Original Research Article

https://doi.org/10.53339/aimdr.2026.12.1.1
E-ISSN: 2395-2822 | P-ISSN: 2395-2814

Distribution of metabolic syndrome components and their relationship with angiographic severity in non-ST elevation myocardial infarction

Md. Aminul Haque[1], Syeda Aleya Sultana[2], Mohammad Abdul Malek[3], Md. Wali-Ur-Rahman[3], Abu Yousuf Md. Shahidul Alam[1], Mst. Zinat Ara[4]

[1]Department of Medicine and Cardiology, Combined Military Hospital, Dhaka, Bangladesh, [2]Department of Medicine and Cardiology, Combined Military Hospital, Dhaka, Bangladesh, [3]Department of Medicine and Cardiology, Combined Military Hospital, Dhaka, Bangladesh, [4]Department of Obstetrics and Gynaecology, Kurmitola General Hospital, Dhaka, Bangladesh

Address for correspondence: Md. Aminul Haque, Department of Medicine and Cardiology, Combined Military Hospital, Dhaka, Bangladesh. E-mail: mahaque_dr@yahoo.com

Abstract

Background: Metabolic syndrome (MetS) is now well acknowledged as a major contributor to the severity of coronary artery disease (CAD). This study examined the correlation between MetS and the angiographic burden of CAD in individuals with non-ST elevation myocardial infarction (NSTEMI).

Methods: A cross-sectional study was carried out involving 192 NSTEMI patients who underwent coronary angiography at a tertiary cardiac center located in Bangladesh. Patients were divided into two groups: MetS ($n = 96$) and non-MetS ($n = 96$). The severity of angiographic findings was assessed using the vessel score, total stenosis score (TSS), and extension score (ES). Correlation and multivariable regression analyses were conducted to determine the predictors of CAD burden.

Results: The MetS group had significantly higher mean vessel score (2.12 vs. 1.66; $P < 0.001$), TSS (9.26 vs. 6.06; $P < 0.001$), and ES (53.7% vs. 39.1%; $P < 0.001$). Triple vessel disease and high-burden lesions were markedly more prevalent in MetS patients. Waist circumference, elevated blood pressure, triglycerides, and low high-density lipoprotein-cholesterol independently predicted CAD burden. MetS score correlated positively with all angiographic severity indices.

Conclusion: MetS is strongly associated with greater angiographic severity of CAD in NSTEMI, underscoring its role as a key modifiable risk cluster. Addressing MetS may be critical in preventing severe coronary outcomes.

Keywords: Angiographic burden, cardiovascular risk, coronary artery disease, metabolic syndrome, non-ST elevation myocardial infarction

Introduction

Coronary artery disease (CAD) is the primary cause of morbidity and mortality worldwide, posing the greatest clinical and economic burden in cardiovascular medicine. According to the World Health Organization, cardiovascular illnesses account for almost 18 million fatalities each year, representing approximately one-third of global deaths, with CAD alone accounting to over 9 million deaths.[1] While the burden of CAD was previously concentrated in high-income nations, low- and middle-income countries today bear over 75% of worldwide cardiovascular mortality, demonstrating a major epidemiological transformation driven by urbanization, aging populations, and lifestyle changes.[2] Bangladesh, like many South Asian countries, is experiencing a rapid rise in CAD incidence, despite traditionally lower historical prevalence – suggesting a shift in risk factor exposure and public health priorities.

Acute clinical symptoms of CAD are usually classified as acute coronary syndromes (ACSs), which include unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Globally, the proportional burden of ACS subtypes has shifted significantly, with NSTEMI now surpassing STEMI in numerous countries, including South Asia.[3] This trend has been attributed to population aging, improved early recognition of myocardial ischemia, and the clustering of cardiometabolic risk factors in urbanized populations.[4] Despite often having lower early in-hospital mortality, NSTEMI patients experience higher long-term mortality, increased rates of recurrent ischemia, and greater healthcare utilization than their STEMI counterparts.[5] This evolving disease profile necessitates a refined understanding of upstream determinants of CAD severity, especially within resource-constrained healthcare systems where risk stratification is pivotal.

