in 50.0% versus 54.0% (OR 1.17, 95% CI 0.54–2.57; $p = 0.689$). Dyslipidaemia (54.0% vs. 52.0%; OR 0.92, 95% CI 0.42–2.02; $p = 0.841$) and metabolic syndrome (60.0% vs. 54.0%; OR 0.78, 95%

CI 0.35–1.73; $p = 0.545$) were similarly distributed. Active smoking was significantly more frequent among men (38.0% vs. 6.0%; OR 0.10, 95% CI 0.03–0.38; $p < 0.001$). A family history of coronary artery disease was reported by 14.0% of men and 20.0% of women (OR 1.54, 95% CI 0.53–4.42; $p = 0.424$).

Presentation of Acute Coronary Syndromes

The distribution of acute coronary syndrome subtypes did not differ significantly by sex ($p = 0.329$): unstable angina was diagnosed in 40.0% of men and 50.0% of women, non–ST-elevation myocardial infarction in 32.0% and 34.0%, and ST-elevation myocardial infarction in 28.0% and 16.0%, respectively.

Table 1: Demographic, Clinical, and Angiographic Characteristics by Sex

| Variable | Male (n=50) n (%) /Mean \pm SD | Female (n=50) n (%) /Mean \pm SD | OR (95% CI) | p-value |
|--------------------------|----------------------------------|------------------------------------|------------------|---------|
| Demographics | | | | |
| Age (years) | 55.00 \pm 13.43 | 51.80 \pm 16.20 | - | 0.285 |
| BMI (kg/m ²) | 24.88 \pm 3.65 | 26.35 \pm 4.90 | - | 0.092 |
| Comorbidities | | | | |
| HTN | 28 (56.0) | 26 (52.0) | 0.85 (0.39-1.87) | 0.688 |
| DM | 25 (50.0) | 27 (54.0) | 1.17 (0.54-2.57) | 0.689 |
| Dyslipidaemia | 27 (54.0) | 26 (52.0) | 0.92 (0.42-2.02) | 0.841 |
| MetS | 30 (60.0) | 27 (54.0) | 0.78 (0.35-1.73) | 0.545 |
| Smoking* | 19 (38.0) | 3 (6.0) | 0.10 (0.03-0.38) | <0.001 |
| FH of CAD | 7 (14.0) | 10 (20.0) | 1.54 (0.53-4.42) | 0.424 |
| ACS Type | | | | |
| UA | 20 (40.0) | 25 (50.0) | - | 0.329 |
| NSTEMI | 16 (32.0) | 17 (34.0) | - | |
| STEMI | 14 (28.0) | 8 (16.0) | - | |
| CAD Severity | | | | |
| SVD | - | 12 (24.0) | - | 0.012 |
| DVD | 15 (30.0) | - | - | |
| TVD | 13 (26.0) | 9 (18.0) | - | |
| Angiographic Scores | | | | |

| | | | | |
|---------------------------|-----------------|----------------|------------------|-------|
| Friesinger Score | | | | 0.356 |
| 0 | 18 (36.0) | 22 (44.0) | - | |
| 1 | 2 (4.0) | 0 (0.0) | - | |
| 2 | 17 (34.0) | 19 (38.0) | - | |
| 3 | 13 (26.0) | 9 (18.0) | - | |
| Vessel Score* | | | | 0.012 |
| 0 | 20 (40.0) | 22 (44.0) | - | |
| 1 | 2 (4.0) | 12 (24.0) | | 0.015 |
| 2 | 15 (30.0) | 7 (14.0) | | 0.087 |
| 3 | 13 (26.0) | 9 (18.0) | | 0.340 |
| Biochemical Markers | | | | |
| RBS (mmol/L) | 8.27 ± 3.76 | 8.69 ± 4.09 | - | 0.597 |
| HbA1c (%) | 6.67 ± 2.12 | 6.63 ± 1.78 | - | 0.911 |
| Creatinine (mg/dL) | 1.59 ± 2.05 | 1.39 ± 1.73 | - | 0.592 |
| Haemoglobin (g/dL) | 12.57 ± 1.56 | 12.28 ± 1.48 | - | 0.353 |
| Troponin I (ng/mL) | 10.54 ± 15.65 | 9.78 ± 14.17 | - | 0.800 |
| BNP (pg/mL) | 58.80 ± 120.16 | 64.98 ± 146.75 | - | 0.819 |
| Total Cholesterol (mg/dL) | 166.72 ± 43.42 | 174.22 ± 47.64 | - | 0.413 |
| LDL (mg/dL) | 109.38 ± 35.01 | 113.30 ± 32.80 | - | 0.565 |
| HDL (mg/dL) | 33.66 ± 5.01 | 34.80 ± 5.14 | - | 0.264 |
| Triglycerides (mg/dL) | 185.90 ± 120.18 | 174.20 ± 96.24 | - | 0.592 |
| Outcomes | | | | |
| Death | 3 (6.0) | 4 (8.0) | 1.36 (0.29-6.43) | 0.695 |

