Association between Non-alcoholic Fatty Liver and Coronary Artery Calcification Using CT Coronary Calcium Score Scan

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) and coronary artery disease (CAD) are interrelated conditions linked by shared metabolic risk factors. CT-based coronary artery calcium (CAC) scoring is a valuable tool for cardiovascular risk assessment, but its potential for evaluating hepatic steatosis has not been fully explored.

Objectives: To investigate the association between NAFLD and CAC using CT-based coronary artery calcium scans.

Patients and methods: A retrospective cross-sectional study was conducted involving 404 adult patients referred to cardiovascular screening or CAD risk evaluation between January 2022 and December 2023. Patients were stratified into two groups based on the presence of coronary artery calcification (CAC vs. non-CAC). Hepatic steatosis was assessed through unenhanced CT attenuation values and liver-to-spleen attenuation ratios. Clinical variables including BMI, diabetes, hypertension, and lipid profile were also evaluated. CAC scores were computed using the Agatston method.

Results: Among 404 patients, 236 had detectable CAC. The CAC group exhibited significantly higher BMI (median: 27.5 vs. 25.05 kg/m²; $p<0.001$) and greater prevalence of metabolic comorbidities. Hepatic attenuation values were significantly lower in the CAC group (median liver HU: 38.5 vs. 52; $p<0.001$), as was the liver-to-spleen ratio (0.78 vs. 1.03; $p<0.001$). Strong negative correlations were found between CAC scores and hepatic attenuation ($r = -0.623$) as well as liver-to-spleen ratio ($r = -0.633$). BMI was positively correlated with CAC score ($r = 0.501$; $p < 0.001$) and negatively correlated with hepatic attenuation ($r = -0.761$). Logistic regression identified both BMI and CAC score as significant predictors of fatty liver ($p < 0.001$).

Conclusion: This study demonstrates a strong association between coronary artery calcification and hepatic fat accumulation as detected on routine non-contrast CT scans. CT imaging may thus serve a dual role in evaluating both CAD risk and NAFLD, particularly in populations with elevated BMI and metabolic risk profiles.

Keywords: NAFLD; Coronary Artery Disease; Coronary Artery Calcium Score; CT Imaging; Hepatic Attenuation.

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Introduction

Cardiovascular diseases, especially coronary artery disease (CAD), are the leading global cause of morbidity and mortality. CAD is characterized by the accumulation of atherosclerotic plaques in coronary arteries, resulting in myocardial ischemia or infarction **(Libby et al., 2019)**.

Non-Alcoholic Fatty Liver Disease (NAFLD), a key feature of metabolic syndrome, is now one of the most common chronic liver diseases, impacting about 25% of the global population **(Younossi et al., 2023)**. NAFLD spans from simple steatosis to non-alcoholic steatohepatitis (NASH), which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma. It is also recognized as a systemic disease, contributing to extrahepatic complications, particularly cardiovascular diseases **(Targher et al., 2021)**.

Epidemiological and clinical evidence increasingly supports the link between NAFLD and CAD. Studies show that individuals with NAFLD have a higher prevalence of subclinical atherosclerosis and adverse cardiovascular events, independent of traditional risk factors **(Riazi et al., 2022)**. The underlying mechanisms are multifactorial, involving insulin resistance, dyslipidemia, chronic inflammation, and endothelial dysfunction, all of which are shared pathophysiological pathways between NAFLD and CAD **(Ren et al., 2023)**.

Coronary Artery Calcium (CAC) scoring, measured by computed tomography (CT), is a validated, non-invasive tool for quantifying coronary atherosclerosis. As an independent predictor of future cardiovascular events, CAC has become essential in preventive cardiology for refining cardiovascular risk assessment **(Nasir et al., 2021)**. However, its role in detecting CAD risk in patients with NAFLD remains inadequately explored, particularly across varying degrees of hepatic steatosis.

Our ration was to evaluate the association between NAFLD and CAC using CT-based coronary artery calcium scans.

Patients and methods

This was a retrospective cross-sectional study conducted at a tertiary care academic medical center. The study population comprised 404 consecutive adult patients who were referred to the Radiology department for evaluation of CAD risk or for routine cardiovascular screening. This study was done between January 2022 and December 2024 after approval from Ethical Committee, Faculty of Medicine, Tanta University, Tanta, Egypt (Approval code: 36264PR1226/5/25). An informed written consent was obtained from all patients.

