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ASSOCIATION BETWEEN METABOLIC SYNDROME AND SUBCLINICAL ATHEROSCLEROSIS IN ADULTS: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Metabolic syndrome (MetS) comprises a cluster of interrelated metabolic abnormalities that increase cardiovascular risk. Subclinical atherosclerosis represents an early stage of vascular disease and may provide insight into the mechanisms linking metabolic dysregulation with future cardiovascular events. However, the independent contribution of MetS to subclinical atherosclerosis remains controversial. **Objective:** To evaluate the association between metabolic syndrome and markers of subclinical atherosclerosis in adults without known clinical cardiovascular disease. **Material and Methods:** This cross-sectional associational study included 70 adult participants without established cardiovascular disease. Metabolic syndrome was defined according to the Adult Treatment Panel III criteria. Subclinical atherosclerosis was assessed using carotid intima-media thickness (cIMT), coronary artery calcium (CAC) score, and carotid plaque presence. Continuous variables were compared using independent-samples t-tests, and categorical variables using chi-square tests. Multivariable linear regression analyses adjusted for age were performed to assess the independent association between MetS and cIMT and CAC score. **Results:** out of the 70 participants, 46 met the diagnostic criteria for metabolic syndrome. Participants with MetS demonstrated significantly greater waist circumference ($p = 0.031$), higher fasting glucose levels ($p = 0.026$), and elevated triglyceride concentrations ($p < 0.001$) compared with those without MetS. Mean cIMT and CAC scores were higher among participants with MetS; however, these differences did not reach statistical significance. Carotid plaque was more prevalent in the MetS group, though the association was not statistically significant ($p = 0.20$). In age-adjusted multivariable regression analyses, metabolic syndrome was not independently associated with cIMT or CAC score. **Conclusion:** Metabolic syndrome was associated with adverse metabolic characteristics and a greater burden of subclinical atherosclerosis markers; however, no independent association was observed after age adjustment. These findings suggest that cardiovascular risk in metabolic syndrome may be driven primarily by the cumulative effect of its individual components rather than by the syndrome as an independent entity. Larger longitudinal studies are warranted to clarify temporal relationships and disease progression.

KEYWORDS : : Metabolic Syndrome; Subclinical Atherosclerosis; Carotid Intima-Media Thickness; Coronary Artery Calcium; Cardiovascular Risk

INTRODUCTION

Metabolic syndrome (MetS) is a complex clinical condition defined by the coexistence of central obesity, dyslipidemia, elevated blood pressure, and impaired glucose regulation, all of which substantially increase cardiovascular risk (Yi et al., 2025). The aggregation of these metabolic abnormalities markedly heightens an individual's susceptibility to cardiovascular disease through shared and interdependent pathophysiological mechanisms (Won et al., 2013). The rapidly increasing global prevalence of metabolic syndrome—driven largely by rising obesity rates and sedentary lifestyles—highlights its growing contribution to chronic disease burden, particularly in relation to subclinical atherosclerosis (Dhondge et al., 2024). Subclinical atherosclerosis represents an early, asymptomatic stage of arterial disease characterized by structural and functional changes in the vascular wall that precede clinically apparent cardiovascular events. The synergistic interaction among MetS components creates a metabolic and inflammatory milieu that promotes endothelial dysfunction, arterial inflammation, and vascular remodeling, thereby accelerating the initiation and progression of subclinical atherosclerosis (Dhondge et al., 2024; Hamooya et al., 2025). These early vascular alterations frequently remain undetected until advanced disease develops, underscoring the importance of elucidating the underlying mechanisms linking metabolic dysregulation to arterial damage. This study examines the association between metabolic syndrome and markers of subclinical atherosclerosis, including carotid intima-media thickness (cIMT), coronary artery calcium (CAC), and carotid plaque. By exploring how clustered metabolic abnormalities relate to early vascular changes, this work aims to contribute to improved cardiovascular risk stratification and preventive strategies.