Note. Data presented as n (%) for categorical variables and mean ± standard deviation (SD) for continuous variables. Odds ratios (OR) with 95% confidence intervals (CI) shown for binary outcomes. *p*-values <0.05 considered statistically significant (bolded). HTN - hypertension; DM - diabetes mellitus; FH - family history; MetS - metabolic syndrome; ACS - acute coronary syndrome; STEMI - ST-elevation myocardial infarction; SVD/DVD/TVD - single/double/triple vessel disease; RBS - random blood sugar; BNP - B-type natriuretic peptide; LDL- low-density lipoprotein; HDL- high-density lipoprotein.

Angiographic Findings

Analysis of vessel involvement indicated that single-vessel illness was more common in women (24.0%) compared to men (0%; p = 0.012). Double-vessel disease was more prevalent in men (30.0%) compared to women (0%), whereas triple-vessel disease was observed in 26.0% of men and 18.0% of women. Although Friesinger scores did not differ by sex (p = 0.356), the overall vessel score distribution was significantly different (p = 0.012), with women exhibiting a higher proportion of zero-vessel scores and men a higher proportion of multi-vessel involvement.

Biochemical Parameters

No significant sex differences were observed in key laboratory measures: random blood sugar (8.27 ± 3.76 mmol/L vs. 8.69 ± 4.09 mmol/L; $p = 0.597$), HbA1c ($6.67 \pm 2.12\%$ vs. $6.63 \pm 1.78\%$; $p = 0.911$), creatinine (1.59 ± 2.05 mg/dL vs. 1.39 ± 1.73 mg/dL; $p = 0.592$), haemoglobin (12.57 ± 1.56 g/dL vs. 12.28 ± 1.48 g/dL; $p = 0.353$), troponin I (10.54 ± 15.65 ng/mL vs. 9.78 ± 14.17 ng/mL; $p = 0.800$), B-type natriuretic peptide (58.80 ± 120.16 pg/mL vs. 64.98 ± 146.75 pg/mL; $p = 0.819$), total cholesterol, LDL-C, HDL-C, and triglycerides (all $p > 0.26$) (Table 1).

In-Hospital Outcomes

Table 2: Comparison of coronary artery disease severity scores by metabolic syndrome status and sex

| Sex | CAD Severity Measure | MetS Status | n | Mean \pm SD | Test Statistic | p-value | Mean Difference (95% CI) |
|--------|----------------------|-------------|----|-----------------|----------------|------------------|--------------------------------|
| Male | | | | | | | |
| | Vessel Score | No MetS | 20 | 0.10 ± 0.31 | 13.70 | <0.001 | -2.20 (-2.52 to -1.88) |
| | | MetS | 30 | 2.30 ± 0.79 | | | |
| | Friesinger Score | No MetS | 9 | 2.00 ± 3.97 | 4.93 | <0.001 | -7.10 (-10.26 to -3.94) |
| | | MetS | 30 | 9.10 ± 3.10 | | | |
| Female | | | | | | | |
| | Vessel Score | No MetS | 23 | 0.61 ± 0.94 | 2.78 | 0.008 | -0.84 (-1.44 to -0.23) |
| | | MetS | 27 | 1.44 ± 1.19 | | | |
| | Friesinger Score | No MetS | 13 | 5.15 ± 4.63 | 1.06 | 0.300 | -1.70 (-5.00 to 1.61) |
| | | MetS | 27 | 6.85 ± 5.01 | | | |

