Section: Miscellaneous

Original Research Article

ROLE CT CORONARY ANGIOGRAPHY IN EVALUATING SUBCLINICAL ATHEROSCLEROSIS IN HIGH RISK METABOLIC SYNDROME PATIENT

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ABSTRACT

Background: Metabolic syndrome (MetS) is a major driver of premature atherosclerotic cardiovascular disease, particularly in South Asian populations. Conventional risk scores often fail to detect early or subclinical coronary disease in high-risk individuals. Coronary CT angiography (CCTA) enables direct visualisation and quantification of coronary plaque, including high-risk morphology. This study evaluates the role of CCTA in detecting subclinical atherosclerosis among adults with high-risk MetS. **Materials and Methods:** This cross-sectional observational study included 100 adults diagnosed with MetS (NCEP-ATP III criteria) and classified as high-risk based on ≥4 MetS components, long-standing MetS, or additional atherosclerotic risk enhancers. All participants underwent contrast-enhanced CCTA on a ≥64-slice CT scanner. Plaque presence, type, burden (segment involvement score, SIS), stenosis severity, high-risk plaque features, and coronary artery calcium score (CACS) were assessed. Metabolic parameters were correlated with plaque burden. **Result:** Subclinical coronary atherosclerosis was detected in 72% of patients. Plaque prevalence rose significantly with MetS severity (40.9% in mild vs 92.5% in severe; $p < 0.001$). Calcified (22.7% vs 60%; $p = 0.004$) and non-calcified plaques (9.1% vs 42.5%; $p = 0.008$) increased across severity categories. High-risk plaque features were present in 32.5% of severe MetS subjects compared with 4.5% in mild ($p = 0.003$). Median CACS (42 to 152; $p < 0.001$) and mean SIS (0.9 ± 0.7 to 3.0 ± 1.4; $p < 0.001$) also increased markedly. Luminal stenosis ≥50% occurred in 40% of severe MetS patients but was absent in mild cases. Plaque burden showed significant correlations with fasting glucose ($r = 0.42$), triglycerides ($r = 0.38$), waist circumference ($r = 0.36$), BMI ($r = 0.29$), and HDL ($r = –0.31$). MetS severity score demonstrated the strongest association ($\rho = 0.55$; $p < 0.001$). **Conclusion:** Subclinical coronary atherosclerosis is highly prevalent in high-risk MetS patients, with plaque burden and high-risk features rising sharply with metabolic severity. CCTA offers substantial value in early detection and risk stratification, surpassing traditional clinical and biochemical markers. Incorporating CCTA into evaluation algorithms for high-risk MetS populations may facilitate earlier preventive interventions. Prospective studies are required to determine the impact of CCTA-guided management on long-term cardiovascular outcomes.
Keywords: Metabolic syndrome, CT coronary angiography, subclinical atherosclerosis, coronary plaque, high-risk plaque, coronary calcium score.

INTRODUCTION

Metabolic syndrome (MetS)—characterised by central obesity, insulin resistance, dyslipidaemia, hypertension and a pro-inflammatory/pro-thrombotic state—has emerged as a major global driver of atherosclerotic cardiovascular disease (ASCVD).[1-3] Individuals with MetS have a substantially elevated risk of overt coronary artery disease (CAD) and myocardial infarction compared with those without MetS, even after adjustment for conventional cardiovascular risk factors.[4] The atherosclerotic process in MetS remains silent and subclinical for

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many years before progressing to symptomatic disease.[5] Traditional cardiovascular risk prediction tools, such as the Framingham Risk Score and SCORE system offer useful population-level estimates but are limited in detecting early or subclinical atherosclerosis in high-risk individuals.[6-7] Consequently, non-invasive imaging modalities have gained prominence for identifying subclinical coronary disease before the onset of symptoms, thereby enabling improved patient selection for early preventive therapy.[7-9]

Among available imaging options, computed tomography (CT)–based techniques have become central to the non-invasive evaluation of coronary atherosclerosis. Coronary CT angiography (CCTA), in particular, allows direct *in vivo* visualization and quantification of coronary plaque—including calcified, non-calcified, and mixed components—and provides detailed assessments of plaque burden, luminal stenosis, positive remodelling, and high-risk plaque characteristics.[10-12] These capabilities extend beyond coronary artery calcium (CAC) scoring alone, which, while useful in risk prediction, reflects only the calcified component of atherosclerosis and may underestimate early disease.[13-15]

Evidence from asymptomatic and intermediate-risk populations suggests that even non-obstructive plaque detected by CCTA is significantly associated with future cardiovascular events, independent of traditional risk factors.[16-17] Thus, CCTA offers a more sensitive method for early detection of subclinical coronary atherosclerosis and may refine risk stratification in high-risk individuals.

