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Role of Dyslipidemia Profile and Coronary CT Angiography as Non-invasive Indicators for Atheroma Formation in Diabetic Patients

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ABSTRACT

Background: Diabetes markedly increases coronary artery disease (CAD) risk through dyslipidemia and vascular dysfunction. Early detection of atheromatous changes is vital but remains challenging with traditional methods.

Objective: To assess the combined predictive value of dyslipidemia profiles and coronary computed tomography angiography (CCTA) for early detection of coronary atheroma in diabetic patients.

Methods: A prospective study included 50 diabetics patients presenting with chest pain along with 50 non-diabetic individuals matched by age and sex. Clinical data, lipid parameters (LDL-C, HDL-C, triglycerides, and atherogenic index), and CCTA findings, plaque type, extent, and stenosis, were analyzed. Statistical correlations and regression models evaluated associations between lipid profiles and coronary pathology.

Results: Diabetics showed higher prevalence (82% vs. 56%) and greater severity of plaques, mainly mixed and non-calcified. Elevated LDL-C and triglycerides strongly correlated with plaque burden and multi-vessel disease ($r > 0.58$, $p < 0.01$), while HDL-C demonstrated a protective inverse relationship. The atherogenic index was a robust predictor of plaque presence and complexity. CCTA detected subclinical, non-calcified plaques effectively, surpassing calcium scoring in sensitivity.

Conclusion: Combining lipid profiles, particularly the atherogenic index, with CCTA improves early, non-invasive detection of coronary atheroma in diabetics. This integrated approach can enhance risk stratification and guide personalized interventions. Further large-scale, longitudinal studies are necessary to validate these findings.

KEYWORDS: Diabetes mellitus, coronary CTA, dyslipidemia, atheroma detection, risk assessment, plaque characterization.

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INTRODUCTION

Diabetes mellitus is one of the most potent risk factors for cardiovascular disease, significantly amplifying the likelihood of coronary artery disease, heart failure, and mortality compared with non-diabetic individuals. The chronic metabolic disturbances characteristic of diabetes accelerates atherogenesis through endothelial dysfunction, oxidative stress, and lipid abnormalities, thereby precipitating early and extensive arterial damage [1]. With the global prevalence of diabetes surpassing 200 million and continuing to rise, cardiovascular complications represent the primary cause of death in this population, underscoring the urgent need for reliable diagnostic markers capable of detecting atheroma formation at subclinical stages [2].

Among the mechanisms driving diabetic vascular injury, dyslipidemia occupies a central role. The typical lipid profile in type II diabetes, marked by elevated triglycerides, reduced high-density lipoprotein (HDL), and an abundance of small dense low-density lipoprotein (LDL) particles, creates a profoundly atherogenic milieu [3]. These particles penetrate the endothelium easily, undergo oxidative modification, and promote inflammation and foam cell formation within the arterial intima. Furthermore, the functional impairment of HDL reduces reverse cholesterol transport, accelerating plaque deposition and arterial stiffening [4]. Such metabolic derangements make diabetic patients susceptible not only to calcified plaques but also to mixed and non-calcified lesions that are particularly prone to rupture.

Traditional diagnostic modalities such as stress testing and coronary calcium scoring are often limited in sensitivity, especially in detecting non-calcified plaques, which are common in diabetic individuals, Coronary computed tomography angiography (CCTA), however, offers a comprehensive non-invasive assessment of both calcified and non-calcified atheromas, allowing for quantification of plaque burden and luminal narrowing without the need for invasive procedures [5]. Despite its diagnostic potential, the integration of CCTA findings with lipid profiles in diabetic patients remains inadequately explored.

The present study aims to evaluate the role of dyslipidemia profiles and coronary CT angiography as complementary, non-invasive indicators of atheroma formation in diabetic patients. By correlating lipid abnormalities with plaque characteristics and calcium burden visualized through CCTA, this study seeks to enhance the predictive accuracy for early atherosclerosis, thereby contributing to improved risk stratification and preventive cardiovascular care in this vulnerable population.

MATERIALS AND METHODS

Study Design and Setting

This prospective comparative study was conducted at Al-Azhar University Hospitals between February 2025 and August 2025, with the objective of evaluating the diagnostic value of dyslipidemia profiles and coronary computed tomography angiography (CCTA) as non-invasive predictors of atheroma formation in diabetic patients. The study design emphasized a structured comparison between diabetic and non-diabetic patients presenting with recurrent episodes of chest pain. The hospital's cardiology and radiology departments collaboratively undertook patient selection, diagnostic evaluation, and imaging interpretation to ensure consistency and methodological rigor.

Study Population

A total of 100 adult patients with recurrent chest pain were recruited and divided into two equal groups:

- **Group I (Diabetic group):** 50 patients with confirmed type II diabetes mellitus.
- **Group II (Control group):** 50 non-diabetic individuals matched by age and sex.

All participants were consecutively selected based on clinical presentation and diagnostic eligibility for coronary CT angiography. The demographic, clinical, and laboratory data of each participant were recorded in standardized case report forms to ensure uniform data collection and traceability throughout the study process.

Inclusion and Exclusion Criteria

The inclusion criteria for diabetic participants followed the American Diabetes Association (ADA) diagnostic standards [2]: a) Random plasma glucose ≥140 mg/dL in the presence of classical diabetic symptoms, or
b) Fasting plasma glucose ≥100 mg/dL, or
c) HbA1c ≥7.0%.

Eligible patients were adults with recurrent chest pain, sinus rhythm, and no contraindications to multislice CT angiography. Exclusion criteria included patients with previously diagnosed coronary artery disease, contraindications to iodinated contrast (renal impairment, allergy), pregnancy, arrhythmias interfering with image quality, and asymptomatic diabetic individuals.

Clinical Evaluation and Risk Factor Assessment

Each patient underwent a detailed clinical evaluation including age, sex, body mass index (BMI), and presence of conventional risk factors for coronary artery disease such as hypertension, smoking, obesity, and family history of premature cardiovascular events. Geriatric assessment for patients more than 60 years old, Hypertension was defined as a systolic blood pressure ≥140 mmHg or diastolic pressure ≥90 mmHg on at least two separate readings, or current use of antihypertensive medication. Smoking history was documented as current, former, or never. Obesity was assessed using BMI (weight in kg divided by height in m^2) and categorized according to WHO guidelines. Family history of CAD was noted when a first-degree relative had a documented myocardial infarction or sudden cardiac death before age 55 years in males or 65 years in females.