Patients were excluded from the study if they had any known history of alcohol abuse, viral hepatitis, other liver diseases such as cirrhosis or autoimmune hepatitis as well as severe arrhythmia.

Patients were classified into two groups based on the presence of coronary artery calcification as determined by their CT scan results. The first group consisted of 236 patients who had detectable coronary artery calcification (CAC group), while the second group included 168 patients who did not exhibit any detectable CAC (non-CAC group).

Demographic information, including age, sex, height, weight, and BMI, was collected from institutional electronic medical records. Clinical history included the presence of diabetes mellitus, hypertension, dyslipidemia, and smoking status. BMI was categorized as follows: normal weight (<25 kg/m²), overweight (25–29.9 kg/m²), obese (30–34.9 kg/m²), and morbidly obese (≥35 kg/m²).

The CAC group was further stratified based on their total CAC score into four risk categories: not identified (CAC score of 0 AU), low risk (CAC score of 1–99 AU), moderate risk (CAC score of 100–399 AU), and high risk (CAC score of ≥400 AU), in accordance with established guidelines for CAC scoring and risk stratification.

Imaging Protocol

All CT scans were performed using a 160-slice multi-detector CT scanner without contrast administration. For CAC scoring, the Agatston method was used, and scores were calculated using specialized software. The scores for individual coronary segments, including the left main (LM), left anterior descending (LAD), left circumflex (LCx), and right coronary artery (RCA), as well as their branches, were recorded. Hepatic attenuation was measured in both the right and left lobes by placing regions of interest (ROIs), with ROI surface area not less than 150 cm^3, avoiding major vessels and bile ducts, and an average hepatic HU was computed. The spleen was similarly assessed to derive the liver-to-spleen attenuation ratio. Fatty liver severity was semi-quantitatively classified based on HU thresholds: mild (40–49 HU), moderate (30–39 HU), and severe (<30 HU) **(Bozic et al., 2022)**. The liver/spleen ratio was measured by assessing the attenuation values of the liver and spleen on the non-contrast CT images and calculating their ratio. Patients with liver/spleen ratio < 1 were considered to have fatty liver.

Statistical analysis

The analysis was conducted using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were presented as mean ± SD or median (IQR), as appropriate. Between-group differences were assessed with Student's t-test or the Mann–Whitney U test. Categorical variables were reported as frequencies (%) and compared using the Chi-square or Fisher's exact test. A p-value < 0.05 was considered statistically significant.

Results

A total of 404 patients were enrolled in this study with 236 patients with CAC and 168 without. Baseline characteristics were illustrated in **(Table.1)**.

Table 1. Patients' characteristics among the two groups

Variables		Total patients (N=404)	CAC (N=236)	Non- CAC (N=168)	p-value
Sex	**Male**	236 (58.4%)	144 (61.0%)	92 (54.8%)	0.209‡
	Female	168 (41.6%)	92 (39.0%)	76 (45.2%)	
Age (years)	**Mean ±SD** **Range**	50.96±7.96 32- 67	51.1±8.02 32- 67	50.76±7.9 32- 67	0.805*
BMI (Kg/m^2)	**Median (IQR)** **Range**	26.8 (24-29.7) 19.5- 41	27.5 (24.7- 31.2) 19.5- 41	25.05 (23-27.5) 20.6- 31	**<0.001#**
BMI group	**Normal**	148 (36.6%)	64 (27.1%)	84 (50.0%)	**<0.001‡**
	Overweight	160 (39.6%)	96 (40.7%)	64 (38.1%)	0.601‡
	Obese	84 (20.8%)	64 (27.1%)	20 (11.9%)	**<0.001‡**
	Morbid obese	12 (3.0%)	12 (5.1%)	0 (0%)	**0.002‡**
Comorbidities	**DM**	184 (45.5%)	132 (55.9%)	52 (31.0%)	**<0.001‡**
	HTN	220 (54.5%)	148 (62.7%)	72 (42.9%)	**<0.001‡**
	Dyslipidemia	212 (52.5%)	144 (61.0%)	68 (40.5%)	**<0.001‡**
	Smoker	144 (35.6%)	92 (39%)	52 (31%)	0.120‡

p>0.05 is non-significant; p≤0.05 is significant. * Student T test #Mann-Whitney U test, ‡ Chi-square test. BMI: Body mass index, DM: Diabetes Mellitus, HTN: Hypertension.