In MetS, the pro-atherogenic environment created by insulin resistance, dyslipidaemia, endothelial dysfunction, and chronic low-grade inflammation accelerates the progression of coronary plaque, even in the absence of symptoms.[18-19] While previous research has demonstrated associations between MetS components and surrogate markers such as carotid intima–media thickness and coronary calcium score,[7,13] there remains a relative paucity of data specifically evaluating the utility of CCTA in characterising subclinical coronary atherosclerosis in MetS patients.[20-21]

Given the rising prevalence of MetS in South Asia and other developing regions, coupled with the significant burden of premature CAD,[1-3,22] early detection of subclinical coronary atherosclerosis is crucial for guiding individualised preventive strategies. CCTA may offer significant clinical value by identifying silent coronary plaque, quantifying total plaque burden, and revealing high-risk plaque morphology in patients with MetS who might otherwise be underestimated by conventional risk scores. Therefore, the present study aims to evaluate the role of CT coronary angiography in detecting and characterising subclinical atherosclerosis in adults with high-risk metabolic syndrome.

MATERIALS AND METHODS

This was a hospital-based cross-sectional observational study conducted in the Department of Radiology and medicine, Department of medicine, at a tertiary care teaching hospital. A total of 100 consecutive adults with metabolic syndrome (MetS) who fulfilled the inclusion criteria were enrolled.

Eligibility Criteria

Inclusion Criteria

1. Adults aged **≥ 18 years** diagnosed with **Metabolic Syndrome** as per **NCEP-ATP III** criteria (presence of ≥3 of the following):
   - Abdominal obesity
   - Elevated fasting blood glucose
   - Elevated triglycerides
   - Reduced HDL-cholesterol
   - Elevated blood pressure
2. Patients categorized as **high-risk**, defined as:
   - Presence of ≥4 MetS components, **or**
   - Long-standing MetS (≥5 years), **or**
   - Additional atherosclerotic risk enhancers (smoking, family history of premature CAD, chronic inflammatory state)
3. Patients who underwent **contrast-enhanced coronary CT angiography** (CCTA) during the study period.

Exclusion Criteria

1. Known coronary artery disease (prior MI, PCI, CABG).
2. Symptomatic patients with typical angina.
3. Renal impairment (eGFR < 60 mL/min/1.73 m²).
4. Contrast allergy or contraindication to iodinated contrast.
5. Pregnancy or lactation.
6. Inadequate CT image quality (motion artefact, arrhythmia).

Clinical and Laboratory Assessment

All participants underwent a detailed clinical evaluation including:

- Age, sex, anthropometry (BMI, waist circumference)
- Blood pressure measurement
- Lifestyle factors (smoking, alcohol, activity levels)

Biochemical parameters performed after overnight fasting included:

- Fasting blood glucose
- Lipid profile—LDL, HDL, triglycerides
- HbA1c
- Renal and liver function tests

MetS severity categories were documented based on number of components and risk enhancers.

CT Coronary Angiography Protocol

Scanner and Acquisition

CCTA was performed using a **64-slice or higher multi-detector CT scanner**.

Scan parameters included:

- Retrospective or prospective ECG-gated acquisition
- Tube voltage: 100–120 kVp

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- Tube current modulation applied
- Slice thickness: 0.6–0.75 mm
- Reconstruction in axial, MPR, curved-MPR, and 3D volume-rendered formats

Contrast Administration

- 60–80 mL of non-ionic iodinated contrast injected at 4–5 mL/s
- Bolus tracking used to initiate acquisition
- Beta-blockers administered when required to achieve HR <65 bpm
- Sublingual nitroglycerin given unless contraindicated

Image Analysis

All images were evaluated independently by **two experienced radiologists** blinded to clinical details. Discrepancies were resolved by consensus.

