**DeepCatch-Derived Body Composition Metrics for Noninvasive Prediction of MASLD Subtypes, Fibrosis, and Liver-Related Outcomes**

Dong Hyun Kim1\*, Na Hyun Cho1\*, Hye Won Lee2-4

1 Yonsei University College of Medicine, Seoul, Korea 2 Department of Internal Medicine, Yonsei University College of Medicine, Seoul, Korea  
3 Institute of Gastroenterology, Yonsei University College of Medicine, Seoul, Korea 4 Yonsei Liver Center, Severance Hospital, Seoul, Korea

\*These authors contributed equally to this work.

**Corresponding authors**

Hye Won Lee, M.D., Ph.D.

Department of Internal Medicine, Yonsei University College of Medicine

50-1 Yonsei-ro, Seodaemun–gu, Seoul, 03722, South Korea

Tel: +82-2-2228-2288, E-mail: [lorry-lee@yuhs.ac](mailto:lorry-lee@yuhs.ac)

**Manuscript word count:**

**Total number of figures and tables:** figures, table (Supplementary: figures, table)

**Abbreviations:**

**Abstract**

**Background/Objectives:**

**Methods:**

**Results:**

**Conclusion:**

**Abstract word count:**

**Keywords:**

**Introduction**

The global burden of metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is rapidly rising in parallel with increasing rates of obesity and type 2 diabetes, with adult prevalence projected to exceed 55% by 2040 [1]. MASLD presents significant diagnostic challenges, particularly in identifying patients with metabolic dysfunction-associated steatohepatitis (MASH), the progressive and clinically significant form of the disease. While liver biopsy remains the gold-standard for diagnosing MASH and staging liver fibrosis, its invasiveness, cost, risk of complications, and sampling variability limit its utility in widespread clinical practice [2, 3].

Among non-invasive approaches, ultrasound-based imaging is commonly regarded as the clinical standard for initial evaluation of hepatic steatosis [4]. However, its diagnostic performance may be limited in individuals with high visceral adiposity, as ultrasound may not adequately capture the full spectrum of disease severity in these patients [5]. In contrast, cross-sectional imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) offer higher sensitivity and reproducibility in quantifying liver fat and evaluating abdominal body composition.

Recently, Deep Learning based software such as DeepCatch have emerged, enabling automated extraction and analysis of CT-derived body composition metrics—including visceral fat area (VFA), skeletal muscle area (SMA), and liver–spleen attenuation and volume—within seconds. In this study, we aim to evaluate whether DeepCatch-generated CT biomarkers can serve as reliable surrogates for non-invasive diagnosis of MASLD phenotypes, including steatosis severity and fibrosis staging, offering a potential alternative to invasive liver biopsy.

This study aims not only to validate DeepCatch-derived metrics against established clinical benchmarks but also to assess their clinical utility in redefining non-invasive standards for MASLD diagnosis and monitoring in the era of precision medicine.

**Methods**

**Patient Selection**

This retrospective study included patients who underwent abdominal computed tomography (CT) imaging between March 2006 and July 2023. The date of the CT scan was used as the reference point for deriving body composition metrics using DeepCatch, an automated image-processing software.  
Patients were eligible for inclusion if they had available CT imaging along with corresponding laboratory or FibroScan data obtained within 1 year and 3 months of the CT date. Liver biopsy results and clinical information were reviewed to determine eligibility. Patients were excluded if they lacked definitive liver pathology or had liver disease attributed to non-metabolic etiologies such as viral hepatitis, cholestatic liver disease, or hepatic injury unrelated to metabolic dysfunction.

For liver-related event (LRE) analysis, the LRE date was defined as the time difference between the CT scan and the earliest documented event (ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome, or liver transplantation). For patients without an event, the duration from CT to the last follow-up was used.

**MASLD Spectrum Classification Based on Histology**

The MASLD spectrum was categorized into four groups—None, MASLD, MASH, and Cirrhosis—based on liver biopsy findings, using the NAFLD Activity Score (NAS) and fibrosis staging from the NASH CRN and SAF criteria. “None” included patients without hepatic steatosis. “MASLD” included patients with hepatic steatosis ≥5% who did not exhibit definitive histologic features of steatohepatitis. This group encompassed both typical simple steatosis cases (NAS < 4) and probable MASH cases with NAS = 4 in the absence of marked ballooning or inflammation. “MASH” required steatosis, lobular inflammation, and ballooning each scoring ≥1, with NAS ≥ 5. “Cirrhosis” was defined by fibrosis stage 4, regardless of the presence of active steatohepatitis.