There was no notable difference in sex distribution between the groups (p=0.209), with males constituting 61.0% of the CAC group and 54.8% of the non-CAC group. Similarly, age did not differ significantly between the groups (mean ± SD: 51.1±8.02 vs. 50.76±7.9 years; p=0.805). However, BMI was significantly

higher in patients with CAC, with a median BMI of 27.5 kg/m² (IQR: 24.7–31.2) compared to 25.05 kg/m² (IQR: 23–27.5) in those without CAC (p<0.001). When categorized, the CAC group showed a significantly higher proportion of obesity (27.1% vs. 11.9%; p<0.001) and morbid obesity (5.1% vs. 0%; p=0.002), while the non-CAC group had a significantly higher proportion of normal BMI (50% vs. 27.1%; p<0.001). The burden of metabolic comorbidities was also significantly higher among patients with CAC. Diabetes mellitus (55.9% vs. 31.0%; p<0.001), hypertension (62.7% vs. 42.9%; p<0.001), and dyslipidemia (61% vs. 40.5%; p<0.001) were all more prevalent in the CAC group. Regarding smoking status, smoking rates did not differ significantly between groups.

As regards patients with CAC, the mean total CAC score was 275.37 ± 480.46 AU, with values ranging from 3 to 2330 AU. Participants were classified into four CAC groups. Over half of the sample (128 participants, 54.2%) fell within the low-risk group (1–99 AU). Moderate CAC scores (100–399 AU) were observed in 68 individuals (28.8%), while 40 participants (16.9%) had severe calcification (≥400 AU). Segmental analysis revealed that the LAD had the highest mean score at 117.96 ± 189.43 AU (range: 0–980), followed by the LCx with 63.73 ± 137.67 AU and the RCA with 61.95 ± 121.84 AU. LM showed a lower mean score of 16.36 ± 34.47 AU, while other branches (including diagonal and obtuse marginal arteries) contributed minimally, with a mean of 15.86 ± 37.93 AU, as shown in **(Table.2).**

Table 2. CA score among CAC groups.

Variables		CAC **(N=236)**
CA score	**Mean± SD** **Range**	275.37± 480.46 3- 2330
CA group:	**Not identified (0 AU)**	0 (0%)
	Low risk (1-99 AU)	128 (54.2%)
	Moderate risk (100-399 AU)	68 (28.8%)
	High risk (≥400 AU)	40 (16.9%)
LM	**Mean± SD** **Range**	16.36± 34.47 0- 220
LAD	**Mean± SD** **Range**	117.96± 189.43 0- 980
LCx	**Mean± SD** **Range**	63.73± 137.67 0- 788
RCA	**Mean± SD** **Range**	61.95± 121.84 0- 560
Others (D, OM)	**Mean± SD** **Range**	15.86± 37.93 0- 184

p>0.05 is non-significant; p≤0.05 is significant. * Student T test #Mann-Whitney U test, ‡ Chi-square test. CA: Coronary artery classification.

(Table.3) showed that the median HU of the left hepatic lobe were significantly lower in the CAC group (39 [IQR: 29–49]) compared to the non-group (53 [IQR: 49–56], p<0.001), with similar findings in the right hepatic lobe (39 vs. 51 HU, p<0.001). The average hepatic attenuation also showed a marked reduction in the CAC group (median 38.5 HU) compared to non-CAC patients (median 52 HU, p<0.001). In contrast, splenic attenuation did not significantly differ between groups (p=0.380). The liver-to-spleen attenuation ratio was significantly lower in the CAC group (median 0.78) than in the non-CAD group (median 1.03, p<0.001). The prevalence of fatty livers was significantly higher in the CAC group compared to the non-CAC group. Specifically, 27.1% of CAC

patients had no fatty liver, while 76.2% of non-CAC patients did not have fatty liver ($p < 0.001$). Mild fatty liver was more common in the CAC group (20.3%) compared to the non-CAC group (9.5%, $p = 0.003$). Moderate and severe fatty livers were also more prevalent in the CAC group (25.4% and 27.1%, respectively) compared to the non-CAC group (9.5% and 4.8%, respectively, both $p < 0.001$).

