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Clinical, Biochemical Risk Factors and Echocardiographic Patterns of NSTEMI Patients with and without Metabolic Syndrome

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Abstract

Background: *Metabolic syndrome (MetS) increases cardiovascular risk and may affect the clinical, biochemical, echocardiographic, and angiographic profiles of individuals with non-ST elevation myocardial infarction (NSTEMI). This study sought to examine NSTEMI patients with and without MetS in order to assess risk patterns and illness severity.*

Methods: *A hospital-based cross-sectional study was conducted on 192 NSTEMI patients, divided equally into groups with (n=96) and without (n=96) MetS. MetS was defined using International Diabetes Federation (IDF) criteria. Clinical, biochemical, echocardiographic, and angiographic parameters were analyzed, and multivariable regression was applied to identify independent predictors of CAD severity.*

Results: *Patients with MetS were older (≤50 years: 22.9% vs. 42.7%, p<0.001) and had higher prevalence of obesity (64.6% vs. 40.6%, p=0.007), hypertension (65.6% vs. 34.4%, p<0.001), diabetes (64.6% vs. 22.9%, p<0.001), and dyslipidemia (59.4% vs. 28.1%, p<0.001). Biochemically, raised glucose (87.5% vs. 55.2%), hypertriglyceridemia (79.2% vs. 38.5%), and reduced HDL-C (86.5% vs. 49.0%) were more common in MetS (all p<0.001). Echocardiographic findings showed lower mean LVEF (53.0 ± 8.8 vs. 55.7 ± 7.4, p=0.022). Angiographically, MetS patients had higher triple vessel disease (42.7% vs. 15.6%, p<0.001), TSS (9.26 ± 4.29 vs. 6.06 ± 3.07, p<0.001), and ES (53.7 ± 18.1 vs. 39.1 ± 17.6, p<0.001). Regression analysis identified waist circumference, triglycerides, low HDL-C, and blood pressure as independent predictors of CAD severity.*

Conclusion: *MetS significantly worsens clinical, biochemical, echocardiographic, and angiographic severity in NSTEMI, with central obesity, dyslipidemia, and hypertension driving disease burden. Targeted metabolic control may improve risk stratification and outcomes.*

Keywords: *Non-ST elevation myocardial infarction, metabolic syndrome, echocardiography, angiographic severity, risk factors*

I. INTRODUCTION

Acute coronary syndromes (ACS) continue to be a leading cause of cardiovascular morbidity and mortality worldwide, putting a significant pressure on healthcare systems in both developed and developing countries. Non-ST elevation myocardial infarction (NSTEMI) accounts for a significant proportion of ACS patients and is distinguished by a variety of clinical outcomes, persistent ischemia risk, and changing left ventricular (LV) dysfunction.[1] Acute coronary syndromes (ACS) continue to be the leading cause of cardiovascular morbidity and mortality worldwide, putting a significant pressure on healthcare systems in both developed and developing countries. Non-ST elevation myocardial infarction (NSTEMI) accounts for a sizable proportion of ACS patients and is distinguished by a variety of clinical outcomes, persistent ischemia risk, and changing left ventricular (LV) dysfunction.[2] Regional studies from South Asia have highlighted that NSTEMI is increasingly prevalent in younger individuals, frequently presenting with clustering of modifiable risk factors like as diabetes, hypertension, and obesity.[3,4] These observations highlight the urgent need to examine upstream determinants that influence disease severity and prognosis in vulnerable populations.

One significant factor is the metabolic syndrome (MetS), which comprises a group of interconnected cardiometabolic risk factors such as central obesity, dyslipidemia, hypertension, and impaired glucose regulation.[5] According to the International Diabetes Federation (IDF), diagnosis of MetS requires central obesity, defined by ethnic-specific waist circumference cutoffs, along with at least two additional metabolic abnormalities.[6] This definition is particularly relevant to South Asian populations, who demonstrate a greater predisposition to visceral adiposity and insulin resistance at lower body mass indices compared with Western populations.[7] The prevalence of MetS has risen sharply across South Asia, with recent systematic reviews reporting rates between 14% and 46% depending on population studied and diagnostic criteria applied.[8-10] This rise mirrors global trends in obesity and sedentary lifestyles and portends an increasing burden of cardiovascular disease in regions already facing earlier onset and more aggressive forms of coronary artery disease (CAD).