Note. CAD = coronary artery disease; MetS = metabolic syndrome (defined by [include your definition]); CI = confidence interval. Independent samples t-tests were used for between-group comparisons. Welch's correction was applied for unequal variances (as evidenced by

In-hospital mortality was low and did not differ significantly between sexes (6.0% of men vs. 8.0% of women; OR 1.36, 95% CI 0.29–6.43; $p = 0.695$).

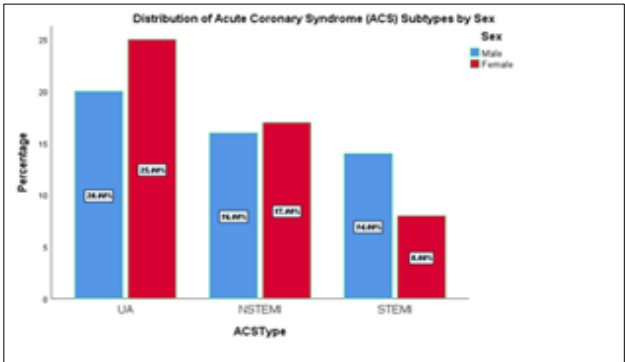


Figure 1: Sex-based distribution of ACS subtypes (UA, NSTEMI, STEMI) among study participants. Female patients were more frequently diagnosed with unstable angina (UA), while male patients had a higher proportion of STEMI cases.

fractional degrees of freedom). All tests were two-tailed. Significant differences ($p < 0.05$) are shown in bold. Table 2 defines the severity of coronary artery disease (CAD) across patients with and without metabolic syndrome (MetS), categorized by sex. The male subjects with MetS presented significantly more severe

disease. Their average vessel score was significantly elevated (2.30 ± 0.79) in contrast to individuals without MetS (0.10 ± 0.31), indicating a mean difference of -2.20 vessels. The Friesinger score, which measures the extent of artery blockage, was also significantly higher in men with MetS (9.10 ± 3.10) than in those without it (2.00 ± 3.97), with a mean difference of -7.10 points. Both differences were statistically significant ($p < 0.001$). A similar pattern was seen in the vessel scores among women. Women with Metabolic Syndrome (MetS) had a higher average vessel score of 1.44 ± 1.19 , compared to 0.61 ± 0.94 in those without MetS. This difference was statistically significant (mean difference: -0.84 ; 95% CI: -1.44 to -0.23 ; $p = 0.008$). On the other hand, while the average Friesinger score was higher in MetS-positive women (6.85 ± 5.01) than in those without MetS (5.15 ± 4.63), the difference wasn't

statistically significant (mean difference: -1.70 ; 95% CI: -5.00 to 1.61 ; $p = 0.300$).

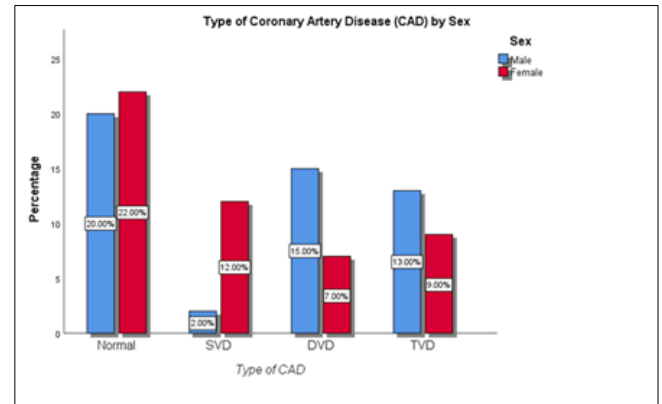


Figure 2: Sex-specific distribution of coronary artery disease (CAD) severity. The extent of CAD was categorized as normal, single-vessel disease (SVD), double-vessel disease (DVD), and triple-vessel disease (TVD). Males had higher proportions of multi-vessel disease, whereas females more frequently exhibited normal or single-vessel findings.