**Study Design**

CT images were analyzed using DeepCatch, which automatically identifies the third lumbar vertebral (L3) level and sets the region of interest from the lower ribs to the iliac crest.

The software quantifies adipose tissue, skeletal muscle, and organ characteristics using both cross-sectional areas at the L3 level and volumetric data across multiple slices. Visceral and subcutaneous adipose tissue were measured by area (cm²) and volume (cm³), and their attenuation in Hounsfield Units (HU) was used to assess fat quality. Skeletal muscle area and muscle attenuation were similarly assessed at the L3 level.

Liver and spleen attenuation values were obtained from precontrast CT scans, and the liver-to-spleen attenuation ratio was calculated as a surrogate marker for hepatic fat accumulation. Liver and spleen volumes were measured using multi-slice segmentation. All area-based metrics were normalized by height squared (cm²/m²), generating indices such as the visceral fat index (VFI) and skeletal muscle index (SMI). While DeepCatch supports all CT phases, precontrast images were used for attenuation analyses to ensure consistency with prior literature. Area and volume measurements are considered stable across imaging phases.

**Endpoints**

The study evaluated four primary outcomes: 1) Classification of MASLD vs no MASLD, 2) Identification of moderate-to-severe steatosis (steatosis score ≥ 2), 3) Prediction of advanced fibrosis (stage ≥ 3), 4) Time to LRE, defined as the first occurrence of ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome, or liver transplantation.

**Statistical Analyses**

All analyses were performed using Python 3.13.3. Continuous variables were dichotomized at the median for odds and hazard ratio analyses. To identify independent predictors of MASLD type, steatosis severity, and fibrosis stage, variables significant in univariable logistic regression (P < 0.05) were entered into multivariable models with backward stepwise selection (removal threshold: P > 0.05). Final models were evaluated using the area under the receiver operating characteristic curve (AUC), and 95% confidence intervals (CIs) were estimated via bootstrap resampling (1,000 iterations, using the 2.5th and 97.5th percentiles).

For LRE analysis, Kaplan–Meier curves were compared using log-rank tests. Univariable and multivariable Cox proportional hazards models with backward stepwise selection were used to estimate hazard ratios (HRs). A two-sided p-value < 0.05 was considered statistically significant.

**Results**

**Patients**

A total of 292 patients who underwent abdominal CT imaging and evaluation using the DeepCatch program, along with available liver biopsy results, were initially screened. Seven patients were excluded due to the absence of definitive liver pathology findings. The remaining 285 patients without cancer-related biopsy results were considered eligible for analysis. Of these, 63 were excluded due to liver disease of non-metabolic etiology, including injury-related, chronic viral, or cholestatic hepatitis. Ultimately, 229 patients with complete DeepCatch-derived metrics and biopsy-confirmed histological data were included in the final analysis (**Figure 1**).

The follow-up duration, defined as the time from baseline CT or FibroScan date to the last follow-up or LRE, had a median of 65.1 months (IQR: 46.2–113.3 months). Baseline characteristics for the overall cohort and stratified comparisons between advanced and non-advanced fibrosis groups are summarized in **Table 1**.

**Distribution of Traditional and DeepCatch-Derived Metrics Across MASLD Subtypes**

We compared the distribution of both traditional and DeepCatch-derived metrics across the four MASLD categories (None, MASLD, MASH, and Cirrhosis). Fibrosis-related markers such as LSM and FIB-4 demonstrated a progressively increasing trend across the disease spectrum, with values rising from None to MASLD, MASH, and peaking in Cirrhosis (**Figure 2**). This gradient reinforces their established role as indicators of fibrosis severity.

In contrast, steatosis-related metrics—including BMI, CAP, visceral fat area, and the liver-to-spleen attenuation ratio (Liver/Spleen HU)—exhibited non-linear, U-shaped or inverse U-shaped distributions. These variables increased from the None group to MASH but declined in patients with cirrhosis. Additional DeepCatch-derived metrics shown in **Supplementary Figure 1**, including liver attenuation, liver PDFF, subcutaneous fat area, and visceral fat area demonstrated similar U- or inverse U-shaped distributions across disease stages. This pattern aligns with known histological regression of hepatic steatosis in advanced fibrosis due to hepatocyte dropout, altered lipid metabolism, and fibrotic remodeling.