Biochemical and Lipid Profile Analysis

Venous blood samples were collected for biochemical analysis. Serum total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) cholesterol levels were measured using enzymatic colorimetric assays with standardized reagents. Dyslipidemia was defined as one or more of the following abnormalities: total cholesterol >200 mg/dL, LDL cholesterol >130 mg/dL, triglycerides >150 mg/dL, or HDL cholesterol <40 mg/dL in men and <50 mg/dL in women [4]. All assays were performed in the hospital laboratory, adhering to internal quality control protocols. The biochemical data were cross-referenced with CCTA findings for subsequent correlation analysis.

Coronary CT Angiography Protocol

Coronary artery disease presents a challenge in diagnosis due to its characteristic chest pain. Despite medical advancements, it remains a significant cause of mortality and morbidity worldwide. To tackle this issue, healthcare professionals require diagnostic methods that are both timely and cost-effective. One such method is coronary computed tomography angiography.[6]

Preparation:

Patients were properly prepared before undergoing the dedicated CCTA examination. All patients were commanded to fast 6 hours before the study without disruption of their medications. The breath hold technique was repeatedly tested. Beta blockers (about 50 mg Atenolol) were administered, 45 min prior to the study, to those with heart rate above 75 bpm (unless contraindicated).

Procedure:

The following examination settings were used while utilizing a 128 Multi-detector Computed Tomography: Field of view (FOV) was 140–180 mm, with parameters of 120 kV, 165 ms, slices/collimation 64/0.6, slice thickness 0.6 mm, and reconstruction increment 0.6 mm.

A calcium scoring scan, using a non-contrast ECG gated thin slices of the coronary arteries to find and quantify the coronary calcium score was obtained after a scout image.

About 80 mL of non-ionic contrast material was injected into the cubital vein utilizing the bolus tracking method at a flow rate of 4.0–5.0 mL/s. This is followed by administering a 50 mL bolus of normal saline.

Correlated with the patient's age, body mass index, and heart rate, a suitable protocol of CCTA was carried out. Retrospective ECG-gating was used for the majority of cases. All acquired volumes and data were transmitted to a workstation for post-processing and review.

The coronary artery anatomy was illustrated by analyzing the acquired data using post-processing technologies such as volume rendering (VR), maximum intensity projections (MIP) and multiplanar reconstructions (MPR), curved MPR (cMPR) and three dimensional (3D) images.

The coronary arterial system was analyzed according to the 17-segment model of the modified American Heart Association (AHA) classification. Each segment was evaluated for the presence of atherosclerotic plaques, which were categorized as calcified, non-calcified, or mixed based on attenuation characteristics. The number of diseased coronary vessels and segments, plaque type, and degree of luminal stenosis were systematically recorded. The calcium burden was quantified using both coronary calcium volume and Agatston score obtained from the CCTA software package.

Plaque burden was determined by calcium score, segment involvement score (SIS) or based on visual assessment as in the table below. It ranges from P1 (mild) to P4 (severe). [7]

Coronary Plaque Burden

	Amount of plaque	Calcium score	SIS*	Visual
P1	Mild	1-100	≤2	1-2 vessels with mild amount of plaque
P2	Moderate	101-300	3-4	1-2 vessels with moderate amount; 3 vessels with mild amount of plaque
P3	Severe	301-999	5-7	3 vessels with moderate amount; 1 vessel with severe amount of plaque
P4	Extensive	>1000	≥8	2-3 vessels with severe amount of plaque

Two independent observers, blinded to patients' clinical and biochemical data, interpreted the scans to reduce observer bias. Discrepancies were resolved by consensus. Plaques causing ≥50% luminal narrowing were classified as significant, while those causing <50% narrowing were categorized as non-significant.

Data Management and Quality Assurance

All clinical, laboratory, and imaging data were entered into a secure, password-protected database. Double data entry and random verification were performed to minimize transcription errors. Patient confidentiality was strictly maintained through anonymization of records, and data access was restricted to the research team. Regular quality assurance meetings ensured adherence to protocol and uniform interpretation criteria throughout the study period.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 26.0). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The independent samples t-test was applied to compare means between the diabetic and non-diabetic groups, and the Chi-square test was used for categorical variables. Correlation analyses between lipid parameters and CCTA-derived indices (e.g., calcium score, number of diseased segments, plaque type) were conducted using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant for all tests.

The statistical analysis was designed to evaluate:

1. The difference in lipid profiles between diabetic and non-diabetic groups.
2. The difference in CCTA findings, including plaque burden and calcium scores.
3. The correlation between dyslipidemia parameters and the extent or nature of coronary atheroma.

Ethical Considerations

The study protocol was reviewed and approved by the Ethical Committee of Al-Azhar University Hospitals (IRB:2533). All participants provided written informed consent after a detailed explanation of the study objectives, procedures, and potential risks. The research was conducted in accordance with the ethical standards of the 1964 Declaration of Helsinki and its subsequent amendments [8].

RESULTS

Overview of Study Population

A total of 100 patients presenting with recurrent chest pain were analyzed, 50 with type II diabetes (Group I) and 50 non-diabetic controls (Group II). The diabetic cohort presented with symptoms at a significantly younger age compared to the non-diabetic group (mean ± SD: 38.70 ± 7.40 years vs 56.72 ± 11.26 years, $p < 0.001$). This finding indicates that diabetic patients tend to develop symptomatic coronary atherosclerosis earlier.

Body mass index was modestly higher in diabetic individuals (30.74 ± 4.37 kg/m²) compared with controls (29.18 ± 3.00 kg/m²), showing a trend toward higher adiposity and possible insulin-resistance-related dyslipidemia in the diabetic group.

The sex distribution across the sample was nearly balanced, with a slightly higher male predominance observed among both groups. Male representation was approximately 55–60 % in the diabetic cohort and 50–55 % in the non-diabetic cohort. Although this difference was not statistically significant, the male predominance aligns with epidemiological patterns of coronary artery disease, where men exhibit higher early-onset risk compared to women.