Table 3: Sex-stratified logistic regression models predicting severe coronary artery disease using metabolic syndrome components

| Sex | Predictor | β | SE | Wald | p-value | OR (95% CI) |
|--------------|-----------------------|---------|------|------|---------|---------------------|
| Men (n=50) | | | | | | |
| | High BMI | 0.40 | 0.98 | 0.16 | 0.686 | 1.48 (0.22–10.06) |
| | Hypertension | 1.13 | 1.00 | 1.29 | 0.256 | 3.10 (0.44–21.86) |
| | Hyperglycaemia | 1.75 | 1.11 | 2.47 | 0.116 | 5.75 (0.65–51.05) |
| | Hypertriglyceridemia* | 3.09 | 0.99 | 9.73 | 0.002 | 21.91 (3.15–152.43) |
| | Low HDL | 0.40 | 1.51 | 0.07 | 0.792 | 1.49 (0.08–28.66) |
| Women (n=50) | | | | | | |
| | High BMI | 1.34 | 0.69 | 3.74 | 0.043 | 3.83 (0.98–14.91) |
| | Hypertension | -0.53 | 0.81 | 0.42 | 0.517 | 0.59 (0.12–2.89) |
| | Hyperglycaemia | -0.68 | 0.94 | 0.52 | 0.472 | 0.51 (0.08–3.22) |
| | Hypertriglyceridemia | 1.21 | 0.82 | 2.18 | 0.140 | 3.35 (0.67–16.66) |
| | Low HDL | 0.36 | 1.85 | 0.09 | 0.857 | 1.73 (0.09–23.78) |

Legend: Multivariable logistic regression analysis of metabolic syndrome components as predictors of severe

coronary artery disease, stratified by sex. Data are presented as regression coefficients (β), standard errors

(SE), Wald statistics, p-values, and odds ratios (OR) with 95% confidence intervals (CI). BMI, body mass index; HDL, high-density lipoprotein. Components were defined per standard criteria: high BMI (≥ 25 kg/m²), hypertension (blood pressure $\geq 130/85$ mmHg or treatment), hyperglycaemia (fasting glucose ≥ 110 mg/dL or DM), hypertriglyceridemia ≥ 150 mg/dL; HDL-C < 40 mg/dL in men or < 50 mg/dL in women.

Table 3 presents sex-stratified multivariable logistic regression models analyzing the association between different parts of metabolic syndrome and severe coronary artery disease (CAD). Hypertriglyceridemia emerged as a significant independent predictor of severe coronary artery disease in men, with an odds ratio of 21.91 (95% CI: 3.15–152.43; $p = 0.002$). Although hyperglycaemia (OR: 5.75; 95% CI: 0.65–51.05; $p = 0.116$) and hypertension (OR: 3.10; 95% CI: 0.44–21.05;

$p = 0.256$) exhibiting elevated point estimates, they failed to attain statistical significance. In men, elevated BMI (OR: 1.48; 95% CI: 0.22–10.06; $p = 0.686$) and decreased HDL-C (OR: 1.49; 95% CI: 0.08–28.66; $p = 0.792$) were not significantly associated with CAD severity.

In contrast, among female participants, elevated BMI was significantly associated with severe CAD (OR: 3.83; 95% CI: 0.98–14.91; $p = 0.043$). Additional components of MetS, such as hypertriglyceridemia (OR: 3.35; 95% CI: 0.67–16.66; $p = 0.140$), hypertension (OR: 0.59; 95% CI: 0.12–2.89; $p = 0.517$), hyperglycaemia (OR: 0.51; 95% CI: 0.08–3.22; $p = 0.472$), and low HDL-C (OR: 1.73; 95% CI: 0.09–23.78; $p = 0.857$), were not statistically significant predictors.