**Prediction of MASLD Type**

To identify predictors of MASLD presence, multivariable logistic regression with backward stepwise selection was performed. The final model showed an AUC of 0.970 (95% CI: 0.928-0.995) **(Figure 3A)**. Higher VFA volume (OR = 130.8, 95% CI: 5.4–3153.7, *p* = 0.003), waist circumference (OR = 70.7, *p* = 0.001), CAP (OR = 14.2, *p* = 0.016), and DBP (OR = 11.4, *p* = 0.017) were strongly associated with MASLD. In contrast, higher VFI (OR = 0.007, *p* = 0.002), LDL (OR = 0.03, *p* < 0.001), PT INR (OR = 0.11, *p* = 0.016), and Liver/Spleen HU (OR = 0.14, *p* = 0.023) were inversely associated.

**Prediction of Steatosis Severity**

To identify independent predictors of moderate-to-severe steatosis (score ≥ 2), a logistic regression model was constructed using backward stepwise selection. The final model achieved an AUC of 0.825 (95% CI: 0.765-0.879) (**Figure 3B**). Liver/Spleen HU was the most significant negative predictor (OR = 0.22, 95% CI: 0.11–0.44, *p* < 0.001), while CAP was positively associated with steatosis severity (OR = 2.76, 95% CI: 1.40–5.44, *p* = 0.003). Additional predictors included visceral fat attenuation (OR = 0.39, *p* = 0.008), PT INR (OR = 0.36, *p* = 0.004), eGFR (OR = 3.11, *p* = 0.002), and female sex (OR = 2.52, *p* = 0.008).

**Prediction of Fibrosis Severity**

For advanced fibrosis (stage ≥ 3), multivariable logistic regression with backward stepwise selection was performed using variables significant in univariable analysis (excluding LSM). The final model retained seven independent predictors: Sketeal Muscle Area, AST, PT INR, LDL, diabetes/prediabetes status, ALT, and CAP. This model yielded strong discriminatory performance with an AUC of 0.835 (95% CI: 0.769-0.891) (**Figure 3C**). Higher sketeal muscle area was significantly protective (OR: 0.27, 95% CI: 0.12–0.59, p = 0.001), while elevated AST (OR: 6.04, 95% CI: 2.32–15.73, p < 0.001) and PT INR (OR: 3.06, 95% CI: 1.42–6.61, p = 0.004) were associated with greater fibrosis risk. Other protective factors included lower LDL and CAP values, while the presence of diabetes or prediabetes remained a significant risk factor (OR: 1.89, p = 0.003).

For significant fibrosis (stage ≥ 2), multivariable logistic regression with backward stepwise selection identified six independent predictors: FIB-4, SMA (Area), LDL, diabetes/prediabetes status, waist-to-height ratio, and PT INR. The final model achieved an AUC of 0.797 (95% CI: 0.759-0.877) (**Supplementary Figure 2**). FIB-4 (OR: 3.81, p < 0.001), PT INR (OR: 3.05, p = 0.001), and diabetes/prediabetes (OR: 1.84, p = 0.001) were positively associated with fibrosis, while higher SMA (Area) (OR: 0.40, p = 0.010) and LDL (OR: 0.39, p = 0.043) were protective.

**Prognostic Value for Liver-Related Events**

We assessed the prognostic value of DeepCatch-derived metrics in predicting LREs, defined as a composite of ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome, or liver transplantation. Kaplan–Meier analysis revealed significant LRE-free survival differences stratified by body composition metrics (**Figure 4**). Both skeletal muscle area and attenuation were significantly associated with lower LRE risk in crude Cox regression models. The hazard ratio for skeletal muscle area was 0.24 (95% CI: 0.09–0.65, *p* = 0.0046), and for skeletal muscle attenuation, it was 0.37 (95% CI: 0.15–0.88, *p* = 0.0247), reinforcing their protective roles in clinical outcomes, as shown in **Supplementary Table 1**. In the adjusted model derived through backward stepwise selection, skeletal muscle area remained a significant independent predictor of lower LRE risk (HR: 0.23, 95% CI: 0.09–0.64, p = 0.0047).