Parameters Assessed

1. **Presence of coronary plaque** (yes/no).
2. **Plaque characterization**:
   - Calcified
   - Non-calcified
   - Mixed
3. **Segment involvement score (SIS)** – number of coronary segments with plaque.
4. **Severity of luminal stenosis**:
   - <25%
   - 25–49%
   - 50–69%
   - ≥70%
5. **High-risk plaque features**:
   - Positive remodelling
   - Low-attenuation plaque
   - Napkin-ring sign
   - Spotty calcification
6. **Coronary Artery Calcium Score (CACS)** measured using Agatston method.

Outcome Measures

Primary outcome:

- **Prevalence and extent of subclinical coronary atherosclerosis** in MetS.

Secondary outcomes:

- Association between MetS severity and:
  - Plaque presence
  - Plaque type
  - Plaque burden
  - High-risk features
  - CACS and stenosis severity

Statistical Analysis

Data were analysed using **SPSS version 25.**

- **Categorical variables**: expressed as frequencies and percentages.
- **Continuous variables**: mean ± SD or median (IQR).
- Group comparisons performed using:
  - **Chi-square / Fisher's exact test** for categorical variables
  - **Independent t-test / ANOVA** for normally distributed variables
  - **Mann–Whitney U / Kruskal–Wallis test** for skewed variables
- **Correlation analysis** using Pearson or Spearman coefficients.
- A **p-value < 0.05** was considered statistically significant

RESULTS

The demographic characteristics of the study participants, as shown in **Table 1**, demonstrate that the mean age was 52.6 ± 9.4 years, with a median of 53 years, indicating a predominantly middle-aged population. Age increased significantly with rising metabolic syndrome severity (p = 0.004), suggesting that advancing age may contribute to worsening metabolic derangement. Waist circumference and BMI showed similar significant upward trends across severity categories (p = 0.001 and p = 0.010, respectively), reinforcing the strong influence of central adiposity on disease progression. **Table 2** presents the sex distribution, showing that although males constituted 58% of the cohort, no statistically significant relationship existed between sex and metabolic syndrome severity (p = 0.63), indicating that the severity of metabolic syndrome was not gender-dependent in this population.

Table 1: Demographic Profile of Study Population (n = 100)

Variable	Mean ± SD	Median (IQR)	Stage Comparison (MetS Severity)	Statistical Test	p-value
Age (years)	52.6 ± 9.4	53 (46–59)	Mild (n=22): 48.3 ± 7.5 Moderate (n=38): 52.4 ± 9.1 Severe (n=40): 55.8 ± 9.9	ANOVA	**0.004**
Waist Circumference (cm)	101.6 ± 9.8	100 (95–108)	Mild: 96.2 ± 8.7 Moderate: 101.3 ± 9.1 Severe: 105.8 ± 9.3	ANOVA	**0.001**
BMI (kg/m²)	29.4 ± 3.8	29.1 (27.0–31.4)	Mild: 27.6 ± 3.1 Moderate: 29.1 ± 3.4 Severe: 30.7 ± 3.7	ANOVA	**0.010**

Table 2: Sex Distribution Across Metabolic Syndrome Severity Groups

Sex	Total (n=100)	Mild (n=22)	Moderate (n=38)	Severe (n=40)	Statistical Test	p-value
Male	58 (58%)	11 (50%)	23 (60.5%)	24 (60%)	Chi-square	0.63
Female	42 (42%)	11 (50%)	15 (39.5%)	16 (40%)	—	—

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The biochemical and metabolic parameters summarised in **Table 3** reveal progressively worsening abnormalities with increasing metabolic syndrome severity. Fasting glucose and triglyceride levels rose significantly ($p < 0.001$ and $p = 0.002$), HDL levels declined markedly ($p = 0.001$), and systolic blood pressure increased steadily ($p = 0.003$), reflecting the clustering and intensification of metabolic disturbances as the syndrome becomes more severe. **Table 4** highlights the coronary CT angiography findings, where 72% of all participants exhibited subclinical coronary atherosclerosis despite being asymptomatic. A striking gradient was observed, with plaque prevalence rising from 40.9% in mild cases to 92.5% in severe metabolic syndrome ($p < 0.001$). Both calcified and non-calcified plaques demonstrated significant increases with severity ($p = 0.004$ and $p = 0.008$), while high-risk plaque features were especially notable in severe metabolic syndrome, occurring in 32.5% of this group compared with only 4.5% of mild cases ($p = 0.003$). The coronary artery calcium score and the segment involvement score also increased markedly with severity (both $p < 0.001$), indicating not only a higher frequency but a greater burden of coronary disease. Additionally, luminal stenosis ≥50% was absent in the mild group but present in 40% of severe metabolic syndrome subjects ($p < 0.001$), underscoring the silent progression of potentially obstructive coronary disease.