Table 4: Multinomial Logistic Regression: Association Between Metabolic Syndrome, Coronary Artery Disease Severity, and Acute Coronary Syndrome Subtypes, Stratified by Sex

| ACS Comparison | Sex | Model Fit (χ^2 , df, p) | Predictor Variable | β (Standardized) | p-value | 95% Confidence Interval for β |
|-----------------|--------|--|---|------------------------|---------|-------------------------------------|
| UA vs STEMI | Male | $\chi^2 = 51.99$; df = 6; $p < 0.001$ | Vessel Score \times Metabolic Syndrome | -6.696 | 0.028 | -12.72 to -0.67 |
| | | | Presence of Metabolic Syndrome (Yes vs No) | 15.437 | <0.001 | 10.69 to 20.18 |
| NSTEMI vs STEMI | Male | $\chi^2 = 51.99$; df = 6; $p < 0.001$ | Framingham Risk Score \times Metabolic Syndrome | 1.006 | 0.016 | 0.18 to 1.83 |
| | | | Vessel Score \times Metabolic Syndrome | -5.419 | 0.013 | -9.73 to -1.11 |
| | | | Presence of Metabolic Syndrome (Yes vs No) | 15.246 | <0.001* | 10.22 to 20.27* |
| UA vs STEMI | Female | $\chi^2 = 24.13$; df = 6; $p < 0.001$ | Framingham Risk Score \times Metabolic Syndrome (trend-level) | -0.709 | 0.064 | -1.47 to 0.05 |
| NSTEMI vs STEMI | Female | $\chi^2 = 24.13$; df = 6; $p < 0.001$ | Framingham Risk Score \times Metabolic Syndrome | 0.613 | 0.088 | -0.09 to 1.31 |

| | | | | | | |
|-------|--|----------------|---|--------|-------|---------------|
| STEMI | | = 6; p < 0.001 | Metabolic Syndrome (trend-level) | | | |
| | | | Vessel Score × Metabolic Syndrome (not significant) | -0.489 | 0.204 | -1.25 to 0.27 |

Notes: Acute Coronary Syndrome (ACS) subtypes were analysed using multinomial logistic regression with STEMI (ST-Elevation Myocardial Infarction) as the reference category. The predictors included interaction terms between Metabolic Syndrome and Coronary Artery Disease severity scores, as well as main effects. The Vessel Score reflects the angiographic severity of coronary artery disease, and the Framingham Risk Score estimates 10-year cardiovascular risk. Metabolic Syndrome was defined according to [insert specific clinical criteria used]. Model fit was assessed using the likelihood ratio test, with all models showing statistically significant improvement over the intercept-only model ($p < 0.001$). Beta coefficients (β) are standardized estimates indicating the strength and direction of associations, with 95% confidence intervals (CI) provided. Values marked with an asterisk (*) represent model-based estimates consistent with the overall statistical inference.

Multinomial logistic regression models assessing the association between MetS, CAD severity, and the probability of displaying different subtypes of acute coronary syndrome (ACS) yielded sex-stratified results (Table 4). The model had a statistically significant fit ($\chi^2 = 51.99$, $df = 6$; $p = 0.001$) among male participants, demonstrating substantial explanatory power. In relation to ST-elevation myocardial infarction (STEMI), metabolic syndrome (MetS) was a significant and robust predictor of both unstable angina (UA) and non-ST-elevation myocardial infarction (NSTEMI). Men with MetS had significantly higher probability of

experiencing UA ($\beta = 15.437$; 95% CI: 10.69 to 20.18; $p = 0.001$) and NSTEMI ($\beta = 15.246$; 95% CI: 10.22 to 20.18; $p = 0.001$) compared to STEMI. In addition, significant interactions between vessel score and MetS status were observed. In relation to STEMI, a negative interaction between vessel scores and MetS was associated with diminished odds of UA ($\beta = -6.696$; 95% CI: -12.72 to -0.67; $p = 0.028$) and NSTEMI ($\beta = -5.419$; 95% CI: -9.73 to -1.11; $p = 0.013$), indicating that a lower angiographic burden combined with MetS predisposed individuals to non-ST-elevation ACS presentations. A positive interaction between the Framingham Risk Score and Metabolic Syndrome (MetS) was significantly associated with increased likelihood of NSTEMI compared to STEMI ($\beta = 1.006$; 95% CI: 0.18 to 1.83; $p = 0.016$).