One key variable receiving increased attention is the metabolic syndrome (MetS), a collection of interrelated risk factors that raise the likelihood of atherosclerotic cardiovascular disease and Type 2 diabetes. MetS is clinically defined as the presence of central (abdominal) obesity, hypertension, elevated fasting plasma glucose, increased triglycerides, and decreased high-density lipoprotein (HDL) cholesterol, according to the harmonized criteria established by the International Diabetes Federation (IDF) and other international organizations.[6] Globally, approximately 20–25% of the adult population meets diagnostic criteria for MetS.[7] However, the burden is disproportionately high in South Asian populations, including Bangladesh, where the prevalence ranges between 25 and 40%, with even higher rates in urban centers.[8] These trends are fueled by rapid urbanization, nutritional transitions, sedentary lifestyles, and genetic predisposition to visceral adiposity even at lower body mass indices.[9]

Beyond epidemiologic associations, a compelling biological rationale links MetS to both the development and severity of CAD. The pathophysiology of MetS promotes an atherogenic environment through insulin resistance, which impairs endothelial nitric oxide production, leading to endothelial dysfunction, impaired vasodilation, and vascular remodeling.[10] Concurrently, the chronic low-grade inflammatory state in MetS, characterized by elevated cytokines such as interleukin-6 and C-reactive protein, facilitates plaque formation and destabilization.[11] Dyslipidemia – particularly hypertriglyceridemia and reduced HDL-C – further contributes to a pro-atherogenic lipid milieu, while hyperglycemia and oxidative stress promote vascular smooth muscle proliferation and intimal thickening.[12] Moreover, a prothrombotic state, marked by increased fibrinogen and plasminogen activator inhibitor-1, predisposes MetS patients to acute thrombotic events. These pathophysiological changes collectively predispose individuals with MetS not only to higher CAD incidence but also to more severe and diffuse coronary atherosclerosis, as reflected in higher angiographic vessel scores and stenosis burden.[13]

Despite the growing recognition of MetS as a cardiovascular risk amplifier, data remain sparse regarding its impact on angiographic severity among patients presenting with NSTEMI in South Asian contexts, particularly in Bangladesh. Most available studies have either pooled diverse ACS populations or focused on Western cohorts, limiting regional applicability. Given the unique ethnic risk factor profiles, younger age at presentation, and resource limitations in South Asian cardiac care, such data are crucial for improving risk stratification, treatment prioritization, and secondary prevention strategies. This study, therefore, aims to evaluate the association between MetS and angiographic burden of CAD among patients presenting with NSTEMI in a tertiary care center in Bangladesh, thereby addressing a critical gap in cardiovascular epidemiology in the region.

Methods

This 12-month cross-sectional study was carried out in the Department of Cardiology at the National Heart Foundation Hospital and Research Institute (NHFH&RI) in Dhaka, Bangladesh. Purposive sampling was used to enroll patients who were admitted with NSTEMI and underwent coronary angiography (CAG) during their index hospitalization. A priori, the sample size was calculated to be 96 members per group (n = 192). Patients were separated into two groups: Group I (NSTEMI with MetS) and Group II (NSTEMI without MetS). Patients with NSTEMI of either gender who underwent CAG during admission met the inclusion criteria. Exclusion criteria were STEMI or unstable angina, past ACS, prior percutaneous coronary intervention/Coronary artery bypass grafting, severe hepatic/renal disease preventing CAG, refusal of CAG, and NSTEMI with congenital/valvular cardiac disease, cardiomyopathy, or severe systemic sickness. NSTEMI was characterized by ischemic electrocardiogram abnormalities (ST-segment depression ≥0.05 mV or T-wave inversion ≥0.2 mV) and/or positive troponin levels in the absence of ST elevation. All participants underwent a clinical evaluation, anthropometric measurements (height, weight, waist circumference, blood pressure), and assessment of cardiovascular risk factors (smoking, diabetes, hypertension, dyslipidemia, obesity, family history of CAD). Echocardiography and laboratory tests (lipid profile, fasting blood sugar, troponin I, CK-MB, serum creatinine) were carried out. MetS was diagnosed using the IDF, 2006 criteria – central obesity (waist circumference ≥90 cm in males or ≥80 cm in women) plus any two of the following: high triglycerides, low HDL-C, raised blood pressure, or impaired fasting glucose. The Sullivan technique was used to assess angiographic severity, including vessel score (0–3, number of vessels with ≥70% stenosis), total stenosis score (TSS) (sum of eight segments, range 0–32; high burden ≥16), and extension score (ES) (percentage of intimal surface involved, 0–100%; high burden ≥50%). The Academic Council of NHFH&RI provided ethical permission, and data were collected prospectively using a standardized form.

