Association of Metabolic Dysfunction-Associated Fatty Liver Disease with Coronary Artery Calcification among Filipino Patients in a Tertiary Hospital in Cebu City

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD), now known as Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), is linked to cardiovascular disease. This renaming emphasizes the role of metabolic problems. Coronary artery calcification (CAC) reflects early coronary artery disease, but data on the MAFLD-CAC link is limited.

Objective: To demonstrate the association between metabolic dysfunction-associated fatty liver disease (MAFLD) based on its criteria and coronary artery calcification, as measured by CT CAC score.

Methods: This single-center retrospective study involved adult Filipino patients who underwent CT CAC scoring between January 2021 and January 2023. Clinical and laboratory data were obtained via review of electronic records.

Results: This study involved 147 patients with an average age of 62 years, primarily females (57.14%), and mostly falling into the Obese-Class I category (31.29%). The most common comorbidities were hypertension (95.24%), dyslipidemia (62.59%), and diabetes mellitus (38.1%). In terms of CAC scores using the CT Agatston method, majority (30.61%) had low calcium buildup (Stage 2 with scores between 1-99). Approximately 26.53% had higher liver fat content with liver HU below 40, while 73.47% had lower liver fat content with HU equal to or greater than 40. Furthermore, 25.17% of patients with fatty livers and other risk factors were diagnosed with MAFLD, while 74.83% were not. The p-value indicated a significant difference in proportions, suggesting a lower proportion of MAFLD among those who had undergone CT CAC scoring. However, the Pearson Chi-Square statistic (4.051) and the p-value (0.256) indicated no statistically significant association between MAFLD and CT CAC.

Conclusion: The study found a notably lower proportion of MAFLD diagnoses in patients who underwent CT CAC scoring. Additionally, there was no statistically significant link between MAFLD and CT CAC.

Keywords: MAFLD, Coronary Artery Calcification

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a progressively common condition with a global prevalence of ~ 25–30%. It is significantly associated with the burden of cardiovascular disease.[1] A link exists between metabolic syndrome and non-alcoholic fatty liver disease which initially begins as steatohepatitis and eventually progresses to cirrhosis and hepatocellular carcinoma.[2] In 2020, an international panel of experts led a consensus-driven process to change the name NAFLD into a term that highlights the contribution of systemic metabolic dysregulation, hence the term "metabolic dysfunction-associated fatty liver disease," or "MAFLD".[1] The consensus also projected a set of simple positive criteria to diagnose and assess individuals for the disease process.[3]

Coronary artery calcification (CAC) is an indicator of subclinical coronary artery disease, correlated strongly with the extent and progression of atherosclerosis, and it represents the atherosclerotic burden in arterial beds, specifically the coronary arteries.[4] The Agatston score is the most widely used CAC scoring system in clinical practice and research.[5] In current practice, the multi-detector computed tomography (MDCT) is a non-invasive method to diagnose coronary artery disease in patients with mild or moderate risk factors. The use of CAC scoring has also been emphasized in the latest 2018

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Table I. Agatston Score, Plaque Burden, and Degree of Coronary Artery Obstruction

Agatston Score	Plaque Burden	Probability of Significant CAD
0	No plaque	Very low
1-99	Minimal to at least mild	Low to mild/minimal
100-399	At least moderate	Non-obstructive CAD, likely, although obstructive disease possible
400 and above	Severe/excessive	High likelihood of at least one significant coronary artery stenosis

ACC/AHA Cholesterol guidelines, to guide in the decision of initiating or withholding statin therapy in intermediate-risk or selected borderline-risk adults.[6]

The relationship between MAFLD and CAC has been a topic of interest among various researchers, and the presence of MAFLD, independent of the other traditional risk factors, increases one's risk for cardiovascular events and even mortality.[7]

Objectives: The general objective is to demonstrate the association between metabolic dysfunction-associated fatty liver disease (MAFLD) based on its criteria and coronary artery calcification, as measured by CT CAC score. The specific objectives are the following: (1) To determine the baseline clinical characteristics and risk factors of Filipino patients in a tertiary hospital in Cebu City who underwent CT CAC, in terms of the following: age, sex, BMI (kg/m^2), comorbidities, medication history, blood pressure, laboratory findings. (2) To diagnose fatty liver disease using the National Institute of Health-Multi-Ethnic Study of Atherosclerosis (NIH-MESA) CT diagnostic criteria among patients who underwent CT CAC, (3) To classify patients as MAFLD or no-MAFLD based on its diagnostic criteria: Fatty liver on CT scan, one or more of the three metabolic conditions: Overweight or obesity (BMI $\geq$ 23 kg/m^2), Type 2 Diabetes Mellitus, and at least two of seven metabolic risk abnormalities, and (4) To determine the extent of the patient's coronary artery calcium score and its relation to fatty liver disease

