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Research Article

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Correlation of Coronary Artery Calcium Score with Vessel-Specific Stenosis & Plaque Burden

Shaily Panwar¹, Preeti Tiwari¹ & Dhruv Singh^{1*}

¹Department of Radiology, Maharani Laxmi Bai Medical College, Jhansi, India

HIGHLIGHTS

- Vessel-wise calcium distribution assessed
- RCA showed highest calcium burden
- CCTA evaluated stenosis severity
- Plaque burden correlated with calcium
- CCTA complemented calcium scoring

Key Words:

Coronary artery calcium score
CCTA
Agatston score
CAD-RADS
Coronary stenosis

ABSTRACT

Introduction: Coronary artery calcium scoring (CACS) quantifies calcified atherosclerotic burden, whereas coronary CT angiography (CCTA) directly evaluates coronary lumen, stenosis and plaque extent. Because coronary atherosclerosis is heterogeneous across individual vessels, vessel-wise comparison of calcium burden with CCTA-defined stenosis and CAD-RADS plaque burden may provide a more clinically meaningful assessment than total calcium score alone. **Aim & Objective:** To evaluate the vessel-wise distribution of coronary artery calcium, compare it with vessel-specific stenosis on CCTA, and assess the relationship of calcium burden and stenosis severity with CAD-RADS plaque burden categories in clinically suspected coronary artery disease patients. **Material & Methods:** This prospective observational study included 196 clinically suspected CAD patients evaluated in the Department of Radio-Diagnosis, Maharani Laxmi Bai Medical College, Jhansi, from March 2025 to February 2026. All participants underwent non-contrast ECG-gated calcium scoring followed by CCTA. Agatston scores and stenosis severity were assessed for the LMA, LAD, LCX, RCA and PDA, and CCTA plaque burden was categorized as P1, P2 or P3. **Results:** CAC was most frequently detected in the RCA (73.5%), followed by the LAD (59.7%), LMA (47.4%), LCX (33.2%) and PDA (13.3%); mean vessel-specific CAC was highest in the RCA (25.6 ± 46.6) and LAD (25.3 ± 42.8). On CCTA, any stenosis was present in all LAD and RCA assessments, and the RCA had the highest mean stenosis ($41.6 \pm 25.3\%$). Severe stenosis occurred in 23.5% of LMA and 19.4% of RCA assessments. P1, P2 and P3 plaque burden accounted for 51.5%, 16.8% and 31.6% of patients. Mean total CAC was markedly higher in P2 (148.7 ± 115.8) and P3 (140.5 ± 174.7) than in P1 (19.2 ± 21.3), while severe stenosis occurred in 22.8%, 51.5% and 53.2%, respectively. **Conclusion:** Coronary calcium and stenotic burden showed marked vessel-wise heterogeneity, with the greatest calcium and luminal disease concentrated in the RCA and LAD. Higher CAD-RADS plaque burden categories were accompanied by substantially greater calcium burden than P1 and a higher frequency of severe stenosis. CACS therefore provides useful information on calcified disease extent, but CCTA remains essential for defining vessel-specific luminal narrowing and overall plaque burden.



* Corresponding Author: Dhruv Singh, e-mail: drdhruvsmeena84@gmail.com

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INTRODUCTION

Coronary artery disease (CAD) is one of the principal manifestations of systemic atherosclerosis & develops through progressive accumulation of lipid-rich, fibrous, & calcified plaques with in the walls of the epicardial coronary arteries [1]. In clinically suspected patients presenting with symptoms such as chest pain, exertional dyspnea, or other features suggestive of myocardial ischemia, accurate non-invasive characterization of coronary atherosclerosis is important for determining both the anatomical extent and severity of disease. Contemporary cardiac computed tomography offers complementary assessment through coronary artery calcium scoring (CACS) and coronary computed tomography angiography (CCTA) [2]. Calcium scoring detects and quantifies calcified coronary atherosclerosis without intravenous contrast, whereas CCTA provides direct visualization of the coronary lumen, arterial wall, plaque morphology, and degree of luminal stenosis. The Agatston score remains the most widely used method for quantification of coronary artery calcium and serves as an established marker of overall calcified atherosclerotic burden [3].