Table 3. Hepatic Attenuation Indices among the two groups

Variables		Total patients (N=404)	CAC (N=236)	Non-CAC (N=168)	p- value
LT hepatic Lobe HU	**Median (IQR) Range**	48 (34- 54) 17- 68	39 (29- 49) 17-67	53 (49- 56) 29- 68	**<0.001**#
RT hepatic Lobe HU	**Median (IQR) Range**	46 (35- 51) 19- 65	39 (31-47) 19-63	51 (48- 52) 25- 65	**<0.001**#
Average	**Median (IQR) Range**	47 (35- 52) 18- 65	38.5 (30- 48.5) 18- 65	52 (50- 53) 27- 64.5	**<0.001**#
Spleen HU	**Median (IQR) Range**	50 (49- 51) 44- 58	51 (49- 53) 44- 58	50 (49- 51) 48- 53	0.380#
Liver/spleen ratio	**Median (IQR) Range**	0.91(0.68- 1.04) 0.37- 1.24	0.78 (0.58- 1.01) 0.37- 1.20	1.03 (1.01- 1.06) 0.51- 1.24	**<0.001**#
Fatty liver:	**No fatty liver**	206 (51.5%)	64 (27.1%)	128 (76.2%)	**<0.001**‡
	Mild fatty liver	59 (14.9%)	48 (20.3%)	16 (9.5%)	**0.003**‡
	Moderate fatty liver	70 (17.5%)	60 (25.4%)	16 (9.5%)	**<0.001**‡
	Severe fatty liver	65 (16.3%)	64 (27.1%)	8 (4.8%)	**<0.001**‡

p>0.05 is non-significant; p≤0.05 is significant. #Mann-Whitney U test, ‡ Chi-square test.

BMI showed a moderate positive correlation with CA score ($r = 0.501$, $p < 0.001$). In contrast, hepatic attenuation values exhibited strong negative correlations with CA score (including left hepatic lobe HU ($r = -0.636$), right hepatic lobe HU ($r = -0.586$, $p<0.001$), average hepatic HU ($r = -0.623$, $p<0.001$), and liver-to-spleen ratio ($r = -0.633$, $p<0.001$). No significant correlations were found between CA score and patient age ($r = 0.019$, $p = 0.702$) or splenic HU ($r = 0.086$, $p = 0.084$). Both average hepatic attenuation values (HU) and the liver-to-spleen ratio demonstrated strong and significant negative correlations with total CA score ($r = -0.623$ and -0.633, respectively; $p < 0.001$). Similarly, BMI was strongly negatively correlated with these hepatic attenuation metrics (average HU: $r = -0.761$; liver/spleen ratio: $r = -0.740$; $p < 0.001$). (**Table 4, 5 and Fig.1)**

Table 4. Correlation between CA scores, with different parameters

Variables	CA score	
	r	p- value
Age (years):	0.019	0.705‡
BMI (Kg/m^2):	0.501	**<0.001**‡
LT hep Lobe HU	-0.636	**<0.001**‡
RT hep. Lobe HU	-0.586	**<0.001**‡
average	-0.623	**<0.001**‡
spleen HU	0.086	0.084‡

liver/spleen ratio	-0.633	**<0.001**⁋

p>0.05 is non-significant; p≤0.05 is significant. ⁋ Spearman correlation test.

Table 5. Correlation between liver attenuation values with different parameters.

Variables	Average attenuation values (HU)		Liver/spleen ratio	
	r	p- value	r	p- value
Total CA score	-0.623	**<0.001**⁋	-0.633	**<0.001**⁋
Age (years):	-0.077	0.122⁋	-0.090	0.072⁋
BMI (Kg/m²):	-0.761	**<0.001**⁋	-0.740	**<0.001**⁋

p>0.05 is non-significant; p≤0.05 is significant. ⁋ Spearman correlation test

The binary logistic regression analysis revealed that BMI and total CA score were significant predictors of the outcome, while age was not. Specifically, BMI had a significant positive association (B = 0.368, p = 0.001), with an OR of 1.521 (95% CI: 1.157–1.951), indicating that each unit increase in BMI was associated with a 52.1% increase in the odds of the outcome. Total CA score was also significantly associated (B = 0.057, p < 0.001), with an OR of 2.157 (95% CI: 1.017–3.101), suggesting that higher CA scores substantially increased the odds. (**Table.6, Fig.2)**

Table 6. Multivariate logistic regression for cardiovascular risk factors

Variables	B	S.E.	Wald	df	Sig.	OR	95% C.I	
							Lower	Upper
Age	0.042	0.041	1.073	1	0.300	1.043	0.864	1.865
BMI	0.368	0.114	10.454	1	**0.001**	1.521	1.157	1.951
CA score total	0.057	0.020	7.816	1	**<0.001**	2.157	1.017	3.101