Statistical analysis

The data were analyzed with the Statistical Package for the Social Sciences version 16.0. Continuous variables are shown as mean ± standard deviation, whereas categorical variables are represented as percentages. Group comparisons were made using independent-samples t-tests (continuous) and Chi-square testing (categorical). Correlations were investigated using Spearman's coefficient, and linear regression was used to discover independent predictors of angiographic scores. A two-sided $P < 0.05$ indicated statistical significance.

Results

A total of 192 patients with NSTEMI were included, divided evenly into two groups: Group I (MetS; $n = 96$) and Group II (No MetS; $n = 96$). The MetS group had a substantially higher mean age (58.26 ± 8.7 vs. 52.45 ± 10.9 years; $P < 0.001$) and a greater proportion of persons aged 51–70 years. There was no statistically significant difference in gender distribution between groups; however, males predominated in both (72.9% vs. 81.2%; $P = 0.170$). MetS patients had a considerably higher body mass index (BMI), with a larger proportion falling into the overweight and obese categories. The MetS group had a mean BMI of 25.08 ± 1.53 kg/m^2, compared to 24.08 ± 1.50 kg/m^2 in the non-MetS group ($P < 0.001$). The MetS group had significantly greater waist circumference in both males (96.70 ± 5.06 cm vs. 92.31 ± 5.03 cm; $P < 0.001$) and females (87.81 ± 5.86 cm vs. 82.50 ± 5.82 cm; $P = 0.005$). Notably, all participants in the MetS group showed high waist circumference per IDF criteria, compared to 59.4% in the non-MetS group ($P < 0.001$). The MetS group had significantly higher rates of elevated blood pressure (88.5% vs. 27.1%; $P < 0.001$), diabetes mellitus (75.0% vs. 11.5%; $P < 0.001$), and dyslipidemia (40.6% vs. 26.0%; $P = 0.032$). No statistically significant differences were noted in smoking status ($P = 0.288$) or in the family history of CAD ($P = 0.848$) across the groups [Table 1].

The assessment of angiographic severity found that patients with MetS had much more severe CAD than those without MetS. The vessel score distribution revealed a higher prevalence of AQ4 triple vessel disease (TVD) among MetS patients (42.7% vs. 15.6%), whereas single vessel disease (SVD) was more common in the non-MetS group (45.8% vs. 21.9%). Double vascular disease was distributed similarly in both groups (31.2% vs. 36.5%). The MetS group had a substantially higher mean vessel score (2.12 ± 0.89) than the non-MetS group (1.66 ± 0.76; $P < 0.001$), indicating a greater burden of multivessel involvement. MetS

Table 1: Baseline demographic and clinical characteristics of the study population (*n*=192)