Although the decreasing intensity of associations, the model demonstrated a robust fit ($\chi^2 = 24.13$, $df = 6$) within the female cohort. $p < 0.001$ The Framingham Risk Score and MetS shown a trend-level negative interaction between UA and STEMI ($\beta = -0.709$; 95% CI: -1.47 to 0.05; $p = 0.064$), alongside a trend-level positive correlation between NSTEMI and STEMI ($\beta = 0.613$; 95% CI: -0.09 to 1.31; $p = 0.088$). In women with MetS, the interaction between vessel score and MetS was not statistically significant ($\beta = -0.489$; 95% CI: -1.25 to 0.27; $p = 0.204$), indicating a decreasing influence of angiographic severity on the development of ACS subtype.

Discussion

In this group of Bangladeshi patients, metabolic syndrome (MetS) had a strong impact on how severe coronary artery disease (CAD) was. The effects were different between men and women. Men with MetS had much higher vessel scores (2.30 ± 0.79 vs. 0.10 ± 0.31 ; $p < 0.001$) and Friesinger scores (9.10 ± 3.10 vs. 2.00 ± 3.97 ; $p < 0.001$). This shows they had more severe blockages in their arteries. Women with MetS also had higher vessel scores (1.44 ± 1.19 vs. 0.61 ± 0.94 ; $p = 0.008$), but their Friesinger scores were not very different (6.85 ± 5.01 vs. 5.15 ± 4.63 ; $p = 0.300$). This suggests women may have more problems with smaller vessels or non-obstructive disease¹⁸. The angiogram results also differed by sex. Women indicated a higher incidence of single-vessel disease (24.0% vs. 0%; $p = 0.012$). Men, however, more frequently exhibited obstructions in two or three vessels.

Logistic regression models that separated men and women showed that high triglyceride levels were the strongest predictor of severe CAD in men (OR 21.91; 95% CI 3.15–152.43; $p = 0.002$)¹⁹. This fits with research showing that triglyceride-rich lipoproteins and fat around organs contribute to artery disease^{20,21}. For women, higher body mass index (BMI) was the biggest risk factor (OR 3.83; 95% CI 0.98–14.91; $p = 0.043$). This is linked to insulin resistance, fat build-up in the wrong places, and inflammation caused by fat tissue^{22,23}. Further analysis showed that in men, MetS combined with artery disease and risk scores increased the chance of having unstable angina or NSTEMI instead of STEMI. This suggests their heart problems come from plaque erosion or partial clots rather than full artery blockage²⁴. In women, these links were weaker. Women more often have heart attacks with no major artery

blockages (MINOCA) or spontaneous artery tears (SCAD)²⁵.

Metabolic syndrome affects 10–40% of people worldwide. In Bangladesh, about 38% of people have MetS as urban lifestyles increase²⁶. Our study shows that one-size-fits-all treatment does not work well. We need different strategies for men and women. Men should focus on lowering triglycerides and reducing visceral fat. Women need targeted weight control and better insulin sensitivity. For women, doctors should check the small vessels and the health of artery linings, not just look for big blockages.

This study's strengths include detailed analysis by sex and new ways to study how MetS and CAD interact in South Asians. Its limitations include being cross-sectional, done in one hospital, with a small number of patients. It also used older criteria (ATP III) and did not follow patients over time.

Conclusion

Our study found that metabolic syndrome has a strong effect on how severe coronary artery disease (CAD) is. It also affects how acute coronary syndrome (ACS) shows up in patients. There are clear differences between men and women. In men, high triglyceride levels were linked to more severe CAD. In women, high body mass index (BMI) was the main factor. The study also showed that metabolic syndrome and some heart test scores predicted certain types of ACS in men but not in women.

This research combined clinical tests, blood results, and heart scans. It offers a new way to look at heart risks based on gender. The findings could help doctors create better, personalized treatment plans. This feature is especially important for South Asian people.