Beyond its role as a clustering of risk factors, MetS acts as a powerful cardiovascular risk enhancer. Each component contributes mechanistically to atherosclerosis and myocardial injury: insulin resistance fosters endothelial dysfunction and thrombosis; hypertension accelerates vascular remodeling and plaque instability; dyslipidemia promotes atherogenic lipid deposition; and obesity fuels systemic inflammation and oxidative stress.[11,12] When these factors coexist, their effects are synergistic, amplifying vascular injury beyond the sum of individual risks.[13] Consequently, patients with MetS are more likely to present with complex coronary lesions, diffuse atherosclerosis, and poorer clinical outcomes following ACS.[14,15] In biochemical terms, higher levels of triglycerides, lower HDL cholesterol, and elevated inflammatory mediators such as C-reactive protein (CRP) and interleukins are commonly observed in ACS patients with MetS, further compounding their risk.[16,17]

Echocardiography remains an indispensable, non-invasive tool for the evaluation of myocardial structure and function in NSTEMI patients. It provides critical insights into LV systolic performance, diastolic function, and regional wall motion abnormalities, all of which carry prognostic value. In South Asian cohorts, echocardiography has demonstrated that NSTEMI patients often present with LV systolic dysfunction and diastolic abnormalities at admission, findings associated with worse clinical outcomes.[18] Similarly, 2D echocardiography frequently reveals subclinical wall motion abnormalities in NSTEMI patients, even in the absence of overt heart failure.[19] Emerging evidence suggests that MetS may exacerbate these abnormalities by promoting adverse LV remodeling and impaired myocardial relaxation, thereby worsening long-term cardiac function.[20] However, systematic evaluations comparing echocardiographic patterns of NSTEMI patients with and without MetS remain limited, particularly in South Asia.

Despite the compelling evidence linking MetS to adverse cardiovascular outcomes, important research gaps persist. Many studies have focused primarily on angiographic severity or short-term outcomes in ACS, often combining STEMI and NSTEMI cohorts or relying on small sample sizes. Few have systematically examined the combined influence of clinical, biochemical, and echocardiographic parameters in NSTEMI patients stratified by MetS status. Moreover, regional data from South Asia remain scarce, even though populations in this region demonstrate unique cardiometabolic profiles and face disproportionate disease burden (Sucato et al., 2023). Addressing this gap is crucial, as a more integrated evaluation may refine risk stratification, inform preventive strategies, and improve clinical management of NSTEMI in high-risk groups.

Therefore, the present study was designed to evaluate and compare the clinical characteristics, biochemical risk factors, and echocardiographic patterns of NSTEMI patients with and without MetS. By incorporating comprehensive assessment across these domains, this study aims to clarify the impact of MetS on myocardial involvement in NSTEMI and provide insights into its prognostic implications.

II. METHODS

This hospital based, cross-sectional analytical study was done in the Department of Cardiology, National Heart Foundation Hospital and Research Institute, Dhaka, between August 2013 to August 2014. All 192 consecutive patients with NSTEMI were included. Patients with previous myocardial infarction, percutaneous coronary intervention or coronary artery bypass grafting and significant valvular heart disease, congenital heart disease, specific cardiomyopathies or incomplete angiographic and biochemical data were not included. NSTEMI was diagnosed by the combination of typical ischemic chest pain lasting more than 30 minutes, without persistent ST-segment elevation on an ECG and abnormal serum troponin I levels.