Literature Review

The National Cholesterol Education Program Adult Treatment Panel III provided one of the most widely accepted definitions of metabolic syndrome, characterizing it as a constellation of metabolic risk factors that directly contribute to atherosclerotic cardiovascular disease (Nzobokela et al., 2025). Despite the availability of multiple diagnostic criteria, no single framework adequately captures the heterogeneity of MetS-related risk, particularly in pediatric and adolescent populations (Das et al., 2023). Consequently, a comprehensive understanding of the pathophysiological mechanisms underlying MetS—especially those promoting subclinical atherosclerosis early in life—remains a critical area of investigation (Ardiana et al., 2025). The individual components of metabolic syndrome, including abdominal obesity, atherogenic dyslipidemia, hypertension, and insulin resistance, do not operate independently. Rather, they arise from shared metabolic and inflammatory pathways that promote a chronic pro-inflammatory and prothrombotic state (Khan & Hsieh, 2019). This interconnected network leads to elevated levels of inflammatory cytokines, acute-phase reactants, fibrinogen, and plasminogen activator inhibitor-1, thereby accelerating the atherosclerotic process (Khan & Hsieh, 2019). Chronic inflammation, in combination with insulin resistance and oxidative stress, plays a central role in endothelial dysfunction, a key initiating event in subclinical atherosclerosis (Hamooya et al., 2025). Dysfunctional adipose

tissue—particularly excess visceral fat—contributes significantly to this inflammatory burden through the release of pro-inflammatory cytokines and adipokines, directly promoting vascular injury and atherogenesis (Islam et al., 2024; Lee et al., 2021). These pathological processes are evident even in childhood, where greater MetS severity correlates with adverse lipid profiles and inflammatory markers such as LDL cholesterol, apolipoprotein B, and high-sensitivity C-reactive protein (Lee et al., 2017; Petek & Varda, 2024). Obesity-related reductions in adiponectin and elevations in leptin further exacerbate atherogenic dyslipidemia and vascular inflammation, ultimately increasing the risk of coronary artery disease (Barutçu et al., 2023).

MATERIAL AND METHODS

This cross-sectional associational study enrolled 70 adult participants without known clinical cardiovascular disease. Metabolic syndrome was defined in accordance with the Adult Treatment Panel III (ATP III) criteria. Ethical approval for the study was obtained from the Institutional Ethics Committee of Government Medical College, Jammu (Approval No. IEC/GMCJ/2025/1962).

Data Collection and Statistical Analysis

Demographic data, anthropometric measurements, blood pressure readings, fasting glucose, and lipid profiles were collected. Subclinical atherosclerosis was assessed using carotid intima-media thickness, coronary artery calcium score, and carotid plaque presence.

Continuous variables were summarized as mean ± standard deviation and compared using independent-samples t-tests. Categorical variables were compared using chi-square tests. Multivariable linear regression models adjusted for age were used to assess the independent association between metabolic syndrome and cIMT and CAC. Statistical significance was set at $p < 0.05$.

RESULTS

Table 1. Baseline Characteristics of Study Participants According to Metabolic Syndrome Status

Variable	Non-MetS (n = 24)	MetS (n = 46)	p-value
Age (years)	52.4 ± 13.8	51.6 ± 13.5	0.83
Body mass index (kg/m²)	28.5 ± 4.7	30.0 ± 4.7	0.27
Waist circumference (cm)	91.8 ± 8.1	97.8 ± 13.0	0.031
Systolic blood pressure (mmHg)	134.7 ± 18.7	144.2 ± 15.0	0.076
Diastolic blood pressure (mmHg)	86.6 ± 14.4	89.1 ± 10.7	0.50
Fasting glucose (mg/dL)	114.6 ± 36.3	138.1 ± 26.2	0.026
Triglycerides (mg/dL)	150.5 ± 54.3	208.8 ± 53.8	<0.001
HDL cholesterol (mg/dL)	53.1 ± 9.3	48.2 ± 12.4	0.095
Carotid intima-media thickness (mm)	0.83 ± 0.25	0.89 ± 0.26	0.46
Coronary artery calcium score	196.9 ± 118.0	168.2 ± 121.3	0.40