Operational Definition of terms: Fatty liver is diagnosed by the NIH-MESA CT criteria as a liver HU attenuation of < 40, signifying a liver fat content of > 30%.[9] Metabolic dysfunction-associated fatty liver disease is defined as the evidence of fatty liver on CT scan in the presence of one or more of the following three metabolic conditions: Overweight or obesity (BMI $\geq$ 23 kg/m^2), Type 2 Diabetes Mellitus, and at least two of seven metabolic risk abnormalities: waist circumference $\geq$ 90 cm in men and $\geq$ 80 cm in women, serum triglyceride level $\geq$ 150 mg/dL, HDL level < 40 mg/dL in men or < 50 mg/dL in women, systolic blood pressure $\geq$ 130 mm Hg or diastolic blood pressure $\geq$ 85 mmHg or taking antihypertensive medications, prediabetes status based on levels of fasting glucose (100–125 mg/dL)] or hemoglobin A1c (5.7%–6.4%), homeostasis model assessment-estimated insulin resistance score (HMAIR) $\geq$ 2.5, and plasma hs-CRP (highly sensitive C reactive protein) level $\geq$ 0.2 mg/dL. Coronary artery calcium score was measured using a 128-slice MDCT scanner. Calcification in the coronary arteries was defined as high-density lesion > 130 HU according to the Agatston method. The probability of significant CAD and plaque burden according to the Agatston score is shown in *Table I*. The total CAC score was categorized into four stages[8,10]:

- CAC Stage 1 was defined as a total CAC score of 0
- CAC Stage 2 as a total CAC score between 1-99
- CAC Stage 3 as a total CAC score between 100-399
- CAC Stage 4 as a total CAC score of more than or equal to 400

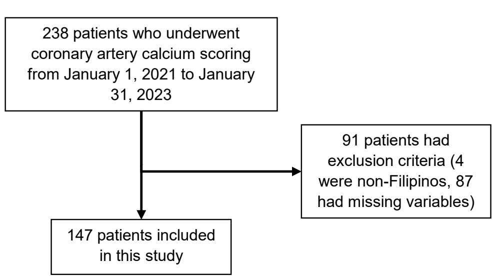

Figure 1. Schematic illustration depicting patient selection for the study

Methodology

This was a single-center, observational retrospective study conducted at a 660-bed capacity, private tertiary hospital located in Cebu City, with approval from the Institutional Review Board. This study included records from January 1, 2021 to January 31, 2023 of patients >18 years old who underwent coronary artery calcium scoring. This data excluded records from patients who were non-Filipinos and those with missing variables like total cholesterol, triglycerides, VLDL, LDL-C, HDL-C, FBS and HbA1c.

A total of 238 patients underwent coronary artery calcium scoring during the study period. The assessment forms and medical records of these patients were obtained at the CT scan unit of the Department of Radiology and reviewed to identify those who fit in the criteria. Among these patients, 91 were excluded due to presence of exclusion criteria. Hence, a total of 147 patients were included in the study (*Figure 1*).

Table II. The Baseline Characteristics of Filipino Patients in a Tertiary Hospital in Cebu City who underwent CT CAC, n = 147

Characteristics	Category
Age in years, Mean (SD)	61.77 (11.21)
Male	63 (42.86)
Female	84 (57.14)
Body Mass Index (BMI), Mean (SD)	26.67 (5.02)
Underweight (<18.5)	5 (3.40)
Normal Weight (18.5-22.9)	25 (17.01)
Overweight (23-24.9)	27 (18.37)
Obese-Class I (25-29.9)	46 (31.29)
Obese-Class II (≥30)	44 (29.93)
Blood Pressure	
<130/85	60 (40.82)
≥130/85	87 (59.18)
Comorbidities	
Hypertension	140 (95.24)
Diabetes Mellitus	56 (38.10)
Dyslipidemia	92 (62.59)
Cerebrovascular Disease	8 (5.44)
Viral Hepatitis	1 (0.68)
Medication History	
Anti-platelet/Anticoagulants	81 (55.10)
Anti-hypertensives	142 (96.60)
Anti-diabetics	53 (36.05)
Lipid-lowering	92 (62.59)
Total Cholesterol, Mean (SD)	179.04 (47.09)
<130	22 (14.97)
130-200	80 (54.42)
>200	45 (30.61)
Triglycerides, Mean (SD)	116.45 (62.97)
<150	119 (80.95)
≥150	28 (19.05)
VLDL, Mean (SD)	23.12 (12.58)
<50	142 (96.60)
>50	5 (3.40)
LDL-C, Mean (SD)	101.21 (41.56)
<70	36 (24.49)
70-189	108 (73.47)
≥190	3 (2.04)
HDL-C (Male), n=63, Mean (SD)	45.63 (14.28)
<40	25 (39.68)
≥40	38 (60.32)
HDL-C (Female), n=84, Mean (SD)	61.42 (19.31)
<50	27 (32.14)
≥50	57 (67.86)
FBS, Mean (SD)	112.34 (34.10)
<100	59 (40.14)
100-125	62 (42.18)
≥126	26 (17.69)
HbA1c, Mean (SD)	5.93 (1.02)
<5.7%	58 (39.46)
5.7-6.4%	65 (44.22)
≥6.5%	24 (16.33)
SGPT, Mean (SD)	41.55 (31.92)
<50	108 (73.47)
≥50	39 (26.53)
Creatinine, Mean (SD)	1.32 (1.67)
<1.5	132 (89.80)
≥1.5	15 (10.20)