The study cohort was split into two equal segments: 96 individuals who fulfilled the diagnostic criteria for metabolic syndrome (MetS) and 96 individuals without MetS. MetS was characterized based on the International Diabetes Federation (IDF) 2005 guidelines, which necessitate central obesity (waist circumference ≥90 cm in men and ≥80 cm in women for South Asians) along with any two of the following: elevated fasting plasma glucose (≥5.6 mmol/L or a prior diagnosis of diabetes), increased triglycerides (≥150 mg/dL), decreased high-density lipoprotein cholesterol (HDL-C <40 mg/dL in men, <50 mg/dL in women), and elevated blood pressure (systolic ≥130 mmHg, diastolic ≥85 mmHg, or current treatment for hypertension).[6] Baseline data included anthropometric measurements, blood pressure, and pertinent clinical details. Laboratory tests encompassed fasting blood glucose, serum lipid profile, and serum creatinine.

All patients underwent transthoracic echocardiography to assess left ventricular systolic function, expressed as left ventricular ejection fraction (LVEF). Coronary angiography was performed in all patients using the standard Judkins technique, and the severity of coronary artery disease was assessed according to Sullivan's method.[21,22] A vessel score was assigned by counting the number of major epicardial coronary arteries (left anterior descending, left circumflex, and right coronary artery) with ≥50% luminal diameter stenosis. The extent of atherosclerosis was quantified using the Total Stenosis Score (TSS), calculated as the sum of stenosis scores of all lesions graded from 1 to 4 based on severity of luminal narrowing (50–74%, 75–89%, 90–99%, and 100%, respectively), with a possible range of 0 to 32. The Extension Score (ES) was used to estimate the proportion of the coronary arterial tree affected by atherosclerosis, expressed as a percentage of the total myocardial territory, with each coronary segment assigned a pre-specified weight; scores ≥16 for TSS and ≥50% for ES were considered severe.[23] Data analysis was carried out using conventional statistical methods. Continuous variables were indicated as mean ± standard deviation (SD) and compared between groups via Student's t-test, while categorical variables were indicated as frequencies and percentages and compared using the chi-square test. Pearson's correlation coefficient was utilized to examine the relationships between MetS score, its individual components, and angiographic severity scores. A multivariable linear regression analysis was conducted to ascertain the independent predictors of TSS and ES, including the components of metabolic syndrome as covariates. A p-value of less than 0.05 was regarded as statistically significant.

RESULTS

Table 1. Baseline Clinical and Demographic Characteristics of the Study Population (n=192)

Variable	Category	NSTEMI with MetS (n=96) (n,%)	NSTEMI without MetS (n=96) (n,%)	p-value
Age (years)	≤50	22 (22.9)	41 (42.7)	<0.001
	51–60	41 (42.7)	29 (30.2)	
	>60	33 (34.4)	26 (27.1)	
Sex	Male	70 (72.9)	78 (81.2)	0.170
	Female	26 (27.1)	18 (18.8)	
BMI (kg/m²)	<25	34 (35.4)	57 (59.4)	0.007
	≥25	62 (64.6)	39 (40.6)	
Hypertension	Present	63 (65.6)	33 (34.4)	<0.001
Diabetes mellitus	Present	62 (64.6)	22 (22.9)	<0.001
Dyslipidemia	Present	57 (59.4)	27 (28.1)	<0.001
Smoking	Present	43 (44.8)	38 (39.6)	0.468
Family history of CAD	Present	24 (25.0)	22 (22.9)	0.741

Patients with NSTEMI and metabolic syndrome were significantly older, with a higher proportion aged 51–60 years compared to those without metabolic syndrome (42.7% vs. 30.2%, p<0.001). Obesity was more prevalent in the metabolic syndrome group, with two-thirds having a BMI ≥25 kg/m² (64.6% vs. 40.6%, p=0.007). Hypertension (65.6% vs. 34.4%, p<0.001), diabetes mellitus (64.6% vs. 22.9%, p<0.001), and dyslipidemia (59.4% vs. 28.1%, p<0.001) were also significantly more common among NSTEMI patients with metabolic syndrome. In comparison, the distribution of sex, smoking habits, and family history of coronary artery disease were similar across both groups.