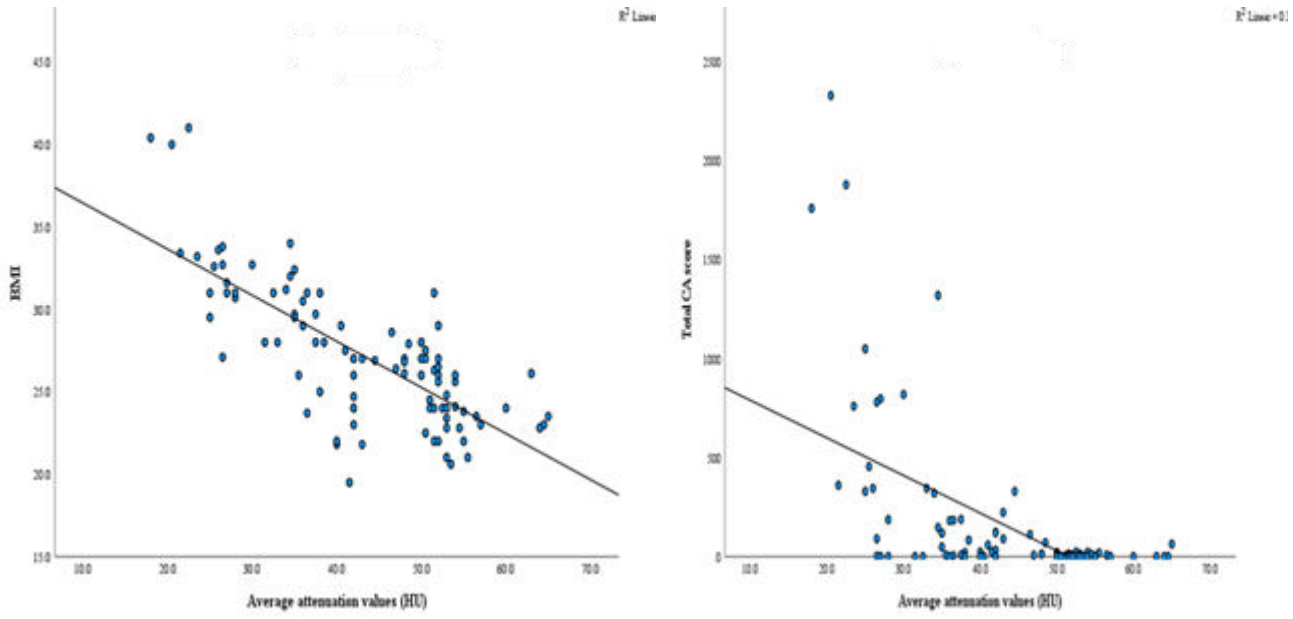

Fig.1. Scatter plot showing correlation between average liver attenuation value with BMI (A) and CA score (B)

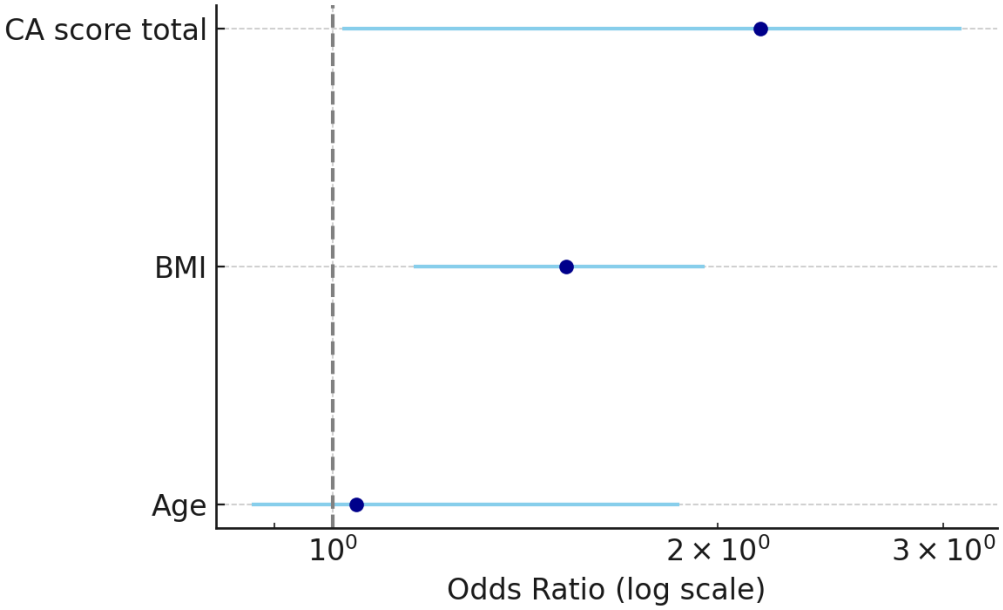

Fig.2. Forest plot for cardiovascular risk factors

Case 1: 64 years old hypertensive male, presented by chest pain and exertional dyspnea.(**Fig. 3).**