Data Collection Process. All CT CAC examinations were performed on a 128 channel multidetector CT scanner, with prospective acquisition, without use of IV contrast medium. The examinations were reviewed by a cardiac imaging specialist. Patients were classified according to the presence of calcified and non-calcified atheromatous disease in coronary arteries. Calcifications in the coronary arteries were defined as high-density lesion > 130 HU according to the Agatston method. The total CAC score was categorized into four stages: CAC stage 1 was defined as a total CAC score of 0, CAC stage 2 as a total CAC score between 1-99, CAC stage 3 as a total CAC score between 100-399, and stage 4 as a total CAC score of ≥ 400. Attenuation of hepatic parenchyma was measured by a Radiology resident in the visualized liver segments in HU by applying a region of interest (ROI) of at least 5 cm^2. Fatty Liver was diagnosed based on the NIH-MESA CT criteria with a liver HU attenuation of < 40, signifying a liver fat content of > 30%. The collected data were encoded and tabulated manually in a Microsoft Excel® spreadsheet with coded identifiers and were stored in a secure, password-protected laptop.

Results

A total of 147 patient records, covering three years from January 1, 2021, to January 31, 2023, were examined. The baseline characteristics of patients who underwent CT CAC were shown in *Table II*. On average, the participants were about 62 years old, and the majority of them were women, making up 57.14% of the group. The largest portion of patients, around 31.29%, fell into the Obese-Class I category, with an average body mass index (BMI) of 26.67 kg/m^2. Following closely behind were those in the Obese-Class II category, accounting for 29.93% of the population, with the overweight group at 18.37%, normal weight at 17.0%, and the underweight category at 3.40%.

Approximately 59.1% of patients had BP readings ≥ 130/85 mmHg, while 40.82% had readings < 130/85 mmHg. Furthermore, the prevailing comorbidity observed was hypertension, affecting 95.24% of the participants, with dyslipidemia coming next at 62.59%, and diabetes mellitus at 38.1%. The vast majority of the population, totaling 96.6%, were taking medications for hypertension. Additionally, 62.59% were on lipid-lowering medications, 55.1% were on antiplatelets/anticoagulants, and 36.05% were using anti-diabetic medications.

For the blood tests, the majority (54.42%) exhibited total cholesterol levels falling within the range of 130-200 mg/dl, with an average total cholesterol level of 179.04 mg/dl. Approximately 80.95% of the population had triglyceride levels < 150 mg/dl, with an average of 116.45 mg/dl. Moreover, 96.6% showed VLDL levels < 150 mg/dl, with an average of 23.12 mg/dl. Most patients (73.4%) had LDL levels within the range of 70-189 mg/dl, with an average of 101.21 mg/dl. As for HDL levels, the majority of males (60.32%) had results ≥ 40 mg/dl, averaging at 45.63 mg/dl. Most females (67.86%) on the other hand, displayed levels ≥ 50 mg/dl, with an average of 61.42 mg/dl. Within the study group, the average FBS level was 112.34 mg/dl. Majority (42.1%) of the group had impaired fasting glucose, defined as FBS levels

Table III. The Patient's Coronary Artery Calcium Score, n = 147

CT Agatston Coronary Artery Calcium score	Value
Mean Score (SD)	442.14 (909.95)
Stage	
Stage 1 (0)	34 (23.13)
Stage 2 (1 – 99)	45 (30.61)
Stage 3 (100 – 399)	28 (19.05)
Stage 4 (>400)	40 (27.21)