Table 3: Metabolic and Biochemical Parameters

Parameter	Mean ± SD	Median (IQR)	Mild	Moderate	Severe	Statistical Test	p-value
Fasting Glucose (mg/dL)	143.6 ± 32.1	138 (122–165)	124.8 ± 21.5	140.2 ± 28.7	159.3 ± 34.8	ANOVA	**<0.001**
Triglycerides (mg/dL)	209.4 ± 51.3	198 (174–236)	184.2 ± 41.5	204.9 ± 45.1	228.6 ± 56.3	ANOVA	**0.002**
HDL (mg/dL)	38.8 ± 6.4	38 (34–43)	42.3 ± 5.2	38.7 ± 6.1	36.9 ± 6.2	ANOVA	**0.001**
Systolic BP (mmHg)	142 ± 16	140 (130–152)	133.5 ± 12.1	141.4 ± 13.7	148.0 ± 15.5	ANOVA	**0.003**

Table 4: Coronary Atherosclerosis Findings on CCTA

CCTA Parameter	Total (n=100)	Mild (n=22)	Moderate (n=38)	Severe (n=40)	Statistical Test	p-value
Any Coronary Plaque (present)	72 (72%)	9 (40.9%)	26 (68.4%)	37 (92.5%)	Chi-square	**<0.001**
Calcified Plaque	44 (44%)	5 (22.7%)	15 (39.4%)	24 (60%)	Chi-square	**0.004**
Non-calcified (soft) Plaque	28 (28%)	2 (9.1%)	9 (23.7%)	17 (42.5%)	Chi-square	**0.008**
Mixed Plaque	26 (26%)	3 (13.6%)	10 (26.3%)	13 (32.5%)	Chi-square	0.128
High-Risk Plaque Features (any)	18 (18%)	1 (4.5%)	4 (10.5%)	13 (32.5%)	Chi-square	**0.003**
Coronary Artery Calcium Score (Agatston)	118 ± 64	95 (44–178)	Mild: 42 ± 25 Moderate: 112 ± 55 Severe: 152 ± 68	Kruskal–Wallis	**<0.001**	
Segment Involvement Score (SIS)	2.1 ± 1.4	0.9 ± 0.7	1.8 ± 1.0	3.0 ± 1.4	ANOVA	**<0.001**
Luminal Stenosis ≥50%	21 (21%)	0 (0%)	5 (13.1%)	16 (40%)	Fisher's Exact	**<0.001**

Table 5 further illustrates the relationship between metabolic parameters and plaque burden, showing significant positive correlations between plaque burden and fasting glucose ($r = 0.42$), triglycerides ($r = 0.38$), BMI ($r = 0.29$), and waist circumference ($r = 0.36$), while HDL exhibited a moderate negative correlation ($r = –0.31$). The strongest association was observed between the metabolic syndrome severity score and plaque burden ($\rho = 0.55$, $p < 0.001$), demonstrating that worsening metabolic derangement corresponded with increased coronary involvement. Collectively, the findings from Tables 1 through 5 clearly indicate that metabolic syndrome severity is strongly associated with both the presence and the extent of subclinical coronary atherosclerosis, highlighting CT coronary angiography as an effective tool for early detection and risk stratification in high-risk metabolic syndrome patients.

Table 5: Correlation Between Metabolic Parameters and Coronary Atherosclerosis

Metabolic Parameter	Correlation with Plaque Burden (SIS)	p-value
Fasting Glucose	**r = 0.42**	**<0.001**
Triglycerides	**r = 0.38**	**0.001**
HDL	**r = –0.31**	**0.003**
BMI	**r = 0.29**	**0.004**
Waist Circumference	**r = 0.36**	**0.001**
MetS Severity Score	**r = 0.55**	**<0.001**