Table 2. Biochemical Profile of the Study Population

Variable	Category	NSTEMI with MetS (n=96) (n,%)	NSTEMI without MetS (n=96) (n,%)	p-value
Fasting glucose (mg/dL)	<5.6	12 (12.5)	43 (44.8)	<0.001
	≥5.6 or diabetes	84 (87.5)	53 (55.2)	
Triglycerides (mg/dL)	<150	20 (20.8)	59 (61.5)	<0.001
	≥150	76 (79.2)	37 (38.5)	
HDL-C (mg/dL)	Normal	13 (13.5)	49 (51.0)	<0.001
	Reduced	83 (86.5)	47 (49.0)	
LDL-C (mg/dL)	Normal (<130)	50 (52.1)	53 (55.2)	0.440
	Elevated (≥130)	46 (47.9)	43 (44.8)	
Creatinine (mg/dL)	Normal	91 (94.8)	93 (96.9)	0.659
	Elevated	5 (5.2)	3 (3.1)	

The biochemical profile of the study population is presented in Table 2. Patients with metabolic syndrome had significantly higher prevalence of impaired fasting glucose or diabetes (87.5% vs. 55.2%, p<0.001), hypertriglyceridemia (79.2% vs. 38.5%, p<0.001), and reduced HDL-C (86.5% vs. 49.0%, p<0.001) compared with those without metabolic syndrome. In comparison, there was no significant difference in LDL-C levels and serum creatinine values between the two groups.

Table 3. Distribution of Metabolic Syndrome Components

Component	With MetS (n=96) (n,%)	Without MetS (n=96) (n,%)	p-value
Raised waist circumference	84 (87.5)	38 (39.6)	<0.001
Raised blood pressure	63 (65.6)	33 (34.4)	<0.001
Raised fasting glucose	62 (64.6)	22 (22.9)	<0.001
Raised triglycerides	76 (79.2)	37 (38.5)	<0.001
Reduced HDL-C	83 (86.5)	47 (49.0)	<0.001
Patients fulfilling 3 components	10 (10.4)	-	-
Patients fulfilling 4 components	54 (56.2)	-	-
Patients fulfilling 5 components	32 (33.3)	-	-

Table 3 shows the distribution of individual components of metabolic syndrome among the study groups. Raised waist circumference, elevated blood pressure, impaired fasting glucose, hypertriglyceridemia, and reduced HDL-C were all significantly more common in patients with metabolic syndrome compared to those without (p<0.001 for each). Among the metabolic syndrome group, the majority fulfilled either four (56.2%) or five (33.3%) diagnostic components, while only 10.4% met the minimum of three criteria.

Table 4. Echocardiographic Findings

Variable	Category	With MetS (n=96) (n,%)	Without MetS (n=96) (n,%)	p-value
LVEF (%)	≥55 (Normal)	39 (40.6)	60 (62.5)	0.009
	45–54 (Mild dysfunction)	32 (33.3)	26 (27.1)	
	30–44 (Moderate dysfunction)	20 (20.8)	12 (12.5)	
	<30 (Severe dysfunction)	1 (1.0)	0 (0.0)	
Mean LVEF ± SD	53.0 ± 8.8	55.7 ± 7.4	0.022	

As shown in Table 4, left ventricular systolic function was significantly lower in NSTEMI patients with metabolic syndrome. Normal LVEF (≥55%) was observed less frequently in the MetS group compared with those without MetS (40.6% vs. 62.5%, p=0.009). Conversely, moderate dysfunction (30–44%) was more common in the MetS group (20.8% vs. 12.5%). Severe dysfunction was rare overall but present in one patient with MetS. The mean LVEF was also significantly lower among patients with MetS compared to those without (53.0 ± 8.8 vs. 55.7 ± 7.4, p=0.022).