Coronary calcium scoring quantifies calcified plaque burden throughout the coronary arterial tree, but its anatomical distribution may provide clinical value beyond the total Agatston score. Calcium may involve the LM, LAD, LCx, RCA, or branches, producing patterns ranging from isolated single-vessel to diffuse multivessel disease [4]. Patients with similar total scores may differ substantially in disease distribution. Greater numbers of calcified coronary vessels have been associated with progressively higher mortality risk, independent of total calcium burden and conventional cardiovascular risk factors [5].

Vessel-wise assessment is particularly relevant because atherosclerotic disease does not carry identical anatomical significance in every coronary segment. Involvement of the LM or proximal

LAD may represent a substantial burden of disease because these vessels supply large territories of myocardium. Conversely, calcium distributed across the LAD, LCx, and RCA indicates a more diffuse pattern of coronary atherosclerosis [6]. Evaluation of single-vessel, double-vessel, triple-vessel, & left-main involvement therefore allows calcium scoring to be correlated more directly with the anatomical findings observed on CCTA. The regional distribution of coronary calcium has also demonstrated incremental prognostic value, with left-main calcification and increasing numbers of calcified vessels associated with adverse outcomes independently of total calcium burden [5].

Despite its clinical usefulness, coronary calcium scoring represents only the calcified component of the atherosclerotic process. A zero or low calcium score does not completely exclude coronary atherosclerosis because early lesions may predominantly consist of non-calcified or mixed plaques [7]. CCTA addresses this limitation by enabling direct visualization of calcified, partially calcified, and non-calcified plaques while simultaneously measuring the severity of coronary luminal narrowing [8]. In the SCOT-HEART population, patients with a calcium score of zero could still demonstrate non-obstructive disease, obstructive disease, and adverse plaque phenotypes on CCTA, demonstrating that CACS and CCTA provide related but non-identical information regarding coronary disease [9].

CCTA enables comprehensive vessel-by-vessel assessment of coronary atherosclerosis, including plaque presence, composition, location, and degree of luminal stenosis. These findings can be correlated with coronary artery calcium score (CACS) to evaluate plaque burden and stenosis severity [10]. However, the relationship is not always linear, as severe calcification may occur without critical stenosis, while significant narrowing may result from non-calcified plaque. Low-attenuation non-calcified plaque on CCTA is particularly

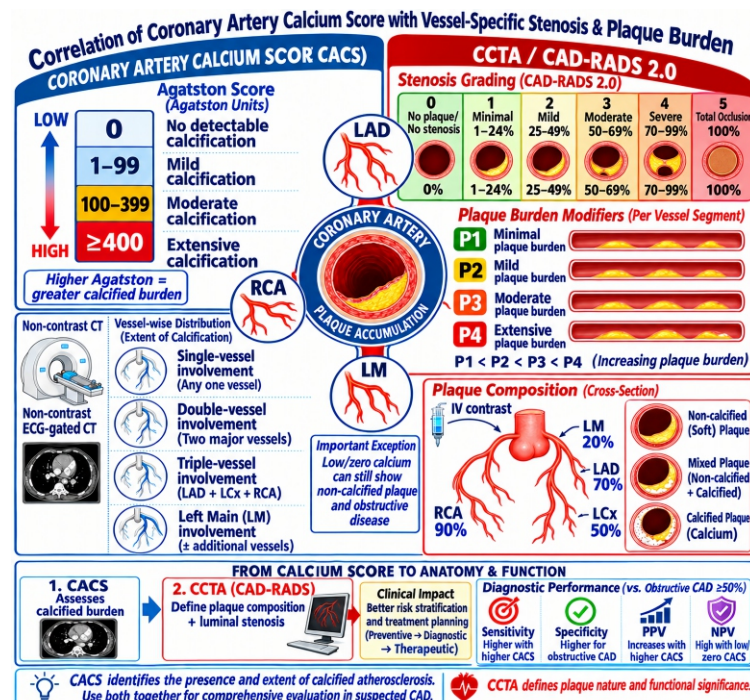


Figure 1. Correlation of coronary artery calcium score with vessel-specific stenosis and plaque burden on coronary CT angiography (CCTA) using CAD-RADS 2.0.