Variable	Group I (MetS, *n*=96) (%)	Group II (No MetS, *n*=96) (%)	*P*-value
Age (years)			
21–30	2 (2.1)	3 (3.1)	
31–40	0 (0.0)	11 (11.5)	
41–50	17 (17.7)	31 (32.3)	
51–60	43 (44.8)	32 (33.3)	
61–70	27 (28.1)	13 (13.5)	
71–80	7 (7.3)	6 (6.2)	
Mean±SD	58.26±8.7	52.45±10.9	<0.001**
Sex			
Male	70 (72.9)	78 (81.2)	0.170
Female	26 (27.1)	18 (18.8)	
BMI (kg/m^2)			
18.5–22.9	10 (10.4)	42 (43.8)	<0.001**
23–24.9	58 (60.4)	41 (42.7)	
25–29.9	27 (28.1)	13 (13.5)	
≥30	1 (1.0)	0 (0.0)	
Mean±SD	25.08±1.53	24.08±1.50	<0.001**
Waist circumference (cm)			
Male (mean±SD)	96.70±5.06	92.31±5.03	<0.001**
Female (mean±SD)	87.81±5.86	82.50±5.82	0.005**
Elevated waist circumference[1]	96 (100)	57 (59.4)	<0.001*
Raised blood pressure	85 (88.5)	26 (27.1)	<0.001*
Diabetes mellitus	72 (75.0)	11 (11.5)	<0.001*
Dyslipidemia	39 (40.6)	25 (26.0)	0.032*
Smoking status			
Current	28 (29.2)	40 (41.7)	0.288
Recent	8 (8.3)	9 (9.4)	
Former	16 (16.7)	13 (13.5)	
Never	44 (45.8)	34 (35.4)	
Family history of CAD	17 (17.7)	16 (16.7)	0.848

[1]Elevated waist circumference defined as≥90 cm in males, ≥80 cm in females. Statistical significance: $P \leq 0.05$; *P* from Chi-square test (), unpaired *t*-test (**). MetS: Metabolic syndrome, SD: Standard deviation, BMI: Body mass index, CAD: Coronary artery disease

patients reported higher rates of high-burden stenosis (≥16) compared to non-MetS patients (9.4% vs. 1.0%, $P = 0.009$). MetS patients had significantly higher average TSSs (9.26 ± 4.29 vs. 6.06 ± 3.07; $P < 0.001$). Similarly, ES analysis revealed that over half (46.9%) of the MetS group showed substantial coronary involvement (score ≥50), while only 24.0% in the non-MetS group ($P = 0.001$). The MetS group had a significantly higher mean ES (53.70 ± 18.11) compared to the non-MetS group (39.11 ± 17.59; $P < 0.001$), suggesting more widespread atherosclerotic disease [Table 2].Patients with MetS showed a markedly higher prevalence of triple vessel disease (42.7% vs. 15.6%), whereas SVD was more common in the non-MetS group (45.8% vs. 21.9%). Double vessel disease occurred at similar frequencies in both groups (31.2% vs. 36.5%) [Figure 1].

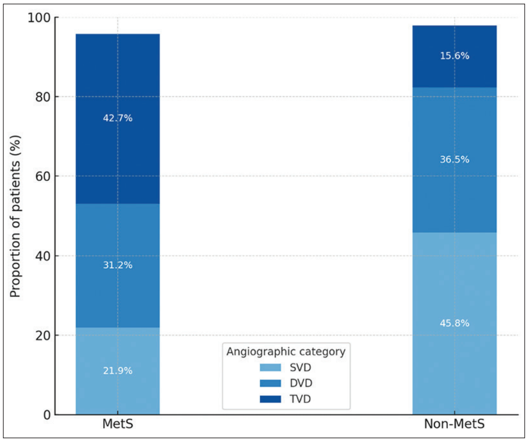

Figure 1: Angiographic severity by group (n=192)

The MetS score showed a strong positive connection with all three angiographic severity indices: vessel score (r = 0.202, $P = 0.005$), (TSS; r = 0.330, $P < 0.001$), and (ES; r = 0.349, $P < 0.001$). Waist circumference, triglycerides, total cholesterol, and LDL-C all showed a positive correlation with angiographic burden ($P < 0.05$). In contrast, HDL-C revealed strong inverse relationships with vascular score (r = −0.228), TSS (r = −0.236), and ES (r = −0.244; all $P = 0.001$). Fasting blood sugar had no significant correlation with any angiographic score, although BMI had a slight correlation with TSS (r = 0.169, $P = 0.019$).