**Discussion**

In this study, we evaluated the utility of CT-based body composition metrics derived from the DeepCatch software in characterizing the spectrum of MASLD—from steatosis to fibrosis—and in predicting long-term liver-related events. By automatically extracting adipose tissue, skeletal muscle, and organ-specific parameters from routine CT images, DeepCatch-derived metrics were shown to be valuable noninvasive biomarkers across multiple diagnostic and prognostic endpoints, including disease classification, steatosis severity, fibrosis staging, and liver-related event-free survival.

MASLD presents ongoing diagnostic and risk stratification challenges in clinical practice, particularly when distinguishing progressive forms such as MASH or identifying patients at risk for fibrosis-related complications. While transient elastography and MRI-based techniques such as CAP and PDFF are increasingly used, their limited accessibility and higher cost reduce their applicability in routine care. Moreover, traditional indices like BMI and FIB-4 may underperform in patients with altered fat distribution or sarcopenia. Our findings suggest that DeepCatch-derived metrics—particularly visceral fat area, liver-to-spleen attenuation ratio, skeletal muscle area, and the adjusted visceral fat index—may offer meaningful, scalable alternatives for MASLD phenotyping.

Among the endpoints assessed, DeepCatch metrics demonstrated the strongest diagnostic performance for steatosis severity. Liver-to-spleen HU achieved the highest AUC (0.815) for detecting moderate-to-severe steatosis, surpassing commonly used tools such as CAP, PDFF, and BMI. In contrast, while adjusted visceral fat index showed promise in fibrosis staging (AUC = 0.715), its performance was more modest than for steatosis. This discrepancy may be partly attributed to the inverse-U-shaped distribution of fat-based features across the MASLD spectrum. In cirrhosis, hepatic steatosis often regresses due to hepatocyte loss and fibrotic remodeling, which can obscure the signal captured by fat metrics and reduce predictive accuracy in advanced fibrosis, in contrast to fibrosis-specific markers such as LSM.

The prognostic analysis reinforced the relevance of body composition phenotypes in long-term outcomes. Higher skeletal muscle area and attenuation—reflecting preserved muscle mass and quality—were associated with lower risk of liver-related events. Similarly, lower adjusted visceral fat index values were also protective, underscoring the importance of maintaining muscle integrity and minimizing visceral adiposity as prognostic factors in MASLD.

Several limitations should be noted. The retrospective single-center design may limit the generalizability of the findings, and biopsy, while used as the diagnostic gold standard, is subject to inherent sampling variability. Additionally, our analysis was based on baseline imaging alone; the role of longitudinal changes in body composition over time warrants further study. External validation in prospective multicenter cohorts will also be necessary to confirm the clinical utility of DeepCatch metrics.

In conclusion, CT-derived body composition metrics obtained via DeepCatch offer robust, noninvasive tools for the diagnosis and monitoring of MASLD. While particularly strong in steatosis assessment, fat-based metrics showed more modest performance in fibrosis prediction, likely reflecting biological shifts in advanced disease. Nevertheless, their demonstrated prognostic value for liver-related events highlights their potential for integration into risk stratification models, advancing personalized management strategies in hepatology.

**References**

1. Younossi, Z.M., M. Kalligeros, and L. Henry, *Epidemiology of metabolic dysfunction-associated steatotic liver disease.* Clinical and Molecular Hepatology, 2025. **31**(Suppl): p. S32-S50.

2. Abdelhameed, F., et al., *Non-invasive Scores and Serum Biomarkers for Fatty Liver in the Era of Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD): A Comprehensive Review From NAFLD to MAFLD and MASLD.* Current Obesity Reports, 2024. **13**(3): p. 510-531.

3. Chowdhury, A.B. and K.J. Mehta, *Liver biopsy for assessment of chronic liver diseases: a synopsis.* Clinical and Experimental Medicine, 2023. **23**(2): p. 273-285.

4. Ballestri, S., et al., *Diagnostic accuracy of ultrasonography for the detection of hepatic steatosis: an updated meta-analysis of observational studies.* Metabolism and Target Organ Damage, 2021. **1**(1): p. N/A-N/A.