important because it is associated with higher stenosis severity and future myocardial infarction risk [11]. CAD-RADS provides a standardized framework for interpreting and reporting coronary CTA findings. CAD-RADS 2.0 classifies coronary artery disease primarily according to the most severe luminal stenosis detected on CCTA [12]. CAD-RADS 0 indicates absence of plaque or stenosis, whereas increasing categories represent progressively greater disease severity, with CAD-RADS 5 indicating total coronary occlusion. This system improves consistency, reproducibility, and communication of CCTA findings while facilitating anatomical risk stratification [12].

An important advancement in CAD-RADS 2.0 is the greater emphasis placed on overall coronary plaque burden rather than relying exclusively on the single most severe stenotic lesion. Plaque burden modifiers from P1 to P4 describe increasing amounts of coronary atherosclerotic plaque and allow disease extent to be incorporated into the standardized CCTA report [12]. This distinction is clinically important because two patients with the same maximum percentage stenosis can have substantially different total atherosclerotic burdens depending on the number of vessels and segments involved. Quantitative CCTA studies have further demonstrated that total and non-calcified plaque burden can provide important prognostic information beyond calcium scoring alone [13].

Correlating coronary artery calcium score (CACS) with CCTA can help determine how accurately calcified plaque burden reflects anatomical coronary artery disease. Assessment of CACS in relation to LM, LAD, LCx and RCA involvement, number of diseased vessels, stenosis severity, CAD-RADS category, and overall plaque burden may demonstrate whether higher calcium scores correspond to more extensive disease. This may support CACS as a useful predictor of CCTA-defined coronary atherosclerotic severity [8]. **Figure 1.** Correlation of coronary artery calcium score (CACS) with vessel-specific stenosis and plaque burden on coronary CT angiography (CCTA) using CAD-RADS 2.0. CACS quantifies calcified atherosclerotic burden, whereas CCTA characterizes plaque composition, luminal narrowing, and coronary disease severity. The aim of this study was to determine the predictive value of coronary artery calcium scoring in relation to CT angiography findings in clinically suspected coronary artery disease. The objectives were to assess correlation with stenosis severity, determine diagnostic accuracy, sensitivity, specificity, PPV and NPV, stratify patients by calcium score, and evaluate its potential role as an initial screening tool.

MATERIALS & METHODS

This prospective observational study was conducted in the Department of Radio-Diagnosis, Maharani Laxmi Bai Medical College, Jhansi, from March 2025 to February 2026, including 196 adults with suspected coronary artery disease undergoing calcium scoring and CCTA. Non-contrast ECG-gated CT quantified coronary calcium using the Agatston method, while contrast-enhanced ECG-gated CCTA assessed vessel-specific stenosis and plaque burden. Calcium scores and stenosis were

categorized according to predefined severity grades, and overall plaque burden was classified using CAD-RADS P1-P3. CCTA was interpreted by an experienced radiologist blinded to CAC scores. Descriptive statistics were applied, with ethical approval and informed consent obtained before enrollment.