Case 2: 54 years old non-diabetic and non-hypertensive male, presented by chest pain. (**Fig.4).**

Case 3: 64 years old hypertensive and diabetic female, presented by chest pain and palpitation. (**Fig.5**).

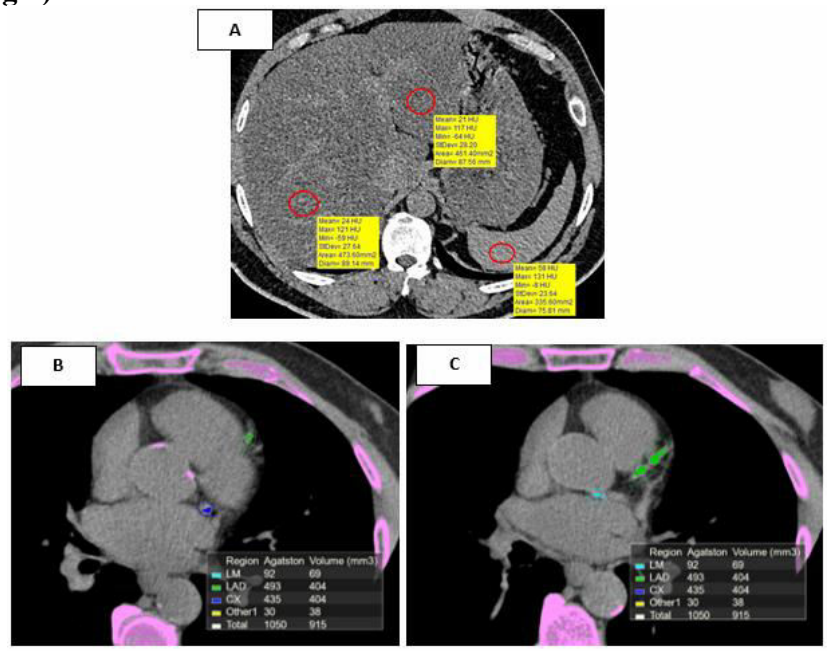

Fig.3. A: an axial non contrast enhanced CT image at the upper abdomen demonstrates low attenuation of the liver in comparison to the spleen (Right hepatic lobe= 24 HU, left hepatic lobe= 21 HU and spleen= 58 HU), with hepatic to splenic ratio=0.38. B & C: Axial non contrast enhanced image of coronary calcium scoring showing severe coronary arterial atherosclerotic calcifications with Agatstone score= 1050