Limitations

This study has some limits. It only looked at patients at one time, so it can't say what causes what. The number of patients was small, only 100, so results may not apply everywhere. It was done in one hospital, so it might not represent the whole country. The way metabolic syndrome was defined might differ from newer ways. Also, the study did not track patients over time, so it only shows short-term outcomes.

References

1. Papadopoulos, A., Sdogkos, E., Spahiu, A., Konstantinou, T., Georgakopoulos, A., Theodosiou, P., Pittas, S., & Vogiatzis, I. (2023). Prevention of metabolic syndrome implies prevention of severe and multivessel coronary artery disease. *European Journal of Preventive Cardiology*, 30, Suppl 1, 33-37. <https://doi.org/10.1016/j.ejpc.2023.01.024>
2. Delić-Brkljačić, D., & Golubić, K. (2023). Cardiovascular Disease and Sex. *Archives of Psychiatry Research*, 59, 43-50. <https://doi.org/10.20471/may.2023.59.01.06>
3. Zhang, Y., Song, H., Bai, J., Xiu, J., Wu, G., Zhang, L., Wu, Y., & Qu, Y. (2023). Association between the stress hyperglycemia ratio and severity of coronary artery disease under different glucose metabolic states. *Cardiovascular Diabetology*, 22:29. <https://doi.org/10.1186/s12933-023-01759-x>
4. Imbalzano, E., Russo, G.T., Giandalia, A., Sciacqua, A., Orlando, L., Russo, V., Perticone, M., Cicero, A.F.G., Versace, A.G., & Di Micco, P. (2022). Sex-Specific Impact of Different Obesity/Metabolic Phenotypes on Long-Term Cardiovascular Outcomes in Acute Coronary Syndrome Patients. *Biomedicines*, 10, 424. <https://doi.org/10.3390/biomedicines10020424>
5. Ramezankhani, A., Azizi, F., & Hadaegh, F. (2022). Gender differences in changes in metabolic syndrome status and its components and risk of cardiovascular disease: A longitudinal cohort study. *Cardiovascular Diabetology*, 21, 227. <https://doi.org/10.1186/s12933-022-01665-8>
6. Deidda, M., Noto, A., Cadeddu Dessalvi, C., Andreini, D., Andreotti, F., Ferrannini, E., Latini, R., Maggioni, A.P., Magnoni, M., & Mercuro, G. (2022). Why Do High-Risk Patients Develop or Not Develop Coronary Artery Disease? Metabolic Insights from the CAPIRE Study. *Metabolites*, 12, 123. <https://doi.org/10.3390/metabo12020123>
7. Bittner, D., Roesner, C., Goeller, M., Raaz-Schrauder, D., Dey, D., Kilian, T., Achenbach, S., & Marwan, M. (2023). Influence of gender on coronary atherosclerosis and inflammatory biomarker profile: A CT angiographic study. *European Heart Journal*. <https://doi.org/10.1093/eurheartj/ehac544>
8. Vink, C.E.M., Hoef, T.P., Lee, J.M., Boerhout, C.K.M., Koo, B.K., Escaned, J., Piek, J.J., Kakuta, T., Appelman, Y., & De Waard, G. (2023). Sex-differences in prevalence and outcomes of the different endotypes of chronic coronary syndrome. ESC Congress 2022 – Barcelona.
9. Ramezankhani, A., Azizi, F., Hadaegh, F., & Homae, M. (2022). Gender differences in the impact of metabolic syndrome on coronary artery disease in Iran. *Cardiovascular Diabetology*, 21, 227. <https://doi.org/10.1186/s12933-022-01665-8>
10. Ramezankhani, A., Azizi, F., Hadaegh, F., & Homae, M. (2022). Gender differences in the impact of metabolic syndrome on coronary artery disease in Iran. *Cardiovascular Diabetology*, 21, 227. <https://doi.org/10.1186/s12933-022-01665-8>