RESULTS

A total of 196 clinically suspected CAD patients underwent both coronary calcium scoring and CCTA. In accordance with the selected analysis, the results are presented only for vessel-wise Agatston score distribution, vessel-wise CCTA stenosis, and the association of CAC and stenosis with CAD-RADS plaque burden. Vessel-wise CAC distribution was highest in the RCA, where calcification was present in 73.5% of patients, followed by the LAD (59.7%) and LMA (47.4%) (**Table 1**). The LCX showed calcification in 33.2%, whereas the PDA was least affected at 13.3%. Mean CAC burden was highest in the RCA (25.6 ± 46.6) and LAD (25.3 ± 42.8). No individual vessel demonstrated a severe Agatston score of ≥ 300 , indicating that severe total CAC, when present, reflected the cumulative contribution of multiple coronary vessels rather than a single vessel score reaching this threshold (**Figure 2a & 2b**).

CCTA demonstrated vessel-specific differences in luminal narrowing. Any degree of stenosis was recorded in all LAD and RCA assessments, whereas LMA, LCX and PDA involvement was 41.8%, 68.9% and 27.0%, respectively (**Table 2**). The RCA had the highest mean stenosis ($41.6 \pm 25.3\%$) and median stenosis (48.0%). Severe stenosis was most frequent in the LMA (23.5%) and RCA (19.4%), while the LAD was characterized mainly by minimal (53.1%) and moderate (39.3%) stenosis. The LCX and PDA showed no moderate or severe stenosis in the selected dataset (**Figure 3a & 3b**).

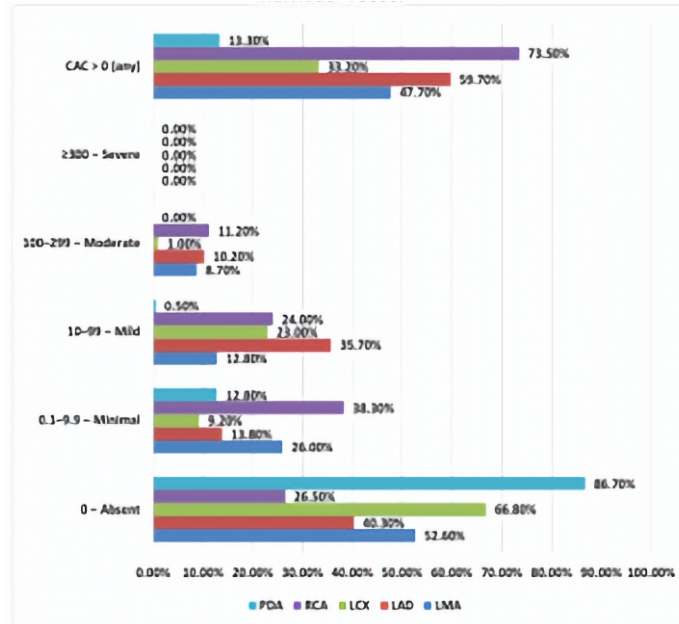
Low plaque burden (P1) was the most frequent CAD-RADS plaque burden category (51.5%), followed by P3 (31.6%) and P2 (16.8%). Mean total CAC was substantially higher in P2 (148.7 ± 115.8) and P3 (140.5 ± 174.7) than in P1 (19.2 ± 21.3), although the mean CAC value did not increase monotonically from P2 to P3. Severe stenosis was present in 22.8% of P1, 51.5% of P2 and 53.2% of P3 patients, while mild stenosis predominated in P1 (66.3%). In the higher plaque-burden groups, the RCA and LAD showed the greatest vessel-specific calcium and stenosis values, indicating concentration of more advanced disease in these vessels (**Table 3**).

DISCUSSION

The vessel-wise analysis demonstrated a distinctly non-uniform distribution of coronary atherosclerotic burden. Calcification was most frequent in the RCA (73.5%), followed by the LAD (59.7%), LMA (47.4%), LCX (33.2%) and PDA (13.3%); the highest mean CAC values were also recorded in the RCA (25.6 ± 46.6) and LAD (25.3 ± 42.8). On CCTA, the RCA showed the highest mean stenosis ($41.6 \pm 25.3\%$), while the LAD demonstrated some degree of stenosis in all patients, with 39.3% showing moderate stenosis. Severe stenosis was most frequent in the LMA (23.5%) and RCA (19.4%), indicating that the