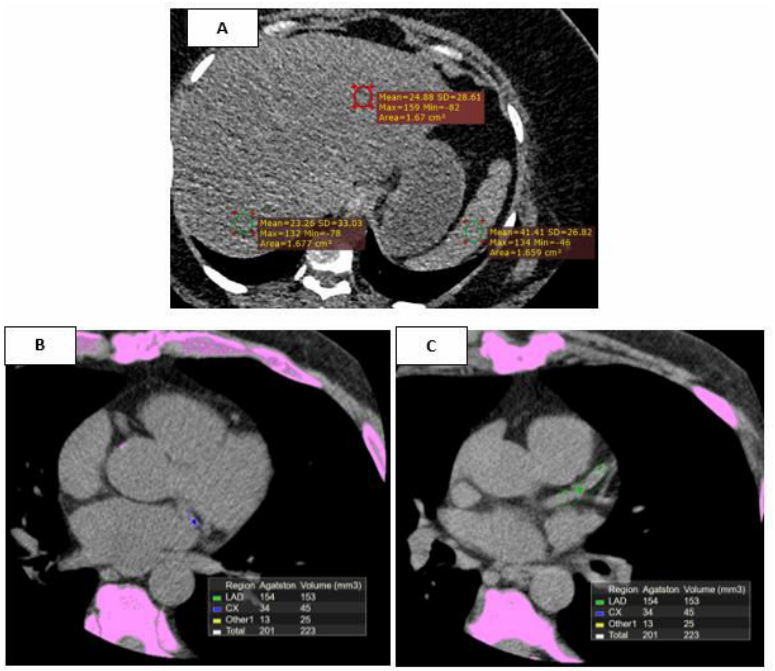

Fig.4. A: An axial non contrast enhanced CT image at the upper abdomen demonstrates low attenuation of the liver in comparison to the spleen (Right hepatic lobe= 23.2 HU, left hepatic lobe= 24.8 HU and spleen= 41.4 HU), with hepatic to splenic ratio=0.57. B & C: Axial non contrast enhanced image of coronary calcium scoring showing moderate coronary arterial atherosclerotic calcifications with Agatstone score= 201.

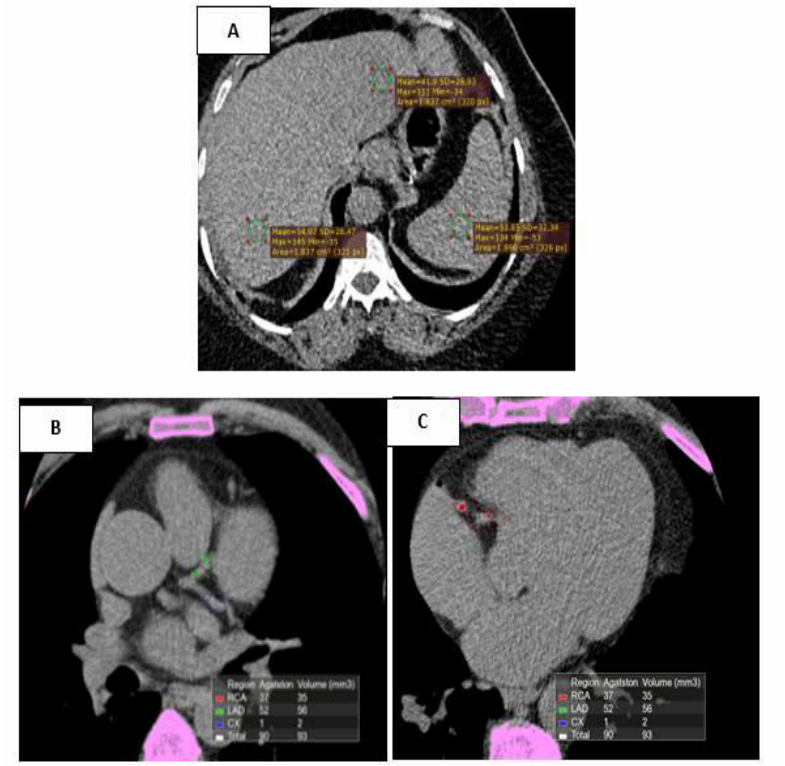

Fig.5. A: An axial non contrast enhanced CT image at the upper abdomen demonstrates mildly low attenuation of the liver in comparison to the spleen (Right hepatic lobe= 54.9 HU, left hepatic lobe= 41.9 HU and spleen= 53.8 HU), with hepatic to splenic ratio=0.94. B & C: Axial non contrast enhanced image of coronary calcium scoring showing mild coronary arterial atherosclerotic calcifications with Agatstone score= 90

Discussion

The primary focus of the current study was on the use of CT to evaluate both hepatic attenuation and coronary artery calcification scores to understand any potential correlations between these two conditions.

In our study, patients with CAC had significantly higher BMI and were more likely to have metabolic comorbidities, including diabetes mellitus, hypertension, and dyslipidemia, compared to those without CAC. These results align a meta-analysis by **Wang et al. (2024)** who found that individuals with metabolic syndrome were at a considerably higher risk for CAD, especially in the presence of NAFLD. Their study reported a two- to threefold increased risk of CAD among NAFLD patients with metabolic syndrome, reinforcing our findings that patients with CAC exhibit a higher prevalence of comorbidities.

Our study's segmental analysis revealed that the LAD had the highest mean CAC score, followed by the LCx and the RCA, with the LAD being the most calcified. This finding is in agreement with previous studies, such as the one by **Sun et al. (2015)**, which used coronary CT angiography to evaluate the distribution of CAC. They found that the LAD consistently had the highest calcification scores, attributed to its hemodynamic characteristics and its role in supplying the anterior wall of the heart. This supports our finding that the LAD was the most affected in our study, with a mean score of 117.96 AU. **Arslan and Yenerçağ (2020)** also observed similar patterns in their study, noting that the LAD is the most commonly calcified vessel, particularly in patients with metabolic diseases like NAFLD.