In multivariable regression, waist circumference (β = 0.207 for TSS; β = 0.183 for ES), raised blood pressure (β = 0.161 for TSS; β = 0.162 for

Table 2: Angiographic severity of coronary artery disease in the study population (n=192)

Angiographic parameter	Group I (MetS, n=96) (%)	Group II (No MetS, n=96) (%)	P-value
Vessel score			
Score 0 (No significant lesion)	4 (4.2)	2 (2.1)	<0.001*
Score 1 (SVD)	21 (21.9)	44 (45.8)	
Score 2 (DVD)	30 (31.2)	35 (36.5)	
Score 3 (TVD)	41 (42.7)	15 (15.6)	
Mean±SD	2.12±0.89	1.66±0.76	<0.001**
Total stenosis score			
<16	87 (90.6)	95 (99.0)	0.009*
≥16	9 (9.4)	1 (1.0)	
Mean±SD	9.26±4.29	6.06±3.07	<0.001**
Extension score			
<50	51 (53.1)	73 (76.0)	0.001*
≥50	45 (46.9)	23 (24.0)	
Mean±SD	53.70±18.11	39.11±17.59	<0.001**

Statistical significance: $P \leq 0.05$; P from Chi-square test (), unpaired t-test (**). CAD: Coronary artery disease, SVD: Single vessel disease, DVD: Double vessel disease, TVD: Triple vessel disease, MetS: Metabolic syndrome, SD: Standard deviation

ES), elevated triglycerides (β = 0.164 for TSS; β = 0.146 for ES), and reduced HDL-C (β = −0.152 for TSS; β = −0.157 for ES) were identified as independent predictors of higher angiographic burden (all $P < 0.05$). Diabetes, age, and gender were not independently linked with angiographic severity [Table 3].

MetS score, waist circumference, total cholesterol, LDL-C, and triglycerides demonstrated positive correlations with vessel, stenosis, and ESs, while HDL-C showed consistent inverse correlations across all angiographic indices. Fasting blood sugar and BMI displayed no or only weak associations [Figure 2].

A sensitivity analysis found that patients with MetS had a significantly greater frequency of high-burden CAD at all angiographic thresholds. A TSS of ≥16 was observed in 9.4% of MetS patients

Table 3: Correlation of MetS score and components with angiographic severity, and multivariable predictors of CAD burden (*n*=192)

(A) Correlation coefficients between MetS score/components and angiographic scores			
Variable	**Vessel score r (*P*)**	**TSS r (*P*)**	**ES r (*P*)**
MetS score	0.202 (0.005)	0.330 (<0.001)	0.349 (<0.001)
Waist circumference	0.230 (0.001)	0.248 (0.001)	0.238 (0.001)
Total cholesterol	0.181 (0.012)	0.199 (0.006)	0.202 (0.005)
LDL-C	0.163 (0.024)	0.180 (0.012)	0.165 (0.022)
HDL-C	–0.228 (0.001)	–0.236 (0.001)	–0.244 (0.001)
Triglyceride	0.179 (0.013)	0.241 (0.001)	0.219 (0.002)
Fasting blood sugar	–0.053 (0.466)	–0.043 (0.557)	–0.015 (0.840)
BMI	0.091 (0.208)	0.169 (0.019)	0.137 (0.059)

(B) Multivariable linear regression: independent predictors of angiographic burden				
Predictor	**TSS β (95% CI)**	***P*-value**	**ES β (95% CI)**	***P*-value**
Waist circumference	0.207 (0.026–0.222)	0.014	0.183 (0.055–0.985)	0.029
Raised blood pressure	0.161 (0.120–2.516)	0.031	0.162 (0.598–11.97)	0.030
Low HDL-C	−0.152 (−0.181–−0.040)	0.041	−0.157 (−0.874–−0.032)	0.035
Triglyceride	0.164 (0.002–0.024)	0.021	0.146 (0.003–0.107)	0.039

r: Correlation coefficient, β: Regression coefficient. Significant values (*P*<0.05) in bold. Variables evaluated but not independently associated in the final models: diabetes, age, sex (all *P*>0.05). MetS: Metabolic syndrome, CAD: Coronary artery disease, TSS: Total stenosis score, ES: Extension score, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BMI: Body mass index, CI: Confidence interval