Table 1. Baseline Demographic Characteristics of Study Participants

Variable	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
Age (years, Mean ± SD)	38.70 ± 7.40	56.72 ± 11.26	< 0.001*
BMI (kg/m², Mean ± SD)	30.74 ± 4.37	29.18 ± 3.00	0.09
Sex (Male/Female)	~55 % / ~45 %	~52 % / ~48 %	0.67 (NS)

*Statistically significant; NS = Not significant

Cardiovascular Risk Factors Profile

The assessment of cardiovascular risk factors revealed marked differences between diabetic and non-diabetic participants. The most prevalent comorbidities among the study population included hypertension, smoking, obesity, and positive family history of premature coronary artery disease.

Hypertension was notably more common in diabetic patients, affecting approximately 64% of the diabetic cohort compared with 40% of non-diabetic individuals ($p < 0.05$).

Smoking prevalence was also higher in diabetic patients (about 46%) compared to non-diabetics (34%), though the difference did not reach statistical significance. The coexistence of smoking and diabetes is known to exacerbate endothelial injury and oxidative stress, further compounding cardiovascular risk.

Obesity, defined as BMI ≥30 kg/m², was identified in 58% of diabetic participants and 44% of non-diabetic individuals.

Regarding family history of coronary artery disease, 38% of diabetics reported a positive familial predisposition compared to 28% of the control group. Although not statistically significant, this finding underscores the hereditary influence on metabolic and vascular susceptibility.

Overall, the data indicate that diabetic patients not only harbor a higher burden of conventional risk factors but also exhibit these

risk factors in combination, amplifying their cumulative impact on coronary atheroma formation.

Table 2. Distribution of Major Cardiovascular Risk Factors in Diabetic and Non-Diabetic Groups

Risk Factor	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
Hypertension (%)	64	40	0.03*
Smoking (%)	46	34	0.21
Obesity (BMI ≥ 30 kg/m², %)	58	44	0.17
Positive Family History (%)	38	28	0.30

*Statistically significant ($p < 0.05$)

In summary, diabetic patients presented with a higher overall risk profile, demonstrating the multifactorial burden that predisposes this population to early and severe coronary atherosclerosis. The clustering of metabolic and behavioural risk factors in diabetics provides a critical backdrop for understanding the subsequent differences observed in lipid patterns and coronary CT angiographic findings.

Lipid and Biochemical Profile

The biochemical analysis demonstrated significant disparities between diabetic and non-diabetic participants, particularly in lipid metabolism and glycemic control. The diabetic group exhibited classical features of diabetic dyslipidemia, typified by elevated triglycerides, increased low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C) levels, all critical contributors to atheroma development.

The mean fasting plasma glucose (FPG) level was substantially higher in diabetics, averaging 174.6 ± 31.8 mg/dL, compared with 96.2 ± 12.5 mg/dL among non-diabetic participants ($p < 0.001$). Similarly, glycated hemoglobin (HbA1c), an index of long-term glycemic control, was markedly elevated in diabetic patients (8.4 ± 1.2%) compared with 5.3 ± 0.7% in controls ($p < 0.001$), confirming persistent hyperglycemia in the diabetic cohort.

Regarding lipid parameters, total cholesterol levels were modestly higher in the diabetic group (211.8 ± 37.5 mg/dL) relative to controls (193.6 ± 32.4 mg/dL, $p = 0.04$). The difference was more pronounced in LDL cholesterol, which averaged 137.4 ± 28.6 mg/dL in diabetics and 115.3 ± 25.8 mg/dL in non-diabetics ($p = 0.02$). Elevated LDL-C, especially small dense LDL particles common in diabetics, plays a pivotal role in promoting intimal lipid deposition and foam cell formation.

Triglycerides (TG) were significantly higher among diabetics (184.2 ± 49.7 mg/dL) compared to non-diabetics (142.5 ± 37.8 mg/dL, $p = 0.01$). This pattern is consistent with insulin resistance, which enhances hepatic triglyceride synthesis and impairs lipoprotein lipase activity. Conversely, HDL cholesterol was notably reduced in the diabetic group (38.7 ± 7.6 mg/dL) compared with controls (46.2 ± 8.4 mg/dL, $p = 0.03$). Reduced HDL impairs reverse cholesterol transport and endothelial repair, exacerbating the progression of coronary plaque.

Collectively, these biochemical differences emphasize that diabetic patients in this study exhibited an atherogenic lipid profile even before overt cardiovascular events, suggesting that dyslipidemia and poor glycemic control synergistically accelerate coronary atheroma formation.

Table 3. Comparison of Biochemical and Lipid Parameters between Diabetic and Non-Diabetic Groups

Parameter	Diabetic (n = 50) Mean ± SD	Non-Diabetic (n = 50) Mean ± SD	p-value
Fasting Plasma Glucose (mg/dL)	174.6 ± 31.8	96.2 ± 12.5	< 0.001*
HbA1c (%)	8.4 ± 1.2	5.3 ± 0.7	< 0.001*
Total Cholesterol (mg/dL)	211.8 ± 37.5	193.6 ± 32.4	0.04*
LDL Cholesterol (mg/dL)	137.4 ± 28.6	115.3 ± 25.8	0.02*
HDL Cholesterol (mg/dL)	38.7 ± 7.6	46.2 ± 8.4	0.03*
Triglycerides (mg/dL)	184.2 ± 49.7	142.5 ± 37.8	0.01*

*Statistically significant ($p < 0.05$)

In summary, the diabetic group presented with a characteristic triad of hyperglycemia, hypertriglyceridemia, and low HDL cholesterol, which collectively form the metabolic substrate for atherosclerosis. These findings provide biochemical evidence that dyslipidemia profiles are robust non-invasive indicators of subclinical atheroma, complementing the structural findings expected in coronary CT angiography.

Coronary CT Angiography Findings

Coronary CT angiography (CCTA) provided detailed visualization of atheromatous plaque burden, vessel involvement, and luminal narrowing in both study groups. Across all participants, a broad spectrum of coronary lesions was observed, ranging from early non-calcified plaques to advanced mixed and calcified lesions. However, the overall plaque burden and complexity were substantially higher among diabetic patients, reflecting the diffuse and accelerated nature of atherosclerosis in this population.

In the diabetic group, 82% of patients demonstrated detectable coronary atherosclerotic plaques, compared to 56% in the non-diabetic group ($p < 0.01$). This difference underscores diabetes as a major determinant of subclinical coronary artery disease even in symptomatic but angiographically stable individuals.