One of the novel aspects of our study is the investigation of hepatic attenuation values as measured by CT and their relationship with CAC scores. We found that patients with CAC had significantly lower hepatic attenuation values compared to those without CAC, suggesting that increased liver fat content, which correlates with decreased hepatic attenuation, may serve as an indicator of cardiovascular risk in NAFLD patients. These findings are in line with **Cucoranu et al. (2023)** who observed that lower hepatic attenuation values were significantly correlated with higher CAC scores, with a correlation coefficient of $r = -0.31$ ($p < 0.0001$). This aligns with our findings of a strong negative correlation between hepatic attenuation and CAC scores, particularly in the left hepatic lobe ($r = -0.636$) and right hepatic lobe ($r = -0.586$). Their study also emphasized that BMI was negatively correlated with hepatic attenuation ($r = -0.40$, $p < 0.0001$), a finding that supports our own results, where BMI was strongly negatively correlated with hepatic attenuation ($r = -0.761$ for average HU, $r = -0.740$ for liver/spleen ratio, $p < 0.001$).

In contrast, **Moradi et al. (2024)** did not find a notable correlation between NAFLD and CAC scores in their study, which included 365 participants. Their findings suggest that factors other than hepatic fat content, such as epicardial adipose tissue (EAT) thickness and EAT density, may be stronger predictors of CAC burden. While we observed a significant relationship between hepatic attenuation and CAC scores, their results suggest that the pathophysiological mechanisms linking NAFLD and CAC are multifactorial, involving more than just hepatic fat content. Their study identified EAT thickness (OR = 1.803) and EAT density (OR = 0.671) as significant predictors of CAC, indicating that fat deposits around the heart play a crucial role in coronary calcification. These factors were not considered in our study, and future research could explore their interplay with hepatic attenuation in predicting coronary artery disease.

Our findings show a higher prevalence of fatty liver in CAC patients compared to non-CAC patients, aligning

with previous studies that suggest an association between CAD and NAFLD. Similar results have been reported by studies such as **Bozic et al. (2022)**, which found a significant correlation between increased hepatic steatosis and the presence of coronary artery disease. The higher incidence of moderate and severe fatty liver in the CAC group further supports the notion that fatty liver may be a risk factor for cardiovascular diseases, likely due to shared metabolic pathways, such as insulin resistance and chronic inflammation.

However, our study's findings also highlight a noteworthy difference in the distribution of fatty liver severity between groups, with a significantly lower prevalence of severe fatty liver in the non-CAC group, which contrasts with findings of study by **Arslan et al. (2020)** who indicated that more severe forms of fatty liver are closely linked to worse cardiovascular outcomes, suggesting that the severity of fatty liver may be a more critical predictor for CAD than its mere presence. The discrepancy in severity between groups could also be influenced by the heterogeneity of patient populations and diagnostic methods used across different studies.

In our study, we performed correlation and logistic regression analyses to explore the relationships between BMI, hepatic attenuation, and CAC scores. Our results demonstrated a strong negative correlation between hepatic attenuation values and CAC scores, as well as a moderate positive correlation between BMI and CAC scores. This is consistent with findings from **Kim et al. (2012)** study, who observed a similar correlation in a cohort of patients with metabolic syndrome, where higher liver fat content, indicated by lower hepatic attenuation, was associated with more severe coronary artery calcification.

Lastly, **Kumar et al. (2023)** explored the prevalence of CAD in patients with NAFLD and found that 71.9% of NAFLD patients had evidence of CAD, a significantly higher proportion compared to the general population ($p < 0.0001$). Their study also found that CAC scores were significantly associated with NAFLD severity ($p = 0.043$), with more severe NAFLD correlating with higher CAC scores. This finding parallels our own, where patients with CAC had a significantly higher BMI and more severe metabolic comorbidities, underscoring the importance of evaluating both hepatic and coronary health in patients with NAFLD.

Conclusion

Our study found a significant association between higher CAC scores and lower hepatic attenuation, suggests that a CT scan, which is already commonly used for coronary artery assessment, could be utilized as a non-invasive tool for evaluating NAFLD and predicting CAD risk in at-risk populations. Additionally, BMI was positively correlated with CAC scores, underscoring its role in predicting CAD risk.

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