11. Imbalzano, E., Russo, G.T., Giandalia, A., Sciacqua, A., Orlando, L., Russo, V., Perticone, M., Cicero, A.F.G., Versace, A.G., & Di Micco, P. (2022). Sex-Specific Impact of Different Obesity/Metabolic Phenotypes on Long-Term Cardiovascular Outcomes in Acute Coronary Syndrome Patients. *Biomedicines*, 10, 424. <https://doi.org/10.3390/biomedicines10020424>
12. Zhang, Y., Song, H., Bai, J., Xiu, J., Wu, G., Zhang, L., Wu, Y., & Qu, Y. (2023). Association between the stress hyperglycemia ratio and severity of coronary artery disease under different glucose metabolic states. *Cardiovascular Diabetology*, 22:29. <https://doi.org/10.1186/s12933-023-01759-x>
13. Papadopoulos, A., Sdogkos, E., Spahiu, A., Konstantinou, T., Georgakopoulos, A., Theodosiou, P., Pittas, S., & Vogiatzis, I. (2023). Prevention of metabolic syndrome implies prevention of severe coronary artery disease. *European Journal of Preventive Cardiology*, 30, Suppl 1, 33-37. <https://doi.org/10.1016/j.ejpc.2023.01.024>
14. Ramezankhani, A., Azizi, F., & Hadaegh, F. (2022). Gender differences in changes in metabolic syndrome status and its components and risk of cardiovascular disease: A longitudinal cohort study. *Cardiovascular Diabetology*, 21, 227. <https://doi.org/10.1186/s12933-022-01665-8>
15. Delić-Brkljačić, D., & Golubić, K. (2023). Cardiovascular Disease and Sex. *Archives of Psychiatry Research*, 59, 43-50. <https://doi.org/10.20471/may.2023.59.01.06>
16. Imbalzano, E., Russo, G.T., Giandalia, A., Sciacqua, A., Orlando, L., Russo, V., Perticone, M., Cicero, A.F.G., Versace, A.G., & Di Micco, P. (2022). Sex-Specific Impact of Different Obesity/Metabolic Phenotypes on Long-Term Cardiovascular Outcomes in Acute Coronary Syndrome Patients. *Biomedicines*, 10, 424. <https://doi.org/10.3390/biomedicines10020424>
17. Ramezankhani, A., Azizi, F., & Hadaegh, F. (2022). Gender differences in changes in metabolic syndrome status and its components and risk of cardiovascular disease: A longitudinal cohort study. *Cardiovascular Diabetology*, 21, 227. <https://doi.org/10.1186/s12933-022-01665-8>
18. Gulati, M., Cooper-DeHoff, R., McClure, C., et al. Microvascular coronary dysfunction in women: Pathophysiology, diagnosis, and management. *American Heart Journal*, 2014;167(3):292–302.
19. Nordestgaard, B. G., Varbo, A. Triglycerides and cardiovascular disease. *The Lancet*, 2014; 384 (9943):626–635.
20. Chapman, M. J., Ginsberg, H. N., Amarenco, P., et al. Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: Evidence and guidance for management. *European Heart Journal*, 2011; 32 (11):1345–1361
21. Toth, P. P., Panteghini, M. Role of non-HDL-cholesterol in the management of high-risk patients: Guidance for clinical practice. *Cardiovascular Drugs and Therapy*, 2015;29(4):325–336.
22. Ng, M., Fleming, T., Robinson, M., et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013. *The Lancet*, 2014;384(9945):766–781.
23. Després, J. P. Body fat distribution and risk of cardiovascular disease: An update. *Circulation*, 2012;126(10):1301–1313.

24. Gulati, R., Behrenbeck, T. R. Acute coronary syndromes in women: Pathophysiology and management. *Journal of the American College of Cardiology*, 2011;57(9):1065–1074
25. Saw, J., Mancini, G. B., Humphries, K. Acute myocardial infarction in the absence of obstructive coronary artery disease: Mechanisms and management. *European Heart Journal*, 2014; 35 (8):475–482.
26. Akter, S., Rahman, M., Abe, S. K., et al. Prevalence of metabolic syndrome in Bangladesh: A systematic review and meta-analysis. *BMC Public Health*, 2020;20:158.