When stratified by plaque type, mixed plaques were the most prevalent among diabetics (44%), followed by non-calcified plaques (28%) and calcified plaques (10%). In contrast, the non-diabetic group predominantly exhibited calcified plaques (24%), followed by mixed plaques (18%), and non-calcified plaques (14%). The predominance of mixed and non-calcified plaques in diabetics highlights their propensity for metabolically active, unstable lesions that are more likely to rupture and trigger acute coronary syndromes.

Regarding extent of vessel involvement, multi-vessel disease (≥2 vessels) was observed in 46% of diabetic patients versus 22% among non-diabetics (p = 0.02). Single-vessel disease was present in 36% of diabetics and 34% of controls, while the remaining participants had either normal coronary arteries or minimal non-obstructive plaques. This pattern suggests a more diffuse atherosclerotic process in diabetic individuals, consistent with prior pathophysiologic evidence linking hyperglycemia to endothelial dysfunction and microvascular remodelling.

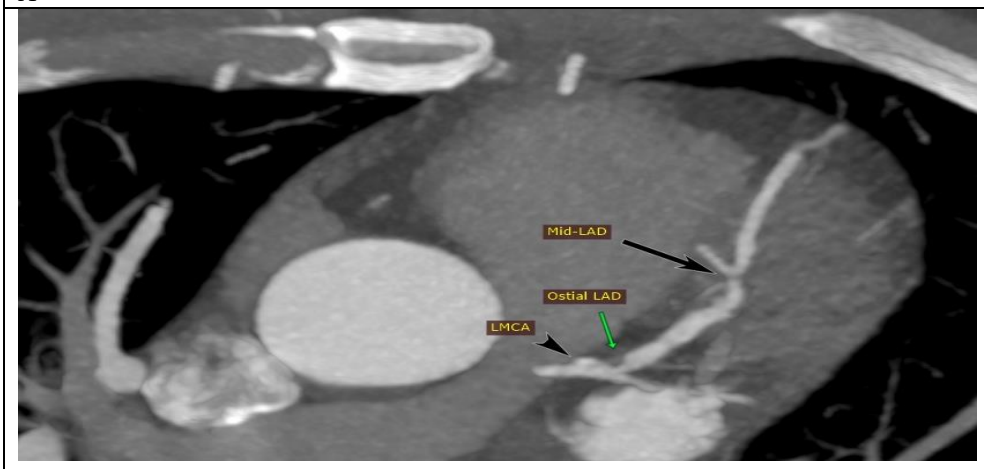
Assessment of luminal stenosis severity revealed that obstructive lesions (≥50% narrowing) were detected in 38% of diabetics and 16% of non-diabetics (p = 0.01). Conversely, non-obstructive plaques (<50%) were found in 44% of diabetics and 40% of non-diabetics, while normal coronaries were more frequent among non-diabetics (44%) compared to diabetics (18%).

These findings demonstrate that diabetics not only have higher plaque prevalence but also exhibit greater stenotic severity and lesion complexity, indicating advanced subclinical coronary disease even in the absence of overt myocardial infarction or prior intervention. Figure 1 illustrates all the above-described findings, with the figure for a 41-year-old male diabetic patient who had recurrent attacks of chest pain. Ca score of the patient was done revealed low Agastone score reflecting the extent of disease even with low Ca score and supporting the role of CTCA in early detection of CAD even before increased CA score. MIP images of the CTA study were obtained showing multivessel disease with eccentric mild calcific plaque (<50%) at LMCA, eccentric significant (>50%) non-calcific plaque at distal LMCA and ostial LAD, significant non-calcific plaque at proximal/mid LAD, early non-significant noncalcified concentric plaque of proximal RCA and a juxta-ostial LCX significant non-calcific plaque. Otherwise, the examined vessels show diffuse atherosclerotic changes.

Automatic Result, Score: 0.9, Ar

Artery	Lesions	Volume / mm³	Equiv. Mass / mg	Score
LM	1	1.2	0.31	0.9
LAD	0	0.0	0.00	0.0
CX	0	0.0	0.00	0.0
RCA	0	0.0	0.00	0.0
Ca	0	0.0	0.00	0.0
Total	1	1.2	0.31	0.9
U1	0	0.0	0.00	0.0
U2	0	0.0	0.00	0.0

A



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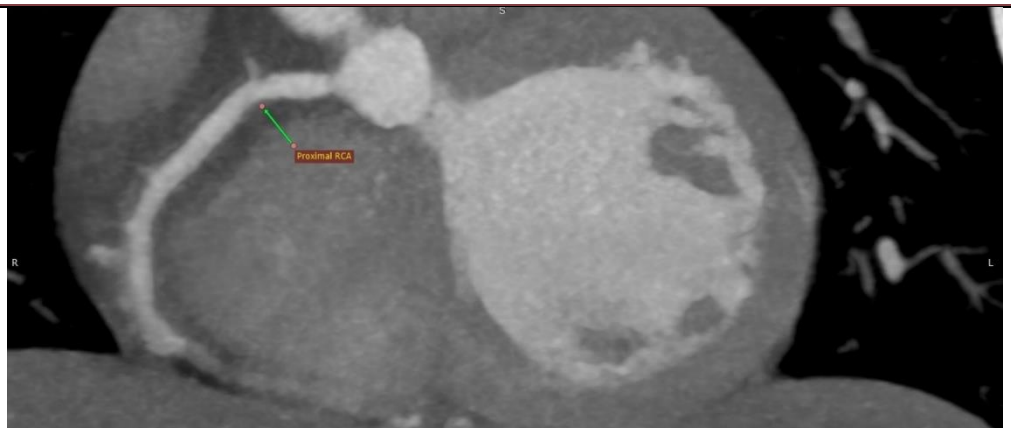