5. Zoncapè, M., A. Liguori, and E.A. Tsochatzis, *Non-invasive testing and risk-stratification in patients with MASLD.* European Journal of Internal Medicine, 2024. **122**: p. 11-19.

**Figure 1.** Study Cohort Flowchart

텍스트, 도표, 폰트, 라인이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

**Figure 2.** Boxplot Analysis of Traditional Metrics and Deepcatch-Derived Metrics Across MASLD Subtypes

(A) Liver Stiffness Measurement (LSM)

(B) Fibrosis-4 Index (FIB-4)

(C) Body Mass Index (BMI)

(D) Controlled Attenuation Parameter (CAP)

(E) Visceral Fat Area

(F) Liver/Spleen HU

텍스트, 스크린샷, 도표, 그래프이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.텍스트, 스크린샷, 도표, 직사각형이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

**도표, 텍스트, 스크린샷, 사각형이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.**도표, 직사각형, 스크린샷, 사각형이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

도표, 스크린샷, 텍스트, 사각형이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.도표, 텍스트, 스크린샷, 직사각형이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

**Figure 3.** ROC Curves Based on Logistic Regression Models for Assessing MASLD Type, Steatosis Severity, and Advanced Fibrosis

(A) Identifying MASLD

(B) Discriminating Moderate-to-Severe Steatosis (Score ≥ 2)

(C) Predicting Advanced Fibrosis (Stage ≥ 3)

텍스트, 라인, 도표, 스크린샷이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.텍스트, 라인, 도표, 그래프이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

텍스트, 라인, 도표, 그래프이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

**Figure 4.** DeepCatch-Derived Metrics for Predicting Liver-Related Event-Free Survival

(A) Kaplan–Meier Curve Stratified by Skeletal Muscle Area

(B) Kaplan–Meier Curve Stratified by Skeletal Muscle Attenuation

텍스트, 스크린샷, 도표, 라인이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.텍스트, 스크린샷, 도표, 라인이(가) 표시된 사진

AI가 생성한 콘텐츠는 부정확할 수 있습니다.