Table 5. Angiographic Severity of Coronary Artery Disease

Variable	With MetS (n=96) (n,%)	Without MetS (n=96) (n,%)	p-value
Single vessel disease	21 (21.9)	44 (45.8)	<0.001
Double vessel disease	34 (35.4)	37 (38.6)	
Triple vessel disease	41 (42.7)	15 (15.6)	
Mean vessel score	2.12 ± 0.89	1.66 ± 0.76	<0.001
Total Stenosis Score (TSS)	9.26 ± 4.29	6.06 ± 3.07	<0.001
Severe stenosis (TSS ≥16)	9 (9.4)	1 (1.0)	0.009
Extension Score (ES, %)	53.7 ± 18.1	39.1 ± 17.6	<0.001
Severe extension (ES ≥50%)	45 (46.9)	23 (24.0)	0.001

Table 5 summarizes the angiographic findings. Patients with metabolic syndrome had a greater burden of multivessel disease, with triple vessel involvement being nearly three times more common compared to those without MetS (42.7% vs. 15.6%, p<0.001). In contrast, single-vessel disease was more frequent in the non-MetS group (45.8% vs. 21.9%). The mean vessel score was significantly higher in patients with MetS (2.12 ± 0.89 vs. 1.66 ± 0.76, p<0.001). Similarly, the total stenosis score (9.26 ± 4.29 vs. 6.06 ± 3.07, p<0.001) and the extension score (53.7 ± 18.1 vs. 39.1 ± 17.6, p<0.001) were significantly greater in the MetS group. Severe disease, defined as TSS ≥16 and ES ≥50%, was also more prevalent among patients with MetS (p=0.009 and p=0.001, respectively).

Table 6. Correlation of MetS Score and Components with Angiographic Severity

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, 0.007	0.248, <0.001	0.238, <0.001
Blood pressure	0.114, 0.113	0.163, 0.024	0.191, 0.008
Fasting blood sugar	0.108, 0.134	0.117, 0.108	0.102, 0.157
Triglycerides	0.179, 0.004	0.241, <0.001	0.219, <0.001
HDL-C	–0.228, 0.002	–0.236, <0.001	–0.244, <0.001

Table 6 demonstrates the correlations between metabolic syndrome score, its individual components, and angiographic severity indices. The overall MetS score showed significant positive correlations with vessel score (r=0.202, p=0.005), total stenosis score (TSS; r=0.330, p<0.001), and extension score (ES; r=0.349, p<0.001). Among the individual components, waist circumference and triglycerides correlated positively with all three severity indices, while reduced HDL-C demonstrated consistent negative correlations (all p<0.01).

Table 7. Independent Predictors of CAD Severity (Multivariable Regression)

Predictor	β (95% CI) for TSS	p-value	β (95% CI) for ES	p-value
Waist circumference	0.207 (0.026–0.222)	0.005	0.183 (0.055–0.985)	0.002
Raised blood pressure	0.161 (0.120–2.516)	0.027	0.162 (0.598–11.97)	0.015
Low HDL-C	–0.152 (–0.181 to –0.04)	0.008	–0.157 (–0.874 to –0.032)	0.002
Raised triglycerides	0.164 (0.002–0.024)	0.016	0.146 (0.003–0.107)	0.007

Multivariable regression analysis (Table 7) identified waist circumference, elevated blood pressure, low HDL-C, and raised triglycerides as independent predictors of coronary artery disease severity. Waist circumference and triglycerides were positively associated with both TSS and ES, while reduced HDL-C remained an inverse predictor. Elevated blood pressure also independently predicted higher TSS and ES values. These findings highlight the strong contribution of central obesity, dyslipidemia, and hypertension to angiographic burden in NSTEMI patients.