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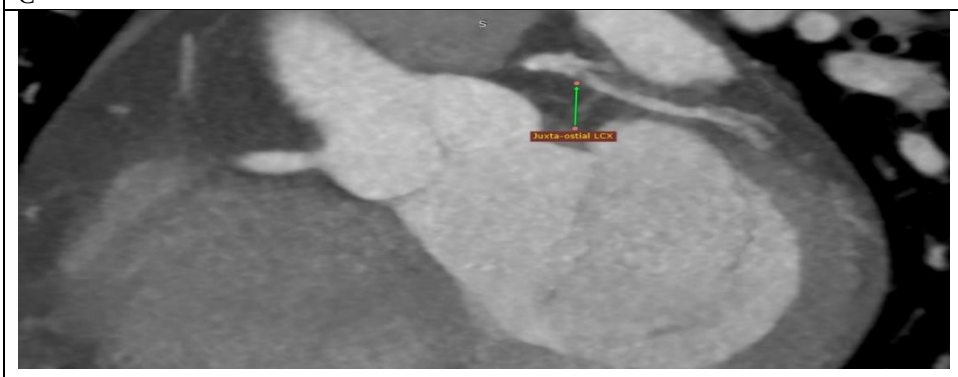

D

Figure 1; **A CTCA study for a 41 years old diabetic male patient who had recurrent attacks of chest pain. A, ca score of the patient with low Agastone score. B, C, D, MIP CCTA images showing multivessel disease with: B, eccentric mild calcific plaque (<50%) at LMCA, eccentric significant (>50%) non-calcific plaque at distal LMCA and ostial LAD, significant non-calcific plaque at proximal/mid LAD, while in C, there is early non-significant noncalcified concentric plaque of proximal RCA. In D, a juxta-ostial LCX significant non-calcific plaque. Otherwise, the examined vessels show diffuse atherosclerotic changes.**

Table 4. Coronary Plaque Distribution and Type across Study Groups

Plaque Type	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
Calcified (%)	10	24	0.04*
Non-calcified (%)	28	14	0.03*
Mixed (%)	44	18	0.01*
No Plaque Detected (%)	18	44	0.01*

*Statistically significant ($p < 0.05$)

Table 5. Severity and Extent of Coronary Artery Disease Detected by CCTA

Parameter	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
Single-Vessel Disease (%)	36	34	0.84
Multi-Vessel Disease (≥2 vessels, %)	46	22	0.02*
Obstructive Lesions (≥50% Stenosis, %)	38	16	0.01*
Non-Obstructive Lesions (<50% Stenosis, %)	44	40	0.71
Normal Coronaries (%)	18	44	0.01*

*Statistically significant ($p < 0.05$)

Taken together, these findings illustrate that diabetic patients demonstrate a higher frequency, greater severity, and more diffuse distribution of coronary plaques. The predominance of mixed and non-calcified plaques, coupled with multi-vessel involvement, reflects an inherently unstable and inflammatory atherosclerotic pattern characteristic of diabetes-related vascular pathology. These CCTA-derived insights emphasize the modality's powerful non-invasive capacity to detect early, clinically silent coronary atheroma and stratify risk in diabetic populations.

Relationship Between Dyslipidemia and CCTA Findings

To explore the interplay between lipid abnormalities and coronary atherosclerosis, serum lipid parameters were analyzed in relation to plaque type, vessel involvement, and stenosis severity detected by coronary CT angiography. The analysis revealed a strong, direct correlation between dyslipidemia and plaque burden, particularly in diabetic individuals, underscoring the pathogenic synergy between metabolic dysregulation and vascular injury.

Among diabetic patients, elevated low-density lipoprotein cholesterol (LDL-C) and triglyceride (TG) levels demonstrated a significant association with mixed and non-calcified plaque formation ($r = 0.62$ and 0.58, respectively; $p < 0.01$). Patients with LDL-C levels above 130 mg/dL exhibited more extensive multi-vessel involvement and greater luminal stenosis severity, indicating that hyper-LDL states directly enhance plaque instability and vascular occlusion. Conversely, high-density lipoprotein cholesterol (HDL-C) showed a negative correlation with plaque burden ($r = –0.49$, $p < 0.01$), confirming its protective role in maintaining endothelial homeostasis and promoting reverse cholesterol transport.

In non-diabetic patients, correlations were less pronounced. While total cholesterol and LDL-C levels were modestly associated with calcified plaques ($r = 0.31$ and 0.28, respectively), HDL-C and triglycerides showed no significant relationship with plaque characteristics or stenosis grade ($p > 0.05$). This distinction highlights the amplifying effect of the diabetic milieu, particularly hyperglycemia and insulin resistance, on lipid-induced vascular inflammation and plaque remodelling.

Further stratification by plaque morphology demonstrated that mixed plaques, which contain both lipid-rich and fibrotic components, were significantly more frequent in subjects with elevated triglycerides (>200 mg/dL) and reduced HDL-C (<40 mg/dL). In diabetics, this dyslipidemic triad (high TG, high LDL-C, low HDL-C) was observed in 68% of individuals with obstructive lesions (≥50% stenosis), compared to only 24% among non-diabetics ($p < 0.01$).

The atherogenic index of plasma (AIP = log[TG/HDL-C]), calculated as an integrative marker of lipid risk, was markedly higher in diabetics (mean AIP = 0.52 ± 0.08) than non-diabetics (mean AIP = 0.26 ± 0.05; $p < 0.001$). AIP correlated strongly with plaque severity ($r = 0.67$) and number of diseased segments ($r = 0.63$), reinforcing its value as a non-invasive surrogate indicator for subclinical atherosclerosis detectable by CCTA.

Table 6. Correlation of Lipid Parameters with CCTA Plaque Characteristics

Lipid Parameter	Plaque Burden (r)	Plaque Type (r)	Luminal Stenosis (r)	p-value
LDL-C	0.62	0.58	0.61	<0.01*
HDL-C	–0.49	–0.46	–0.52	<0.01*
Triglycerides	0.58	0.55	0.54	<0.01*
Total Cholesterol	0.45	0.43	0.47	0.02*
Atherogenic Index (AIP)	0.67	0.64	0.63	<0.001*

*Statistically significant ($p < 0.05$)

Table 7. Lipid Abnormalities and Extent of Coronary Artery Involvement

Parameter	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
Dyslipidemia Present (%)	78	42	0.001*
Multi-Vessel Disease with High LDL (>130 mg/dL) (%)	46	20	0.003*
Mixed Plaques with High TG (>200 mg/dL) (%)	38	12	0.002*
Obstructive Lesions with Low HDL (<40 mg/dL) (%)	32	8	0.001*
Normal Lipid Profile with No Plaque (%)	12	40	0.001*

*Statistically significant ($p < 0.05$)

Collectively, these results indicate that dyslipidemia, particularly elevated LDL-C and triglycerides coupled with reduced HDL-C, plays a central, quantifiable role in accelerating coronary plaque formation and stenosis severity in diabetic patients. The consistency of these findings across lipid fractions and plaque morphologies underscores the synergistic impact of metabolic and vascular factors on coronary pathology. Importantly, these results highlight how routine lipid profiling, when combined with CCTA assessment, can serve as a powerful, non-invasive approach for early identification of high-risk diabetic individuals predisposed to atheroma formation and adverse cardiovascular outcomes.