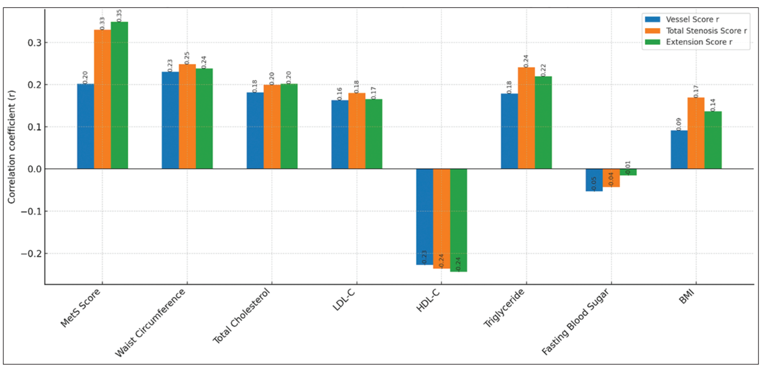

Figure 2: Correlation of metabolic syndrome score and lipid variables with angiographic severity

compared to 1.0% in non-MetS ($P = 0.009$). Similarly, an ES of ≥50 was reported in 46.9% vs. 24.0% ($P = 0.001$), and triple vessel disease was present in 42.7% vs. 15.6% ($P < 0.001$) [Table 4]. Multivariable regression analysis revealed that waist circumference, increased blood pressure, elevated triglyceride levels, and decreased HDL-C serve as independent predictors of higher stenosis and ESs. In contrast, age, sex, and diabetes did not significantly contribute to these outcomes [Figure 3].

Discussion

This study investigated the link between MetS and the angiographic burden of CAD in individuals with NSTEMI. Our data show that MetS is linked with much more unfavorable cardiovascular risk profiles, more widespread angiographic disease, and a higher burden of coronary atherosclerosis.

Demographically, patients with MetS were significantly older and exhibited higher BMI and waist circumference compared to those without MetS. Notably, 100% of MetS patients met the criteria for elevated waist circumference, highlighting the centrality of visceral adiposity in this population. Higher prevalence of diabetes mellitus, dyslipidemia, and raised blood pressure in the MetS group was consistent with known clustering of metabolic risk factors.[14] These findings mirror those of Mahalle *et al.*, who reported higher waist circumference and blood pressure among Indian MetS patients with ACS, reflecting regional parallels in metabolic risk patterns.[15]

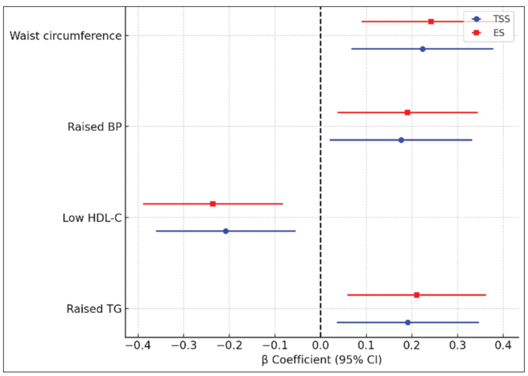

Figure 3: Forest plot showing independent predictors of coronary artery disease severity

Angiographically, MetS patients demonstrated significantly more severe disease. Triple vessel disease (TVD) was detected in 42.7% of MetS patients versus just 15.6% of non-MetS patients. Furthermore, MetS participants had significantly higher mean vessel scores, TSSs, and ESs, indicating both localized and diffuse coronary involvement. These data are closely related to the findings of Miri *et al*. and Zhou *et al*., both of whom found increased rates of multivessel and severe CAD in MetS populations, underlining the atherogenic potential of the syndrome even in acute coronary situations.[16,17]