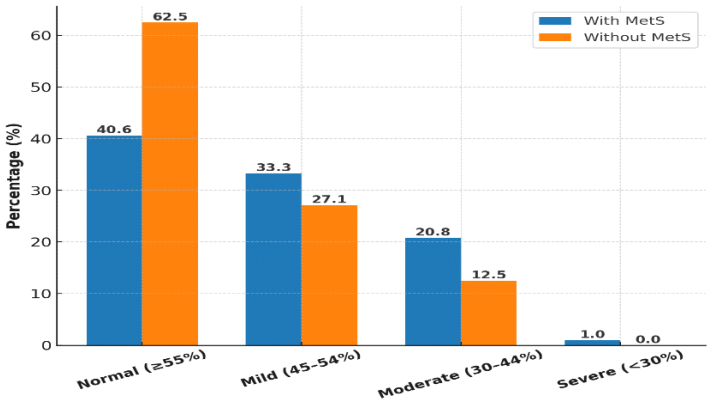

Figure 1. Bar chart of LV function categories (MetS vs non-MetS).

Figure 1 illustrates the distribution of left ventricular systolic function among patients with and without metabolic syndrome. A smaller proportion of patients with MetS had preserved LVEF (≥55%) compared with those without MetS (40.6% vs. 62.5%). In contrast, moderate dysfunction (30–44%) was more frequent in the MetS group (20.8% vs. 12.5%). Severe dysfunction (<30%) was uncommon overall but was observed exclusively in the MetS group. These findings demonstrate an association between metabolic syndrome and impaired systolic function in NSTEMI patients.

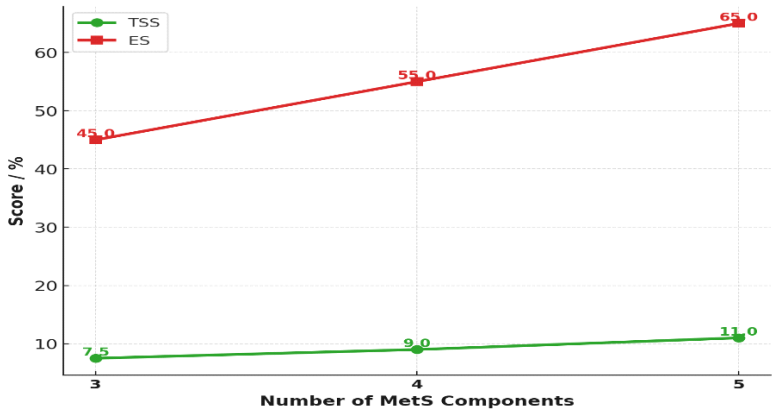

Figure 2. Line chart showing correlation of MetS components with CAD severity (TSS & ES).

Figure 2 illustrates a distinct dose–response relationship between the quantity of metabolic syndrome components and the angiographic severity of coronary artery disease. The total stenosis score (TSS) and extension score (ES) both exhibited a progressive increase with the accumulation of MetS components, escalating from 7.5 and 45.0, respectively, in patients who satisfied three criteria, to 11.0 and 65.0 in those who met all five. This stepwise escalation highlights that greater clustering of metabolic risk factors is associated with markedly worse coronary atherosclerotic burden in NSTEMI patients.

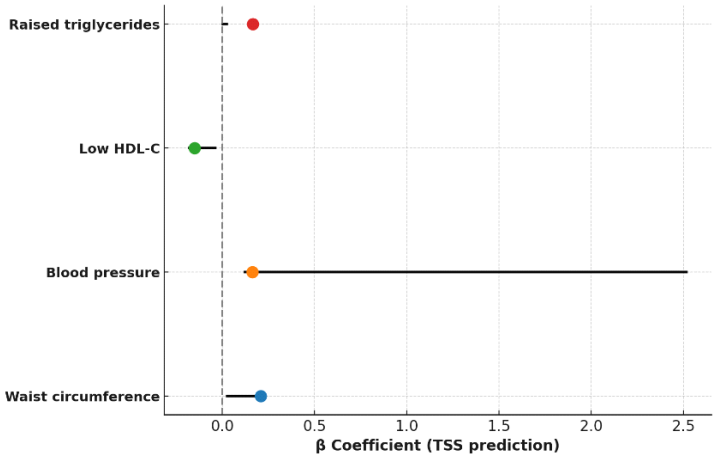

Figure 3. Forest Plot of Independent Predictors of CAD Severity.