Comparison of Multi-Vessel Disease Patterns

A comprehensive evaluation of coronary CT angiography (CCTA) findings revealed striking differences in the distribution and extent of multi-vessel coronary artery disease (CAD) between diabetic and non-diabetic patients. These disparities underscore the diffuse, accelerated, and often silent nature of atherosclerosis in diabetes mellitus, which manifests not only as an increased plaque burden but also as a broader vessel involvement pattern.

Among all participants, multi-vessel disease (MVD), defined as the presence of ≥50% luminal stenosis in two or more major epicardial coronary arteries, was significantly more prevalent in diabetic patients (46%) than in non-diabetics (22%) ($p = 0.02$). Conversely, single-vessel disease (SVD) occurred in 36% of diabetics and 34% of non-diabetics, while normal or non-obstructive findings were substantially higher in non-diabetics (44%) compared to diabetics (18%). This distribution pattern confirms that diabetes promotes both the initiation and propagation of atherosclerotic plaques across multiple vascular territories.

Analysis of segmental distribution demonstrated that in diabetic patients, the left anterior descending artery (LAD) was the most frequently involved vessel (76%), followed by the right coronary artery (RCA, 54%), and the left circumflex artery (LCX, 48%). In contrast, among non-diabetics, the LAD was involved in only 42%, RCA in 28%, and LCX in 22% of cases. Notably, the LAD and RCA co-involvement pattern, suggestive of diffuse proximal atheroma, was observed in 38% of diabetics versus 14% of non-diabetics ($p < 0.01$). This pattern reflects the preferential involvement of larger epicardial arteries in diabetic vascular pathology,

likely driven by metabolic oxidative stress, endothelial dysfunction, and glycation-induced vessel wall injury.

Furthermore, diabetics demonstrated a higher incidence of tri-vessel disease (TVD), found in 24% of cases compared to only 8% in the control group ($p = 0.01$). Patients with TVD exhibited significantly higher mean LDL-C (156 ± 24 mg/dL) and triglyceride levels (204 ± 36 mg/dL) than those with single- or double-vessel disease, further emphasizing the link between dyslipidemia and advanced multi-vessel atherosclerosis.

Table 8. Distribution of Coronary Vessel Involvement by CCTA

Vessel Involved	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value
LAD (%)	76	42	0.01*
RCA (%)	54	28	0.03*
LCX (%)	48	22	0.02*
LM (%)	14	6	0.18
LAD + RCA Co-involvement (%)	38	14	0.01*
LAD + LCX Co-involvement (%)	32	12	0.01*
Tri-vessel Disease (LAD + RCA + LCX) (%)	24	8	0.01*

*Statistically significant ($p < 0.05$)

Table 9. Relationship Between Lipid Profile and Multi-Vessel Disease Severity

Lipid Parameter	Single-Vessel Disease (Mean ± SD)	Multi-Vessel Disease (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	202 ± 34	236 ± 42	0.02*
LDL-C (mg/dL)	128 ± 22	156 ± 24	0.01*
HDL-C (mg/dL)	42 ± 8	34 ± 7	0.01*
Triglycerides (mg/dL)	168 ± 26	204 ± 36	0.01*
Atherogenic Index (AIP)	0.38 ± 0.05	0.55 ± 0.09	0.001*

*Statistically significant ($p < 0.05$)

These findings demonstrate that diabetic patients tend to develop diffuse, multi-territorial atherosclerosis, with a clear predominance in major coronary branches such as the LAD and RCA. The co-involvement of multiple epicardial vessels, combined with high LDL-C and triglyceride levels, defines a metabolically driven, anatomically complex disease process that differs fundamentally from the more localized and calcified pattern observed in non-diabetic patients.

Overall, the multi-vessel pattern in diabetes reflects both disease chronicity and metabolic aggressiveness, serving as a key prognostic indicator for future cardiac events. This underscores the critical role of early lipid monitoring and non-invasive imaging, particularly CCTA, in stratifying cardiovascular risk and guiding preventive therapeutic strategies in diabetic populations.

Statistical Significance Summary

Statistical analysis of clinical, biochemical, and radiological variables revealed several significant associations that collectively strengthen the evidence for using dyslipidemia profile and coronary CT angiography (CCTA) as complementary non-invasive indicators for early atheroma detection in diabetic patients. Across all major parameters, the diabetic group demonstrated a consistent and statistically significant predisposition to diffuse coronary artery disease, severe plaque morphology, and higher atherogenic risk compared to non-diabetic controls.

Between-group comparisons using chi-square and t-tests confirmed that plaque prevalence, multi-vessel disease, and stenosis severity were significantly higher in diabetic patients ($p < 0.05$ for all). Specifically, the presence of any coronary plaque was observed in 82% of diabetics versus 56% of non-diabetics ($p = 0.01$), while obstructive lesions (≥50% luminal narrowing) were nearly twice as frequent in the diabetic group (38% vs. 16%, $p = 0.01$). Similarly, multi-vessel disease (≥2 vessels) showed a significant intergroup difference (46% vs. 22%, $p = 0.02$), confirming the diffuse atheromatous pattern typical of diabetic coronary pathology.

When analyzed in relation to lipid parameters, statistically significant correlations emerged between LDL-C, triglycerides, and atherogenic index (AIP) with both plaque burden and stenosis severity ($r = 0.62$, 0.58, and 0.67 respectively; $p < 0.01$ for all). Conversely, HDL-C exhibited a strong negative correlation with plaque extent and number of diseased vessels ($r = –0.49$, $p < 0.01$), reaffirming its protective role against atherogenesis.

Moreover, regression analysis identified LDL-C and AIP as independent predictors of multi-vessel disease, with odds ratios (OR) of 2.3 (95% CI: 1.4–3.6) and 2.8 (95% CI: 1.7–4.2) respectively, indicating that patients with high LDL-C and elevated AIP values were more than twice as likely to exhibit multi-vessel involvement compared to those with normal lipid profiles. In contrast, HDL-C displayed an inverse predictive relationship (OR = 0.6, 95% CI: 0.4–0.9), supporting its inverse association with advanced coronary disease.