Table 1: Coronary Artery Calcium (Agatston) Score Distribution by Individual Vessel

Agatston Category	LMA	LAD	LCX	RCA	PDA
0 - Absent	103 (52.6%)	79 (40.3%)	131 (66.8%)	52 (26.5%)	170 (86.7%)
0.1-9.9 - Minimal	51 (26.0%)	27 (13.8%)	18 (9.2%)	75 (38.3%)	25 (12.8%)
10-99 - Mild	25 (12.8%)	70 (35.7%)	45 (23.0%)	47 (24.0%)	1 (0.5%)
100-299 - Moderate	17 (8.7%)	20 (10.2%)	2 (1.0%)	22 (11.2%)	0 (0.0%)
≥300 - Severe	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
CAC >0 (any)	93 (47.4%)	117 (59.7%)	65 (33.2%)	144 (73.5%)	26 (13.3%)
Mean ± SD	19.9 ± 45.6	25.3 ± 42.8	7.7 ± 23.8	25.6 ± 46.6	0.8 ± 2.3
Median	0.00	6.29	0.00	5.37	0.00
Max	209.4	210.1	280.0	226.5	15.1

**Figure 2a: Coronary Artery Calcium (Agatston) Score Distribution by Individual Vessel****Figure 2b: Coronary Artery Calcium (Agatston) Score Distribution by Individual Vessel****Table 2: Degree of Coronary Stenosis (%) by Individual Vessel on CCTA**

Stenosis Category	LMA	LAD	LCX	RCA	PDA
0% - Normal	114 (58.2%)	0 (0.0%)	61 (31.1%)	0 (0.0%)	143 (73.0%)
1-24% - Minimal	28 (14.3%)	104 (53.1%)	135 (68.9%)	81 (41.3%)	53 (27.0%)
25-49% - Mild	0 (0.0%)	14 (7.1%)	0 (0.0%)	27 (13.8%)	0 (0.0%)
50-69% - Moderate	8 (4.1%)	77 (39.3%)	0 (0.0%)	50 (25.5%)	0 (0.0%)
70-99% - Severe	46 (23.5%)	1 (0.5%)	0 (0.0%)	38 (19.4%)	0 (0.0%)
Any stenosis (≥1%)	82 (41.8%)	196 (100%)	135 (68.9%)	196 (100%)	53 (27.0%)
Mean % ± SD	24.1 ± 35.0	29.4 ± 26.0	7.9 ± 7.4	41.6 ± 25.3	0.4 ± 0.7
Median %	0.0	22.0	6.0	48.0	0.0