Table IV. The Patient's Liver Hounsfield Unit, n = 147

Liver Hounsfield Unit	Value
Mean Score (SD)	46.66 (10.96)
< 40	39 (26.53)
≥ 40	108 (73.47)

Note: Values are presented in Frequency (Percentage) unless otherwise stated

between 100-125 mg/dl. About 40.1% had normal FBS levels < 100 mg/dl while 17.6% were in the diabetic category, characterized by FBS levels ≥ 126 mg/dl. Regarding glycosylated hemoglobin (HbA1c), the majority (44.22%) displayed values ranging from 5.7 to 6.4%, with an average of 5.93%, indicating impaired glucose tolerance. Additionally, 73.47% had SGPT levels below 50 U/L, with an average value of 41.55 U/L. Furthermore, 89.8% exhibited creatinine levels < 1.5 mg/dl, with an average value of 1.32 mg/dl.

The coronary artery calcification scores, which were determined using the CT Agatston method are presented in *Table III*. The data unveiled a mean score of 442.14, along with a standard deviation of 909.95, signifying a broad spectrum of scores. An examination of the data illustrated a noteworthy disparity in calcium accumulation among the patients. The majority (30.61%) were categorized as having a low level of calcium buildup (Stage 2). Moreover, 27.21% displayed a substantial amount of calcium buildup (Stage 4). Approximately 23.13% of patients showed no detectable calcium (Stage 1) and about 19.05% presented a moderate degree of calcium accumulation (Stage 3).

Data pertaining to the HU of the patients' livers, are presented in *Table IV*. On average, the HU value for these patients was 46.66, with some degree of variability around this mean. Regarding the diagnosis of fatty liver, the data revealed that among the 147 patients examined, approximately 26.53% (39 patients) exhibited liver HU values < 40, indicating a higher liver fat content. The remaining 73.47% (108 patients) exhibited liver HU values ≥ 40, suggesting a lower liver fat content.

Note: Values are presented in Frequency (Percentage); Fatty Liver was diagnosed based on the NIH-MESA CT criteria with a liver HU attenuation of <40, signifying a liver fat content of >30%.

Table V offered an insight into the categorization of the study population in terms of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD). The information disclosed that 37 patients (25.17%) had received a diagnosis of MAFLD, whereas 110 patients (74.83%) had not been diagnosed with MAFLD. The p-value indicated a substantial contrast in the proportions. It strongly implied that there was a notably lower percentage of patients who had been identified as having MAFLD.

A crosstabulation of the study population, classifying them based on the presence or absence of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and their Coronary Artery Calcification category using the CT Agatston method are shown in *Table VI*. The Pearson Chi-Square statistic (4.051) and the p-value (0.256) indicated that there was no statistically significant association between MAFLD and coronary artery calcification based on this data.

Table V. Patients as MAFLD or no-MAFLD based on Given Diagnostic Criteria, n = 147

Patients with		p-Value
Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD)	No Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD)	
37 (25.17)	110 (74.83)	<0.001 *

Note: Values are presented in Frequency (Percentage); * Significantly different population proportions at 0.05

Table VI. The Crosstabulation of Patients' Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and Coronary Artery Calcification, n = 147

CT Agatston Coronary Artery Calcium Category	Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD)		Total
	Without MAFLD	With MAFLD	
Stage 1	25	9	34
Stage 2	35	10	45
Stage 3	24	4	28
Stage 4	26	14	40
Total	110	37	147
Pearson Chi-Square	4.051		
p-Value	0.256		

Discussion

The CAC score indicated presence and extent of coronary atherosclerosis, and were highly predictive of future cardiovascular events independent of traditional risk factors.[4] In our study, we noted that within the group of patients who underwent CAC, there was a higher prevalence among females, particularly in individuals over 62 years of age, and those categorized as Obese-I, defined by having a BMI between 25-29.9 kg/m^2. Within our study population, the most prevalent comorbidities were hypertension, dyslipidemia, and diabetes, with a majority of participants having hypertension, as evidenced by blood pressure readings $\geq$ 130/85 mmHg.

When compared to the study conducted by Hermosilla and colleagues, which included 221 patients who underwent calcium scoring, their research participants had an average age of 56 years (SD ± 11) and were predominantly men.[11] Prior literature also mentioned dyslipidemia, hypertension, and diabetes as the most frequently observed comorbidities.[8,11] Notably, the connection between obesity and diabetes with the severity of coronary artery disease was emphasized in the study by Kirby and colleagues. This relationship was attributed to insulin resistance, which promotes the accumulation of fatty acids and hastens the development of atherosclerosis.[12] In a study by Kim, et al, fasting blood glucose, LDL-cholesterol, triglyceride and SGPT levels were significantly higher among their study population.[13] Abnormally high lipid (triglycerides, total cholesterol, LDL, HDL) or liver panels AST, ALT, alkaline phosphatase, and total bilirubin) were also noted in the study by Kirby, et al but it had had no effect on the presence of CAD.[12] Nevertheless, as observed in our study, majority had impaired fasting blood glucose with average FBS levels of 112.34 mg/dl. The levels of triglycerides, VLDL, LDL, HDL and SGPT were low to normal.