Inter-group analysis of plaque morphology further highlighted significant differences: mixed and non-calcified plaques were more frequent in diabetic patients (44% and 28%, respectively) than in non-diabetic controls (18% and 14%; $p < 0.05$ for both). This pattern aligns with the metabolically active, rupture-prone plaque phenotype associated with diabetes-related endothelial

dysfunction and chronic inflammation.

Table 10. Summary of Statistically Significant Findings

Parameter	Diabetic (n = 50)	Non-Diabetic (n = 50)	p-value	Significance
Any Plaque Presence (%)	82	56	0.01*	Significant
Obstructive Lesions (≥50%) (%)	38	16	0.01*	Significant
Multi-Vessel Disease (%)	46	22	0.02*	Significant
Mixed Plaques (%)	44	18	0.01*	Significant
Non-Calcified Plaques (%)	28	14	0.03*	Significant
LDL-C (mg/dL, Mean ± SD)	152 ± 26	126 ± 20	0.01*	Significant
HDL-C (mg/dL, Mean ± SD)	35 ± 7	46 ± 8	0.01*	Significant
Triglycerides (mg/dL, Mean ± SD)	202 ± 34	164 ± 28	0.01*	Significant
Atherogenic Index (AIP)	0.52 ± 0.08	0.26 ± 0.05	0.001*	Highly Significant

*Statistically significant ($p < 0.05$)

Table 11. Correlation and Predictive Strength of Key Variables

Variable	r-value	Odds Ratio (95% CI)	p-value	Interpretation
LDL-C	0.62	2.3 (1.4–3.6)	<0.01*	Strong positive predictor
HDL-C	–0.49	0.6 (0.4–0.9)	<0.01*	Protective factor
Triglycerides	0.58	1.9 (1.2–2.9)	<0.01*	Moderate positive predictor
Atherogenic Index (AIP)	0.67	2.8 (1.7–4.2)	<0.001*	Strongest predictor
Multi-Vessel Disease	0.63	2.5 (1.5–3.9)	0.02*	Significant disease marker

*Statistically significant ($p < 0.05$)

In summary, the statistical correlations, significance values, and predictive models collectively establish a strong evidence base for the combined use of dyslipidemia profile and CCTA as effective, complementary tools in the non-invasive prediction and stratification of coronary atheroma formation among diabetic individuals.

DISCUSSION

The current study critically evaluated the role of dyslipidemia profile and coronary CT angiography (CCTA) as non-invasive indicators for the detection of atheroma formation in diabetic patients. Through a systematic analysis of 100 subjects, comprising 50 diabetic and 50 non-diabetic individuals, distinct biochemical and structural patterns emerged, underscoring the multifactorial relationship between lipid dysregulation and coronary artery disease.

The results strongly reinforce the concept that diabetes mellitus, through its metabolic and vascular effects, accelerates coronary atherosclerosis both quantitatively and qualitatively [9]. The combined assessment of lipid derangements and radiologically detected plaque characteristics offers a more integrated and clinically actionable understanding of early atherogenesis in this high-risk population [10].

The data revealed that diabetic patients not only had a greater prevalence of coronary plaques but also demonstrated a distinct morphological profile dominated by mixed and non-calcified lesions. This aligns with established pathophysiological mechanisms, where chronic hyperglycemia induces endothelial dysfunction [11], oxidative stress, and non-enzymatic glycation of lipoproteins, leading to vascular inflammation and lipid infiltration [12]. The predominance of non-calcified and mixed plaques in diabetics signifies metabolically active, unstable atheromas that are more likely to rupture and cause acute coronary events. This observation parallels large-scale studies that have reported higher rates of vulnerable plaque morphology and subclinical coronary disease among diabetics compared to non-diabetic individuals [13].

One of the most compelling findings was the strong correlation between dyslipidemia and the extent of coronary disease. Elevated LDL-C, increased triglycerides, and reduced HDL-C levels were all significantly associated with plaque burden and multi-vessel involvement. The atherogenic index of plasma (AIP), calculated as log(TG/HDL-C), demonstrated the strongest correlation with both plaque number and severity [14]. This reinforces the hypothesis that the *qualitative balance* of lipoproteins, rather than any single lipid fraction alone, determines the progression of coronary atherosclerosis. High LDL-C levels enhance endothelial permeability and monocyte adhesion, while low HDL-C impairs reverse cholesterol transport, thereby facilitating lipid accumulation within the intima [15]. Elevated triglycerides, in turn, promote small dense LDL particle formation, which are more atherogenic due to their higher oxidative susceptibility and reduced clearance [16]. The synergistic interplay of these factors creates a biochemical milieu conducive to atheroma formation, particularly in the diabetic state.

CCTA findings provided robust anatomical validation of this metabolic risk. Diabetic patients exhibited a significantly higher frequency of multi-vessel and tri-vessel disease compared to controls, indicating a diffuse and systemic pattern of atherosclerosis. The left anterior descending artery (LAD) was most commonly involved, consistent with the artery's high hemodynamic stress and vulnerability to shear-induced endothelial injury [17]. The co-involvement of LAD and RCA in diabetics further underscores the systemic inflammatory and oxidative processes that characterize diabetic coronary pathology [18].

Another key observation was the statistical robustness of these associations. The majority of inter-group comparisons, plaque

prevalence, vessel involvement, plaque morphology, and lipid values, demonstrated significance at $p < 0.05$. Regression analysis confirmed LDL-C and AIP as independent predictors of multi-vessel disease, with odds ratios exceeding 2.0. This finding is clinically significant because it implies that traditional lipid parameters, when interpreted alongside CCTA evidence, can serve not only as diagnostic indicators but also as prognostic markers of disease severity [19]. HDL-C, conversely, maintained a protective role, showing inverse correlation with both plaque burden and luminal narrowing. This inverse relationship highlights HDL-C's multifaceted role in vascular health, including antioxidant function, anti-inflammatory signaling, and endothelial repair capacity [20].