Figure 3 illustrates the independent predictors of angiographic severity in NSTEMI patients. Waist circumference, elevated blood pressure, and raised triglycerides emerged as significant positive predictors of total stenosis score, whereas low HDL-C was an inverse predictor. The forest plot underscores the dominant role of central obesity and dyslipidemia in driving coronary atherosclerotic burden, with blood pressure exerting an additional contributory effect.

III. DISCUSSION

This study demonstrated that NSTEMI patients with metabolic syndrome (MetS) exhibited a significantly more adverse clinical, biochemical, echocardiographic, and angiographic profile compared with those without MetS. These findings emphasize the amplifying effect of metabolic clustering on the severity of coronary artery disease (CAD) and its clinical manifestations.

From a clinical and demographic perspective, patients with MetS were significantly older, with only 22.9% aged ≤50 years compared to 42.7% in the non-MetS group (p<0.001). Obesity was also more common, with 64.6% of MetS patients having a BMI ≥25 kg/m² versus 40.6% without (p=0.007). In addition, hypertension (65.6% vs. 34.4%, p<0.001), diabetes mellitus (64.6% vs. 22.9%, p<0.001), and dyslipidemia (59.4% vs. 28.1%, p<0.001) were markedly more prevalent in the MetS group. These results are in line with Zhao et al., who showed that older age, obesity, diabetes, and hypertension clustered more frequently among MetS-positive NSTEMI patients, contributing to poorer outcomes.[23] Roy et al. similarly reported that obesity, diabetes, and hypertension were more common in older NSTEMI patients, supporting our observations.[24] Importantly, no significant differences were found in sex distribution (72.9% vs. 81.2% male, p=0.170), smoking status (44.8% vs. 39.6%,

p=0.468), or family history of CAD (25.0% vs. 22.9%, p=0.741), suggesting that metabolic rather than inherited or lifestyle-related risks were the main differentiators in our cohort.

Biochemically, patients with MetS demonstrated a much higher prevalence of impaired fasting glucose or diabetes (87.5% vs. 55.2%, p<0.001), hypertriglyceridemia ≥150 mg/dL (79.2% vs. 38.5%, p<0.001), and reduced HDL-C (86.5% vs. 49.0%, p<0.001). In contrast, LDL-C elevation (47.9% vs. 44.8%, p=0.440) and elevated creatinine (5.2% vs. 3.1%, p=0.659) were not significantly different. These findings are consistent with Firoze et al., who demonstrated that triglyceride and HDL-C abnormalities were strongly associated with CAD severity, while LDL-C was less predictive.[25] Kul et al. similarly found that low HDL-C and high triglycerides were prominent biochemical abnormalities in NSTEMI patients with MetS, supporting our observations.[26]

Regarding the distribution of MetS components, raised waist circumference was present in 87.5% of patients with MetS compared to 39.6% without (p<0.001), raised blood pressure in 65.6% vs. 34.4% (p<0.001), raised fasting glucose in 64.6% vs. 22.9% (p<0.001), raised triglycerides in 79.2% vs. 38.5% (p<0.001), and reduced HDL-C in 86.5% vs. 49.0% (p<0.001). Among those with MetS, 56.2% fulfilled four criteria and 33.3% fulfilled all five. Zhao et al. also highlighted central obesity, hypertension, and lipid abnormalities as dominant clustering factors, consistent with our findings.[23]

Echocardiographic assessment revealed that normal LVEF (≥55%) was significantly less common in MetS patients (40.6% vs. 62.5%, p=0.009), while moderate dysfunction (30–44%) was more frequent (20.8% vs. 12.5%). Severe dysfunction (<30%) occurred only in the MetS group (1.0% vs. 0%). The mean LVEF was also significantly lower (53.0 ± 8.8 vs. 55.7 ± 7.4, p=0.022). These results mirror those of Zhao et al., who showed lower mean LVEF in MetS-positive NSTEMI patients (23). Loutfi et al. further demonstrated that even subtle LV dysfunction is more frequently detected in high-risk NSTEMI patients, while Karakurt et al. confirmed that MetS predisposes to impaired ventricular function, even in patients with preserved EF (27,28).