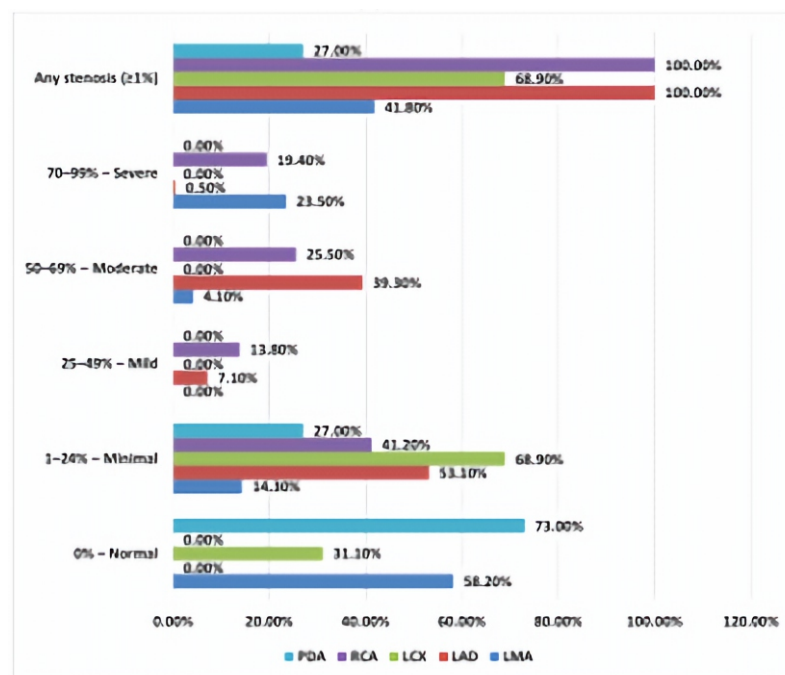


Figure 3a: Degree of Coronary Stenosis (%) by Individual Vessel on CCTA

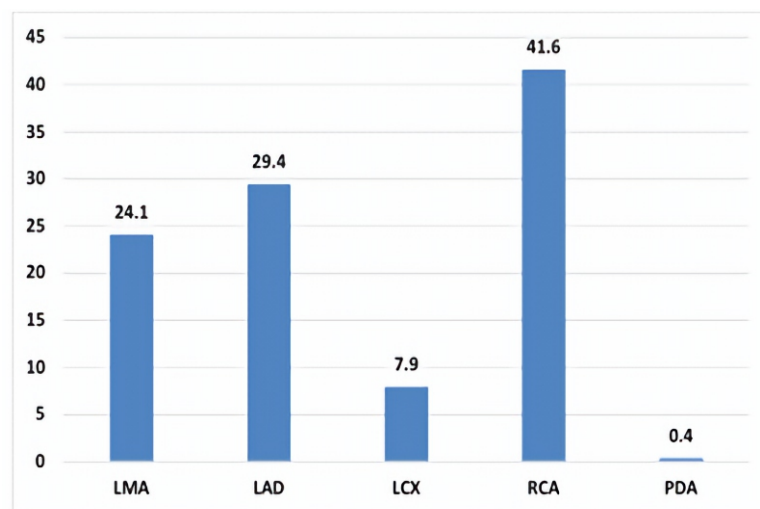


Figure 3b: Degree of Coronary Stenosis (%) by Individual Vessel on CCTA

Table 3: Association of CAD-RADS Plaque Burden Category with CAC Score and Stenosis Severity per Vessel

Parameter	P1 - Low Burden	P2 - Moderate Burden	P3 - High Burden
n (%)	101 (51.5%)	33 (16.8%)	62 (31.6%)
Mean Total CAC ± SD	19.2 ± 21.3	148.7 ± 115.8	140.5 ± 174.7
Median Total CAC	8.4	142.4	43.3
Mean CAC - LMA	1.1	42.7	38.4
Mean CAC - LAD	7.1	42.8	45.7
Mean CAC - LCX	2.7	11.4	14.1
Mean CAC - RCA	8.0	51.1	40.8
Mean CAC - PDA	0.4	0.6	1.5
Mean Stenosis - LMA (%)	9.2	43.2	38.2
Mean Stenosis - LAD (%)	17.1	37.0	45.5
Mean Stenosis - LCX (%)	5.5	10.1	10.5
Mean Stenosis - RCA (%)	29.5	51.1	56.3
Mean Stenosis - PDA (%)	0.3	0.2	0.6
Mild stenosis	67 (66.3%)	4 (12.1%)	16 (25.8%)
Moderate stenosis	11 (10.9%)	12 (36.4%)	13 (21.0%)
Severe stenosis	23 (22.8%)	17 (51.5%)	33 (53.2%)