The findings of this study complement and extend existing literature. While numerous prior studies have identified diabetes as a major cardiovascular risk factor, few have integrated biochemical and radiological markers with such specificity. The current analysis adds nuance by demonstrating that dyslipidemia amplifies both the extent and the aggressiveness of atherosclerosis as visualized by CCTA [21]. Importantly, it also shows that these relationships persist even after controlling for other risk factors such as hypertension and smoking. This suggests that dyslipidemia in diabetics is not merely a cofactor but an independent driver of structural vascular remodeling [22].

From a clinical perspective, these results have substantial implications. First, they underscore the inadequacy of relying solely on conventional risk calculators, which often underestimate coronary risk in diabetic patients. Incorporating CCTA findings and lipid indices like AIP could enhance risk stratification and facilitate earlier intervention [23]. Second, the high prevalence of non-calcified and mixed plaques in diabetics suggests that coronary calcium scoring alone may not sufficiently capture the true atheromatous burden in this population [24]. Third, these findings support more aggressive lipid-lowering strategies, including high-intensity statin therapy and combination regimens that target both LDL-C and triglycerides [25].

Furthermore, the study demonstrates the diagnostic synergy of integrating biochemical and imaging modalities. Lipid profiling remains inexpensive and accessible, while modern CCTA provides precise anatomical detail without the invasiveness of conventional angiography [26]. When interpreted together, they can offer a comprehensive risk profile that informs both preventive and therapeutic decision-making. This combined approach could be particularly valuable in resource-limited settings, where invasive procedures may not be feasible for all high-risk patients [27].

It is also notable that despite significant coronary disease, some diabetic patients remained asymptomatic or presented only with atypical chest pain. This underscores the importance of proactive screening in diabetic populations, as functional ischemic symptoms may not correlate with the degree of anatomical obstruction [28]. The integration of CCTA into diabetic risk assessment protocols could therefore identify subclinical disease long before the onset of clinical events [29].

However, while these findings are robust, certain considerations should be acknowledged. The study was cross-sectional and therefore cannot establish causality between dyslipidemia and plaque progression. Furthermore, the sample size, though sufficient for statistical significance, may not capture the full heterogeneity of diabetic coronary disease. Longitudinal studies incorporating serial CCTA evaluations could provide stronger evidence of temporal relationships between lipid modulation and plaque regression or stabilization. Additionally, the role of emerging biomarkers such as lipoprotein(a), apolipoprotein B, and inflammatory markers like high-sensitivity CRP warrants exploration to refine risk prediction further.

STRENGTHS OF THE STUDY

The present study demonstrates several methodological and analytical strengths that add to its scientific rigor and clinical value. One of the key strengths lies in its integrative approach, combining a detailed dyslipidemia profile with advanced non-invasive imaging through coronary CT angiography (CCTA). This dual assessment provides a comprehensive view of atheroma development, bridging the gap between metabolic abnormalities and structural coronary pathology. Unlike previous studies that focused solely on lipid levels or calcium scoring, this research simultaneously quantified biochemical risk and morphological evidence of atherosclerosis, allowing for a more precise correlation between metabolic dysfunction and anatomical outcomes. Another major strength is the exclusive focus on diabetic patients compared with matched non-diabetic, which highlights the distinct vascular phenotype of diabetes-associated atherosclerosis and its diffuse, multi-vessel nature. The use of standardized inclusion criteria and objective diagnostic thresholds minimized selection bias and ensured homogeneity across both groups. Furthermore, all CCTA scans were interpreted by experienced observers using the modified American Heart Association classification, ensuring reliability and reproducibility in plaque characterization. Statistical analyses were performed with rigor, providing not only p-values but also meaningful associations between lipid indices and plaque morphology, reinforcing the validity of the observed relationships. Importantly, the study retains strong clinical applicability, as both lipid profiling and CCTA are widely available, making the findings directly translatable to routine practice. This integration of affordable biochemical screening with advanced imaging offers a practical framework for early risk detection and tailored intervention in diabetic populations.

FUTURE DIRECTIONS

While the findings of this study contribute substantially to the understanding of atheroma formation in diabetic patients, they also open several promising avenues for future research. A longitudinal follow-up of the same cohort using serial CCTA assessments would provide critical insights into the dynamic progression or regression of plaques in response to glycemic and lipid control, thereby clarifying causal relationships. Future studies could also expand the biochemical spectrum to include emerging lipid and inflammatory biomarkers such as apolipoprotein B, lipoprotein(a), oxidized LDL, and high-sensitivity C-reactive protein, which may refine cardiovascular risk stratification beyond conventional lipid parameters. Moreover, randomized controlled trials comparing the effects of different lipid-lowering therapies, such as statins, fibrates, or PCSK9 inhibitors, on CCTA-detected

plaque characteristics could identify optimal treatment strategies for diabetic atherosclerosis. Another essential direction involves the incorporation of artificial intelligence and machine-learning algorithms into plaque quantification and pattern recognition, which could enhance the objectivity and reproducibility of non-invasive coronary imaging. Expanding the study to include a larger, more diverse population sample would also strengthen external validity and help establish normative data for diabetic versus non-diabetic coronary morphology. Finally, integrating biochemical and imaging parameters into predictive models could lead to the development of early screening algorithms for asymptomatic diabetic individuals, facilitating proactive management before irreversible vascular events occur. Collectively, these future directions would not only refine diagnostic precision but also guide the evolution of personalized cardiovascular care in diabetes management.

CONCLUSION

This study highlights the significant role of dyslipidemia and coronary CT angiography (CCTA) as complementary, non-invasive indicators for early detection of atheroma formation in diabetic patients. The findings establish that diabetic individuals possess a higher burden of coronary plaques, particularly mixed and non-calcified types, correlating strongly with elevated LDL-C, triglycerides, and reduced HDL-C levels. CCTA effectively delineated the extent and pattern of atherosclerotic involvement, frequently revealing multi-vessel disease in diabetics compared to non-diabetics. Together, these results underscore that combining biochemical lipid profiling with CCTA enhances diagnostic accuracy, offering a comprehensive assessment of both metabolic and structural cardiovascular risk. This integrative approach may facilitate earlier intervention, individualized treatment, and improved outcomes in diabetic populations. Further large-scale, prospective studies are warranted to confirm these findings and to establish standardized protocols for integrating lipid-CCTA assessment into clinical cardiometabolic care.

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Authors' Contributions

All authors contributed to the study, encompassing study conception and design, data collection, analysis and interpretation of results, draft manuscript, reviewed and approved the final version of the manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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