**Table 1.** Baseline Characteristics of Patients by Fibrosis Stage

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **Total Patients (n=229)** | **Advanced Fibrosis Patients a (n=56)** | **Non-Advanced Fibrosis Patients b (n=173)** | ***P*** |
| **Age, yrs** | 56.0 (45.0-67.0) | 64.0 (48.8-70.2) | 54.0 (44.0-65.0) | 0.002 |
| **Weight, kg** | 72.0 (63.2-81.7) | 67.2 (59.5-75.3) | 73.8 (64.3-82.4) | 0.004 |
| **Height, cm** | 165.0 (158.0-173.0) | 160.0 (153.7-167.2) | 167.0 (160.0-174.0) | 0.000 |
| **Waist to Height Ratio** | 5.5 (5.1-6.0) | 5.6 (5.3-6.0) | 5.4 (5.1-6.0) | 0.189 |
| **BMI, kg/m2** | 910.2 (841.9-973.4) | 910.8 (838.1-963.4) | 910.2 (844.9-983.8) | 0.630 |
| **VFI, cm3/m2** | 26.4 (24.0-29.5) | 26.3 (23.8-29.0) | 26.4 (24.0-29.5) | 0.589 |
| **SFI, cm3/m2** | 375.6 (261.2-535.8) | 354.6 (188.8-545.6) | 381.2 (264.7-530.7) | 0.193 |
| **TFI, cm3/m2** | 515.1 (371.5-753.1) | 500.3 (306.7-720.5) | 517.3 (384.9-754.0) | 0.390 |
| **SMI, cm3/m2** | 890.1 (687.7-1302.8) | 901.3 (622.4-1267.1) | 890.1 (703.1-1306.1) | 0.173 |
| **VFV, cm3** | 48.0 (42.7-56.0) | 45.7 (41.0-51.3) | 48.8 (43.0-56.8) | 0.049 |
| **SFV, cm3** | 1032.2 (683.4-1425.2) | 894.2 (502.0-1292.5) | 1064.1 (759.6-1474.6) | 0.029 |
| **Spleen Volume, cm3** | 1382.3 (1014.8-1922.2) | 1246.6 (828.6-1795.6) | 1448.7 (1056.7-1951.4) | 0.082 |
| **VFA (Area), cm2** | 199.3 (142.5-291.5) | 223.6 (142.0-318.9) | 194.4 (143.8-277.9) | 0.343 |
| **SFA (Area), cm2** | 149.4 (111.7-189.5) | 142.7 (106.3-175.2) | 151.5 (111.7-193.2) | 0.209 |
| **SMA (Area), cm2** | 179.9 (139.9-254.9) | 180.7 (139.5-259.8) | 177.2 (140.1-252.4) | 0.899 |
| **VFA (Attenuation), HU** | 131.0 (107.1-158.7) | 115.0 (101.5-133.0) | 139.5 (111.5-160.0) | 0.002 |
| **SFA (Attenuation), HU** | -96.8 (-103.6--90.2) | -93.9 (-102.8--84.9) | -97.7 (-103.7--92.1) | 0.022 |
| **SMA (Attenuation), HU** | -101.8 (-109.0--95.2) | -101.3 (-108.4--94.6) | -101.9 (-109.1--95.9) | 0.274 |
| **Liver/Spleen Volume** | 35.9 (29.1-42.4) | 34.2 (25.4-39.4) | 36.2 (30.4-43.4) | 0.020 |
| **Spleen Volume, cm3** | 7.7 (5.7-10.0) | 7.0 (4.1-9.2) | 7.8 (6.0-10.5) | 0.084 |
| **Liver/Spleen HU** | 0.9 (0.7-1.1) | 1.0 (0.8-1.1) | 0.9 (0.7-1.1) | 0.038 |
| **Liver HU, HU** | 46.5 (36.7-56.3) | 46.9 (41.5-55.2) | 45.1 (36.4-56.3) | 0.481 |
| **Spleen HU, HU** | 46.5 (42.7-52.2) | 44.9 (39.7-50.7) | 47.1 (43.1-52.5) | 0.083 |
| **Liver (PDFF), %** | 11.1 (5.8-16.7) | 10.1 (5.7-14.5) | 11.4 (6.2-16.9) | 0.460 |
| **LSM, kPa** | 7.7 (6.1-11.4) | 14.0 (9.9-17.1) | 7.5 (5.1-7.8) | 0.000 |
| **CAP, dB/m** | 280.0 (265.0-319.0) | 280.0 (236.8-298.5) | 280.0 (268.0-320.0) | 0.021 |
| **AST, IU/L** | 43.0 (27.0-77.0) | 54.5 (37.5-68.0) | 39.0 (26.0-79.0) | 0.066 |
| **ALT, IU/L** | 55.0 (28.0-95.0) | 45.0 (18.0-76.8) | 61.0 (32.0-101.0) | 0.034 |
| **T.bil, mg/dL** | 0.7 (0.5-1.1) | 0.8 (0.6-1.4) | 0.7 (0.5-1.1) | 0.009 |
| **PLT, x 103/mm3** | 227.0 (179.0-283.0) | 163.0 (115.0-241.2) | 238.0 (199.0-292.0) | 0.000 |
| **PT INR** | 1.0 (0.9-1.1) | 1.0 (1.0-1.1) | 1.0 (0.9-1.0) | 0.000 |
| **Albumin, g/dL** | 4.4 (3.8-4.7) | 4.2 (3.3-4.7) | 4.4 (3.9-4.7) | 0.042 |
| **Glucose, mg/dL** | 108.0 (95.0-138.0) | 113.0 (97.0-140.0) | 106.0 (95.0-136.0) | 0.405 |