anatomical distribution of calcification broadly paralleled the distribution of luminal disease. Dahdal et al. (2025), in 3,201 patients with suspected CAD, showed that CCTA-derived percent atheroma volume predicted adverse outcomes more strongly than conventional CAC scoring, highlighting the incremental value of direct plaque-burden assessment beyond calcium alone [13]. Manubolu et al. (2024) likewise reported strong correlations of CAC with calcified plaque volume ($r=0.76$) and total plaque volume ($r=0.72$), while CAC sensitivity for $>50\%$ stenosis was lower than calcified plaque volume (68% vs 77%), emphasizing the complementary role of quantitative CCTA [14]. Pedersen et al. (2023), in 10,857 symptomatic patients, found that 1.8% of those with CAC=0 still had obstructive CAD and 0.6% had high-risk CAD, showing that absence of calcium does not completely exclude anatomically important disease [15]. Hashmi et al. (2023) demonstrated a clear stepwise relationship between calcium burden and stenosis severity, with 66.0% of patients in the CAC 100-400 category having severe stenosis [16]. Morais et al. (2023) similarly identified CAC as an independent predictor of significant CAD (OR 1.016 per unit increase, $p<0.001$) [17]. In our plaque-burden analysis, P1 accounted for 51.5%, P2 for 16.8% and P3 for 31.6%; mean total CAC was markedly higher in P2 (148.7 ± 115.8) and P3 (140.5 ± 174.7) than in P1 (19.2 ± 21.3), while severe stenosis increased from 22.8% in P1 to 51.5% in P2 and 53.2% in P3. Moradi et al. (2022) showed that calcium-based parameters predicted CAD-RADS category with 79% accuracy and significant versus non-significant CAD with 81% accuracy [18]. Natanzon et al. (2022) further demonstrated the clinical utility of vessel-specific CAC thresholds, reporting NPVs of 99%, 97%, 92% and 91% for the left main, LAD, LCX and RCA, respectively [19]. Nykonenko et al. (2021) reported significant associations between Agatston burden and the number and complexity of affected coronary arteries, including a correlation with SYNTAX score ($r=0.69$, $p<0.01$), supporting the relationship between coronary calcification and anatomical disease complexity [20]. Tota-Maharaj et al. (2015) showed that involvement of increasing numbers of calcified coronary vessels and left-main calcification independently predicted higher all-cause mortality. Overall, these findings support assessment of CAC distribution, vessel-specific stenosis and CAD-RADS plaque burden as complementary markers of the extent and severity of coronary artery disease [5].

CONCLUSION

The study demonstrated that coronary calcification and luminal stenosis were heterogeneously distributed across the coronary circulation. The RCA and LAD showed the greatest calcium burden, while the RCA had the highest mean stenosis, with severe stenosis most frequent in the LMA and RCA. CAD-RADS P2 and P3 categories demonstrated substantially higher mean CAC than P1 and a greater frequency of severe stenosis. However, calcium burden did not increase consistently across plaque-burden categories, indicating that CACS and CCTA assess related but

distinct aspects of coronary atherosclerosis. Thus, CACS quantifies calcified disease burden, whereas CCTA provides comprehensive plaque and vessel-specific stenosis assessment.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multi-centre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic & management approaches. Such efforts will improve overall patient care & help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

CAC: Coronary Artery Calcium

CACS: Coronary Artery Calcium Scoring

CCTA: Coronary CT Angiography

CAD: Coronary Artery Disease

CAD-RADS: Coronary Artery Disease-Reporting and Data System

LMA: Left Main Artery

LCX: Left Circumflex Artery

RCA: Right Coronary Artery

PDA: Posterior Descending Artery

ECG: Electrocardiography

AUTHOR INFORMATION

Dr. Shaily Panwar: Associate Professor,

<http://orcid.org/0009-0005-6439-412X>

Dr. Preeti Tiwari: Associate Professor,

<http://orcid.org/0009-0006-3407-5926>

Dr. Dhruv Singh: Junior Resident,

<http://orcid.org/0009-0002-3140-0798>

AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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ETHICAL APPROVAL & CONSENT TO PARTICIPATE

All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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¹CSIR-Central Institute of Medicinal & Aromatic Plants, Lucknow, India

²CSIR-National Botanical Research Institute, Lucknow, India

HANDLING EDITOR

Dr. Dinesh Kumar Verma

Research Assistant Professor, School of Allied Health Sciences, Boise State University, Boise, Indiana, USA

e-mail: dineshkumarverma@boisestate.edu

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