In the study conducted by Kim et al., it was found that a high CAC score served as an indicator of an elevated risk of coronary events, whereas a CAC score of zero was associated with an exceptionally low risk of subsequent coronary events.[13] Another observational study revealed that patients with a CAC score > 0 had a significantly higher risk of developing further coronary artery disease and experiencing all-cause mortality.[8] In our study, the analysis of coronary artery calcification scores indicated that the majority of participants fell into the category of having a low level of calcium buildup (Stage 2) or had CAC scores ranging from 1 to 99, signifying a low to mild or minimal likelihood of significant coronary artery disease.

Hermosilla and colleagues conducted a study that demonstrated the use of multi-slice computed tomography (MSCT) to diagnose fatty liver by detecting low attenuation in the hepatic parenchyma. This approach showed a sensitivity of 82% and a specificity of 100%. Hepatic steatosis was found to be strongly associated with various metabolic disorders, including insulin resistance, and had been suggested as a unifying factor for these metabolic issues.[11] In our study, we observed that the mean liver HU value for patients who had CAC was 46.66, with some degree of variability. Within the study population, only approximately 26.53% of individuals were diagnosed with fatty liver.

Non-alcoholic fatty liver disease (NAFLD) has been associated with cardiovascular disease.[1] Its growing burden matched the rising occurrence of obesity in Asia, now estimated to be 29.6% and may have exceeded that in Western populations.[8] The newly proposed term MAFLD depicted the key role of cardiometabolic risk factors and other features of the metabolic syndrome in the pathophysiology of this common and burdensome liver disease.[14] Recently, there had been reports in cross-sectional or cohort studies showing an association between MAFLD and CVD, and in both cases, the risk ratios for CVD were higher in MAFLD populations than in NAFLD populations.[15]

Our study revealed that among 147 patients who underwent CAC scoring, a significantly lower proportion of patients (25.17%) had been diagnosed with MAFLD. In a prospective community-based cohort of 6,232 participants, who were followed for a median of 4.3 years, Liu et al. stated that MAFLD was associated with a greater risk of developing subclinical atherosclerosis.[16] Systemic low-grade inflammation, endothelial dysfunction, increased oxidative stress, insulin resistance and an atherogenic lipoprotein profile were among the multiple cardiometabolic risk factors that were common between MAFLD and CVD.[1]

Furthermore, in a study by Sung et al. using the *Kanbguk Samsung Health Study* cohort database, both NAFLD and MAFLD were associated with higher risk of developing incident CAC, even after adjusting for age, sex, educational level, smoking, physical activity, pre-existing coronary artery disease, plasma low density lipoprotein (LDL)-cholesterol concentrations, or use of lipid-lowering agents. Nevertheless, these associations were stronger for MAFLD.[14]

The relationship between MAFLD and CAC was not consistent in various studies. For instance, McKimmie et al. found no significant connection between hepatic steatosis and CAC in a group of patients with type 2 diabetes. Strong correlations between pro-atherogenic biomarkers and components of the metabolic syndrome indicated that hepatic steatosis might signify more than just general adiposity; it could represent a systemic, inflammatory, pro-atherogenic adipose state.[17] In our study, the results did not show a statistically significant link between MAFLD and coronary artery calcification. This suggested that hepatic steatosis may not be a direct mediator of subclinical cardiovascular disease but could be an associated phenomenon.

It is important to note that our study has certain potential limitations, including a small sample size, uncertainty about the timing of associations, and the potential for selection bias in participants with pre-existing diseases.

Conclusion

This study provided insights into the crucial roles of hypertension, dyslipidemia, and diabetes in the development of coronary artery calcification, which in turn increase the susceptibility of patients to coronary artery disease. The prevalence of obesity also played a part in the occurrence of fatty liver and cardiovascular diseases. However, despite the belief that MAFLD might have a pathophysiological role in atherosclerosis and indicate an elevated risk of coronary disease, our study did not find a significant association between MAFLD and coronary artery calcification. Therefore, further research is recommended to expand the existing body of literature aiming to unravel the relationship between MAFLD and CAC and their contributions to the development of cardiovascular events.

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