| **HbA1c, %** | 6.4 (6.3-6.6) | 6.4 (6.0-6.9) | 6.4 (6.3-6.5) | 0.670 |
| **eGFR, mL/min** | 96.7 (84.4-118.7) | 95.7 (84.3-116.4) | 96.9 (85.0-118.7) | 0.716 |
| **T.chol, mg/dL** | 168.0 (136.0-202.0) | 156.5 (120.0-191.2) | 170.0 (142.0-202.0) | 0.018 |
| **HDL, mg/dL** | 44.0 (39.0-48.0) | 44.0 (34.5-47.2) | 44.0 (40.0-48.0) | 0.080 |
| **LDL, mg/dL** | 106.5 (106.5-106.5) | 106.5 (104.1-106.5) | 106.5 (106.5-113.0) | 0.002 |
| **TG, mg/dL** | 137.0 (112.0-181.0) | 132.0 (102.8-169.0) | 137.0 (121.0-183.0) | 0.044 |
| **SBP, mmHg** | 125.0 (116.0-134.0) | 127.0 (113.0-134.0) | 125.0 (116.0-134.0) | 0.908 |
| **DBP, mmHg** | 79.0 (70.0-87.0) | 77.5 (70.0-86.2) | 79.0 (70.0-87.0) | 0.600 |
| **FIB-4** | 1.4 (0.9-3.0) | 2.9 (1.5-5.4) | 1.2 (0.8-2.1) | 0.000 |
| **Sex** |  |  |  | 0.040 |
| Male | 107 (46.7) | 19 (33.9) | 88 (50.9) |  |
| Female | 122 (53.3) | 37 (66.1) | 85 (49.1) |  |
| **MASLD Type c** |  |  |  | 0.000 |
| None | 20 (8.7) | 0 (0.0) | 20 (11.6) |  |
| MASLD | 114 (49.8) | 0 (0.0) | 114 (65.9) |  |
| MASH | 70 (30.6) | 31 (44.6) | 39 (22.5) |  |
| Cirrhosis | 25 (10.9) | 25 (55.4) | 0 (0.0) |  |
| **Steatosis Score d** |  |  |  | 0.044 |
| 0 | 20 (8.7) | 0 (0.0) | 20 (11.6) |  |
| 1 | 127 (55.5) | 37 (66.1) | 90 (52.0) |  |
| 2 | 66 (28.8) | 15 (26.8) | 51 (29.5) |  |
| 3 | 16 (7.0) | 4 (7.1) | 12 (6.9) |  |
| **Smoking** |  |  |  | 0.743 |
| Yes | 38 (16.6) | 8 (14.3) | 30 (17.3) |  |
| No | 191 (83.4) | 48 (85.7) | 143 (82.7) |  |
| **Liver-Related Event e** |  |  |  | 0.000 |
| Yes | 25 (11.0) | 12 (21.4) | 13 (7.5) |  |
| No | 204 (89.1) | 44 (78.6) | 160 (92.5) |  |
| **Diabetes/Prediabetes Status** |  |  |  |  |
| Diabetes | 56 (24.5) | 19 (33.9) | 37 (21.4) |  |
| Prediabetes | 11 (4.8) | 4 (7.1) | 7 (4.0) |  |
| No | 162 (70.7) | 33 (58.9) | 129 (74.6) |  |
| **Hypertension Status** |  |  |  | 0.042 |
| Yes | 60 (26.2) | 21 (37.5) | 39 (22.5) |  |
| No | 169 (73.8) | 35 (62.5) | 134 (77.5) |  |
| **Dyslipidemia Status** |  |  |  | 0.007 |
| Yes | 57 (24.9) | 22 (39.3) | 35 (20.2) |  |
| No | 172 (75.1) | 34 (60.7) | 138 (79.8) |  |
| **Ischemic Heart Disease Status** |  |  |  | 0.048 |
| Yes | 14 (6.1) | 7 (12.5) | 7 (4.0) |  |
| No | 215 (93.9) | 49 (87.5) | 166 (96.0) |  |
| **Cerebrovascular Disease Status** |  |  |  | 0.428 |
| Yes | 10 (4.4) | 4 (7.1) | 6 (3.5) |  |
| No | 219 (95.6) | 52 (92.9) | 167 (96.5) |  |
| **Nephropathy Status** |  |  |  | 0.183 |
| Yes | 10 (4.4) | 5 (8.9) | 5 (2.9) |  |
| No | 219 (95.6) | 51 (91.1) | 168 (97.1) |  |

a Advanced fibrosis was defined as a histologic fibrosis stage of 3 or 4

b Non-advanced fibrosis was defined as a histologic fibrosis stage of 0 to 2

c MASLD includes both MASLD and probable MASH, while MASH refers to definitive steatohepatitis

d Steatosis Score was graded from 0 to 3 based on liver biopsy: 0: < 5% steatosis, 1: 5 – 33% (mild), 2: 34 – 66% (moderate), 3: > 66% (severe)

**e** Liver-Related Event was defined as the first occurrence of any of the following: ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome (HRS), or liver transplantation (LT), confirmed via clinical documentation or imaging.