The correlation analysis further reinforced the pathophysiological role of MetS components in atherosclerosis. MetS score positively correlated with all angiographic severity indices, particularly with the ES ($r = 0.349$, $P < 0.001$), suggesting that higher metabolic burden translates into more widespread coronary involvement. This is in agreement with Liu *et al*., who demonstrated that increasing MetS score was predictive of both luminal narrowing and plaque extension.[18] Triglycerides, waist circumference, and total cholesterol were positively associated with disease burden, while HDL-C showed an inverse correlation – consistent

Table 4: Sensitivity analysis for high-burden CAD thresholds

CAD burden indicator	Threshold	Group I (MetS)	Group II (non-MetS)	*P*-value
Total stenosis score	≥16	9.4% (9/96)	1.0% (1/96)	0.009
Extension score	≥50	46.9% (45/96)	24.0% (23/96)	0.001
Vessel score	TVD (Score 3)	42.7% (41/96)	15.6% (15/96)	<0.001

CAD: Coronary artery disease, MetS: Metabolic syndrome, TVD: Triple vessel disease with previous work identifying dyslipidemia and visceral obesity as key drivers of atherogenesis.[19]

Our multivariable analysis identified waist circumference, raised blood pressure, elevated triglycerides, and low HDL-C as independent predictors of both stenosis and ESs. Interestingly, diabetes, sex, and age were not significant predictors in the regression model, suggesting that metabolic parameters beyond glycemic control may play a dominant role in plaque burden. These findings echo those from Zhou *et al*., who similarly reported lipid and obesity indices as stronger predictors than age or diabetes status.[17]

Finally, the sensitivity analysis highlighted that MetS patients had a significantly greater likelihood of presenting with high-burden CAD – whether defined by TVD (42.7% vs. 15.6%), TSS ≥16 (9.4% vs. 1.0%), or ES ≥50 (46.9% vs. 24.0%). This high prevalence of severe and diffuse disease in MetS patients reinforces the need for early identification and aggressive risk factor management. Supporting this, Mahalle *et al*. and Liu *et al*. both reported that MetS presence reliably stratified patients into higher-risk angiographic categories.[15,18]

Taken together, our findings confirm that MetS is not only a risk factor for CAD onset but also a potent marker of disease severity and extent in patients presenting with NSTEMI. This underscores the clinical value of identifying MetS in acute settings and supports its inclusion in risk stratification algorithms.

Limitations of the study

The study took place in a single hospital with a small number of participants. As a result, the outcomes may not be indicative of the broader community.

Conclusion

This study highlights a significant link between MetS and the angiographic severity of CAD in individuals experiencing NSTEMI. Patients with MetS exhibited a markedly greater burden of coronary atherosclerosis, as demonstrated by higher vessel, stenosis, and ESs. Independent predictors of increased angiographic burden included central obesity, raised blood pressure, elevated triglycerides, and reduced HDL-C – all hallmark components of MetS. Importantly, correlation and sensitivity analyses affirmed that MetS not only predisposes individuals to CAD but also contributes to its diffuse and severe angiographic presentation. These findings reinforce the need for early identification and targeted management of MetS components to mitigate the risk of adverse cardiovascular outcomes. The study adds to the growing body of evidence supporting the prognostic relevance of MetS in ACSs, particularly in South Asian populations where its prevalence is rapidly rising. Future longitudinal and interventional studies are warranted to explore causality and the impact of MetS control on CAD progression and clinical outcomes.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

References

1. World Health Organization. Cardiovascular Diseases (CVDs); 2023. Available from: https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds) [Last accessed on 2025 Sep 20].
2. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, *et al*. Global burden of cardiovascular diseases and risk factors, 1990-2019: Update from the GBD 2019 study. J Am Coll Cardiol 2020;76:2982-3021.
3. Neumann JT, Goßling A, Sörensen NA, Blankenberg S, Magnussen C, Westermann D. Temporal trends in incidence and outcome of acute coronary syndrome. Clin Res Cardiol 2020;109:1186-92.
4. Puymirat E, Simon T, Cayla G, Cottin Y, Elbaz M, Coste P, *et al*. Acute myocardial infarction: Changes in patient characteristics, management, and 6-month outcomes over a period of 20 years in the FAST-MI program (French registry of acute ST-elevation or non-ST-elevation myocardial infarction) 1995 to 2015. Circulation 2017;136:1908-19.