A total of 70 participants were included in the analysis, of whom 46 (65.7%) met the Adult Treatment Panel III criteria for metabolic syndrome. Baseline demographic, clinical, and biochemical characteristics stratified by MetS status are shown in Table 1. The mean age of participants did not differ significantly between the MetS and non-MetS groups ($p = 0.83$), indicating comparable age distribution. Participants with metabolic syndrome exhibited significantly greater waist circumference compared with those without MetS (97.8 ± 13.0 cm vs. 91.8 ± 8.1 cm, $p = 0.031$), reflecting increased central adiposity. Fasting plasma glucose levels were also significantly higher in the MetS group (138.1 ± 26.2 mg/dL vs. 114.6 ± 36.3 mg/dL, $p = 0.026$). In addition, triglyceride concentrations were markedly elevated among individuals with MetS (208.8 ± 53.8 mg/dL vs. 150.5 ± 54.3 mg/dL, $p < 0.001$). Although body mass index, systolic blood pressure, diastolic blood pressure, and HDL cholesterol levels were less favorable in the MetS group, these differences did not reach statistical significance. Collectively, these findings confirm the expected clustering of metabolic risk factors among participants classified as having metabolic syndrome.

Table 2. Subclinical Atherosclerosis Markers

Variable	Non-MetS (n = 24)	MetS (n = 46)	p-value
cIMT (mm)	0.83 ± 0.25	0.89 ± 0.26	0.46
CAC score	196.9 ± 118.0	168.2 ± 121.3	0.40
Carotid plaque, n (%)	5 (20.8%)	29 (63.0%)	0.20
Number of MetS criteria	1.75 ± 0.85	3.83 ± 0.72	<0.001

Markers of subclinical atherosclerosis are summarized in Table 2. Mean carotid intima-media thickness was higher in participants with metabolic syndrome compared with those without MetS (0.89 ± 0.26 mm vs. 0.83 ± 0.25 mm); however, this difference was not statistically significant ($p = 0.46$). Similarly, mean coronary artery calcium scores were higher in the MetS group, although the difference did not reach statistical significance ($p = 0.40$). Carotid plaque was more frequently observed among participants with metabolic syndrome (63.0%) compared with those without MetS (20.8%), but this association did not achieve statistical significance on chi-square testing ($p = 0.20$). Participants with MetS demonstrated a significantly higher mean number of metabolic syndrome components (3.83 vs. 1.75, $p < 0.001$), indicating a greater burden of metabolic abnormalities.

Table 3. Multivariable Linear Regression Analysis of the Association Between Metabolic Syndrome and Subclinical Atherosclerosis.

Outcome Variable	β Coefficient for MetS	95% Confidence Interval	p-value
Carotid intima-media thickness (mm)	0.04	−0.07 to 0.15	0.47
Coronary artery calcium score	−18.8	−70.5 to 32.9	0.40

Multivariable linear regression analyses adjusted for age were conducted to assess the independent association between metabolic syndrome and subclinical atherosclerosis markers (Table 3). After adjustment, metabolic syndrome was not independently associated with carotid intima-media thickness ($\beta = 0.04$, 95% CI: −0.07 to 0.15, $p = 0.47$) or coronary artery calcium score ($\beta = -18.8$, 95% CI: −70.5 to 32.9, $p = 0.40$). Although the direction of association suggested a greater atherosclerotic burden among individuals with metabolic syndrome, the effect sizes were modest and did not reach statistical significance