Our angiographic data demonstrated a more severe CAD burden among MetS patients, with triple vessel disease significantly more frequent (42.7% vs. 15.6%, p<0.001), and single-vessel disease more common in non-MetS (45.8% vs. 21.9%, p<0.001). The mean vessel score was higher in the MetS group (2.12 ± 0.89 vs. 1.66 ± 0.76, p<0.001), along with greater TSS (9.26 ± 4.29 vs. 6.06 ± 3.07, p<0.001) and ES (53.7 ± 18.1 vs. 39.1 ± 17.6, p<0.001). Severe stenosis (TSS ≥16) occurred more often in MetS (9.4% vs. 1.0%, p=0.009), as did severe extension (ES ≥50%; 46.9% vs. 24.0%, p=0.001). Widecka and Safranow and Wu et al. also confirmed that MetS is associated with more diffuse and severe coronary disease (29,30).

Correlation analysis showed that MetS score correlated positively with 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 and triglycerides correlated positively with all indices (all p<0.01), while HDL-C correlated negatively with vessel score (r=-0.228, p=0.002), TSS (r=-0.236, p<0.001), and ES (r=-0.244, p<0.001). Blood pressure showed weaker but significant associations with TSS (r=0.163, p=0.024) and ES (r=0.191, p=0.008), while fasting glucose showed no significant correlations. These results align with Zhao et al., who highlighted triglycerides, waist circumference, and HDL-C as the strongest correlates of CAD burden (23).

Finally, multivariable regression identified waist circumference (β=0.207 for TSS, p=0.005; β=0.183 for ES, p=0.002), triglycerides (β=0.164 for TSS, p=0.016; β=0.146 for ES, p=0.007), reduced HDL-C (β=-0.152 for TSS, p=0.008; β=-0.157 for ES, p=0.002), and raised blood pressure (β=0.161 for TSS, p=0.027; β=0.162 for ES, p=0.015) as independent predictors of CAD severity. Similar predictors were reported by Kul et al., Zhao et al., and Widecka and Safranow, confirming the dominant contribution of central obesity, lipid abnormalities, and hypertension.[23,26,29]

Collectively, these findings underscore the role of MetS in amplifying the clinical, biochemical, echocardiographic, and angiographic severity of NSTEMI. The independent contributions of waist circumference, triglycerides, low HDL-C, and blood pressure suggest that targeted metabolic interventions may play a crucial role in risk stratification and management of this high-risk population.

Limitations of The Study

The study was carried out in one hospital with a limited sample size. Therefore, the findings may not reflect the entire community.

IV. CONCLUSION

In this study, NSTEMI patients with metabolic syndrome (MetS) exhibited significantly higher clinical, biochemical, echocardiographic, and angiographic burden compared with those without MetS. Older age, obesity, hypertension, diabetes, and dyslipidemia were more common among MetS patients, while echocardiographic assessment revealed lower mean LVEF and greater prevalence of dysfunction. Angiographic analysis showed higher rates of triple vessel disease, greater stenosis and extension scores, and more severe CAD in the MetS group. Importantly, waist circumference, raised triglycerides, reduced HDL-C, and elevated blood pressure emerged as independent predictors of CAD severity, underscoring the critical role of metabolic risk clustering in

disease progression. These findings highlight the importance of early identification and proactive management of MetS components to reduce cardiovascular risk and enhance outcomes in NSTEMI patients, especially within high-risk groups.

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