5. Montalescot G, Dallongeville J, Van Belle E, Rouanet S, Baulac C, Degrandsart A, *et al*. STEMI and NSTEMI: Are they so different? 1 year outcomes in acute myocardial infarction as defined by the ESC/ACC definition (the OPERA registry). Eur Heart J 2007;28:1409-17.
6. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, *et al*. Harmonizing the metabolic syndrome. Circulation 2009;120:1640-5.
7. Ranasinghe P, Mathangasinghe Y, Jayawardena R, Hills AP, Misra A. Prevalence and trends of metabolic syndrome among adults in the Asia-pacific region: A systematic review. BMC Public Health 2017;17:101.
8. Chowdhury MZ, Anik AM, Farhana Z, Bristi PD, Abu Al Mamun BM, Uddin MJ, *et al*. Prevalence of metabolic syndrome in Bangladesh: A systematic review and meta-analysis of the studies. BMC Public Health 2018;18:308.
9. Misra A, Khurana L. The metabolic syndrome in South Asians: Epidemiology, determinants, and prevention. Metab Syndr Relat Disord 2009;7:497-514.
10. Tziomalos K, Athyros VG, Karagiannis A, Mikhailidis DP. Endothelial dysfunction in metabolic syndrome: Prevalence, pathogenesis and management. Nutr Metab Cardiovasc Dis 2010;20:140-6.
11. Rizvi AA. Inflammation markers as mediators of vasculo-endothelial dysfunction and atherosclerosis in the metabolic syndrome and type 2 diabetes. Chin Med J 2007;120:1918.
12. Vlachopoulos C, Rokkas K, Ioakeimidis N, Stefanadis C. Inflammation, metabolic syndrome, erectile dysfunction, and coronary artery disease: Common links. Eur Urol 2007;52:1590-600.
13. Solymoss BC, Bourassa MG, Campeau L, Sniderman A, Marcil M, Lespérance J, *et al*. Effect of increasing metabolic syndrome score on atherosclerotic risk profile and coronary artery disease angiographic severity. Am J Cardiol 2004;93:159-64.
14. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome--a new world-wide definition. A consensus statement from the international diabetes federation. Diabet Med 2006;23:469-80.
15. Mahalle N, Garg MK, Naik SS, Kulkarni MV. Association of metabolic syndrome with severity of coronary artery disease. Indian J Endocrinol Metab 2014;18:708-14.
16. Miri R, Sajjadieh A, Parsamahjoob M, Hajibaratali B, Shekarchizadeh M, Kolahi AA, *et al*. Relationship between metabolic syndrome and angiographic severity of coronary artery disease. ARYA Atheroscler 2016;12:220-5.
17. Zhou J, Liu C, Zhou P, Li J, Chen R, Wang Y, *et al*. Prevalence and impact of metabolic syndrome in patients with multivessel coronary artery disease and acute coronary syndrome. Nutr Metab Cardiovasc Dis 2021;31:2693-9.
18. Liu T, Wu Z, Liu J, Lv Y, Li W. Metabolic syndrome and its components reduce coronary collateralization in chronic total occlusion: An observational study. Cardiovasc Diabetol 2021;20:104.
19. Varounis C, Rallidis LS, Franco OH, Lekakis J. Prevalence of metabolic syndrome and association with burden of atherosclerotic disease in patients with stable coronary artery disease. Curr Med Res Opin 2016;32:1175-81.

How to cite this article: Haque MA, Sultana SA, Malek MA, Wali-Ur-Rahman M, Alam AYMS, Ara Z. Distribution of metabolic syndrome components and their relationship with angiographic severity in non-ST elevation myocardial infarction. Ann. Int. Med. Den. Res. 2026;12(1):1-9.

Source of Support: Nil, **Conflict of Interest:** None declared

Received: 02-Dec-2025; **Revised:** 03-Jan-2026;
Acceptance: 19-Jan-2026; **Published:** 10-Mar-2026