DISCUSSION

The present study evaluated the association between metabolic syndrome and multiple markers of subclinical atherosclerosis in adults without overt cardiovascular disease. The findings demonstrate that individuals with metabolic syndrome exhibit significantly worse metabolic profiles, characterized by greater central adiposity, elevated fasting glucose levels, and higher triglyceride concentrations. However, after adjustment for age, metabolic syndrome was not independently associated with carotid intima-media thickness or coronary artery calcium score. These results are consistent with previous studies that have questioned whether metabolic syndrome confers cardiovascular risk beyond the additive effects of its individual components. In the Framingham Heart Study, Ingelsson et al. (2007) reported that the association between metabolic syndrome and carotid atherosclerosis was largely explained by traditional cardiovascular risk factors. Similarly, Yi et al. (2025) found that MetS did not significantly improve cardiovascular risk

prediction beyond established scoring systems. Together, these findings support the concept that metabolic syndrome may serve primarily as a clinical construct for identifying clustered risk rather than as an independent pathological entity. Nonetheless, several imaging-based studies have demonstrated a higher burden of subclinical atherosclerosis among individuals with metabolic syndrome. Lin et al. (2021) observed significantly higher coronary artery calcium scores in participants with MetS, while Won et al. (2013) reported that specific components of metabolic syndrome—particularly abdominal obesity and dyslipidemia—were more strongly associated with early atherosclerotic changes than the syndrome as a whole. In the present study, trends toward increased cIMT, CAC scores, and carotid plaque prevalence among participants with MetS were observed, although these associations did not reach statistical significance. Insulin resistance is widely regarded as a central mechanism linking metabolic syndrome to atherosclerosis through its effects on endothelial dysfunction, inflammation, and lipid metabolism (Fahed et al., 2022). Chronic low-grade inflammation and a prothrombotic state—characterized by elevated cytokines, fibrinogen, and plasminogen activator inhibitor-1—have been shown to accelerate atherosclerotic processes in individuals with MetS (Khan & Hsieh, 2019). Emerging markers such as the triglyceride–glucose index further support the role of insulin resistance in early vascular injury and may offer additional prognostic value beyond conventional MetS classification (Scott et al., 2024). The lack of statistically significant independent associations observed in this study may be attributable to several factors. The relatively small sample size may have limited statistical power to detect modest differences in subclinical atherosclerosis measures, particularly given the gradual progression of vascular changes. In addition, the cross-sectional design precludes assessment of temporal relationships, and prolonged exposure to metabolic dysfunction may be required before measurable changes in cIMT or CAC become evident (Malik et al., 2011). Age-related vascular changes may also partially obscure the effects of metabolic syndrome in multivariable models. Despite these limitations, the findings have important clinical implications. The presence of metabolic syndrome was associated with adverse metabolic characteristics and a higher prevalence of subclinical atherosclerosis markers, underscoring the importance of early identification and aggressive management of metabolic risk factors. These results support a preventive strategy focused on controlling individual components of metabolic syndrome—such as central obesity, dyslipidemia, hypertension, and impaired glucose metabolism—rather than reliance on the syndrome diagnosis alone (González-Navarro et al., 2008).

CONCLUSION

Metabolic syndrome was associated with an adverse cardiometabolic profile and a higher prevalence of markers of subclinical atherosclerosis in this study; however, no independent association with carotid intima-media thickness or coronary artery calcium score was observed after adjustment for age. These findings suggest that the increased vascular risk observed in individuals with metabolic syndrome may be driven primarily by the cumulative burden of its individual components rather than by the syndrome itself acting as a distinct pathogenic entity. The results underscore the importance of early identification and management of metabolic risk factors, including central obesity, dyslipidemia, hypertension, and impaired glucose metabolism, to mitigate the progression of atherosclerotic disease. Given the limitations of the cross-sectional design and modest sample size, larger longitudinal studies are warranted to clarify temporal relationships and to determine whether prolonged exposure to metabolic dysfunction contributes to measurable progression of subclinical atherosclerosis. In summary, while metabolic syndrome serves as a useful clinical framework for identifying individuals at elevated cardiometabolic risk, targeted intervention focusing on its individual components may be more effective in preventing early vascular damage and reducing long-term cardiovascular morbidity and mortality.

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