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DISCUSSION

In the present cross-sectional study of 100 high-risk metabolic syndrome (MetS) patients, we found that 72% had detectable coronary plaque by CT coronary angiography (CCTA) despite being asymptomatic. Notably, plaque prevalence rose from 40.9% in the mild MetS group to 92.5% in the severe group ($p < 0.001$). Calcified plaques increased from 22.7% to 60% ($p = 0.004$), non-calcified plaques from 9.1% to 42.5% ($p = 0.008$), and high-risk plaque features were present in 32.5% of the severe group (versus 4.5% in the mild; $p = 0.003$). The coronary artery calcium score (CACS) and segment involvement score (SIS) also climbed significantly with MetS severity (median CACS from 42 to 152, $p < 0.001$; mean SIS 0.9 ±0.7 to 3.0 ± 1.4, $p < 0.001$). Metabolic parameters such as fasting glucose ($r = 0.42$), triglycerides ($r = 0.38$), waist circumference ($r = 0.36$), BMI ($r = 0.29$) and HDL ($r = –0.31$) correlated significantly with plaque burden; the MetS severity score had the strongest correlation ($\rho = 0.55$, $p < 0.001$).

These findings corroborate and extend prior literature. For instance, Park et al. examined 5,213 asymptomatic individuals undergoing CCTA and found that MetS (present in ~39% of the cohort) was an independent predictor of significant coronary artery disease (CAD) (OR ~1.99) and of events (RR ~1.67) during median follow-up of 28 months.[22] Their results echo our observation that MetS is associated with a high prevalence of silent coronary atherosclerosis; however, they reported plaque prevalence rather in terms of significant stenosis rather than burden metrics, and their population was much larger. In another study by Kim et al., MetS was shown to predict progression of CACS, coronary stenosis and vulnerable plaque on CCTA (HR ~1.45 for stenosis/plaques after adjustment) in 2,426 asymptomatic subjects. Our results add to these by quantifying multiple plaque types, burden scores (SIS), high-risk features, and by stratifying according to severity of MetS, thus revealing a clear dose-response relationship.[24]

The high prevalence of non-calcified plaque (42.5% in severe MetS) and high-risk features in our cohort is of particular significance, because non-calcified and positively remodeled plaques are known to carry higher risk of rupture and acute events than calcified, stable plaques. Prior smaller studies in morbid obesity found among BMI > 40 kg/m² patients that majority of plaques on CCTA were non- calcified (75%) and low-attenuation, indicating early and vulnerable disease. That our severe MetS group shows similar patterns supports the notion that MetS fosters early phenotypes of high- risk coronary atherosclerosis even before symptoms appear.[25]

The observed correlations between metabolic parameters and plaque burden (fasting glucose, triglycerides, waist circumference, BMI, and HDL) align with the mechanistic understanding of MetS: insulin resistance, visceral adiposity, dyslipidaemia, hypertension and low- grade inflammation all promote endothelial dysfunction, vascular remodelling and plaque formation. Several prior studies link number of MetS components with increasing CACS and plaque burden; for example, Kim et al. found a graded increase in CAD progression with number of MetS components. Our data replicate this graded relationship and show that the combined MetS severity score correlates more strongly ($\rho = 0.55$) than single parameters.[26]

A strength of our study is that it quantifies multiple dimensions of subclinical coronary disease (plaque types, burden, high-risk features) in a well-defined high- risk MetS cohort, and shows a robust dose-response relationship with clinical severity of metabolic derangement. It thereby adds value beyond prior studies which either reported large asymptomatic cohorts without MetS severity stratification or progression studies without full burden characterization.

Limitations: First, this is a cross-sectional design; hence, causal inferences or prognostic outcomes cannot be proven. Long- term follow-up would be needed to assess whether the high prevalence and burden of plaque observed translates into future cardiovascular events. Second, the sample size of 100, though adequate for correlations, is modest compared to large screening cohorts, and may limit generalisability. Third, radiation exposure, contrast nephropathy risk and cost limit routine use of CCTA as a screening tool; hence, the value of imaging must be balanced with risk- benefit and cost-effectiveness considerations. Fourth, despite stratification by severity, residual confounding from other risk factors (e.g., smoking, family history) cannot be excluded.

CONCLUSION

In conclusion, our study demonstrates that in high-risk MetS patients, subclinical coronary atherosclerosis is highly prevalent, and its burden and high- risk features increase significantly with metabolic severity. Combined with published evidence, these data suggest that CCTA may serve as a powerful imaging adjunct for early detection and risk stratification in MetS, enabling more targeted preventive interventions. Future prospective studies are warranted to assess whether CCTA-guided management translates into improved cardiovascular outcomes in this population.

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