**Non-invasive Assessment of MASLD Phenotypes Using CT-Derived Biomarkers: Diagnostic, Staging, and Prognostic Value**

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**Abstract**

**Background:** Metabolic dysfunction-associated steatotic liver disease (MASLD) encompasses a histological spectrum from benign steatosis to steatohepatitis with progressive fibrosis and liver-related events (LREs). Despite the increasing global burden, non-invasive tools to stratify MASLD severity and prognosis remain limited. We aimed to evaluate the utility of deep learning–derived computed tomography (CT) body composition metrics for noninvasive assessment of steatosis, fibrosis, and LRE-free survival within a histology-confirmed MASLD cohort.

**Methods:** We included 192 patients with biopsy-proven MASLD who underwent non-contrast abdominal CT and transient elastography. CT-derived body composition features were extracted using a deep learning-based segmentation software, including liver/spleen attenuation, muscle and fat metrics. Multivariable logistic regression with backward stepwise selection was used to identify independent predictors of steatosis (≥ S1, ≥ S2) and fibrosis (≥ F2, ≥ F3). Prognostic relevance was assessed using Cox proportional hazards models and Kaplan–Meier survival analysis. Scoring systems were derived from regression coefficients and evaluated using AUC and bootstrapped confidence intervals.

**Results:** Liver-to-spleen attenuation ratio was the most robust imaging predictor of steatosis, declining across increasing histologic steatosis scores and yielding high diagnostic performance (AUC = 0.847 for ≥ S2, 0.860 for ≥ S1). For fibrosis staging, skeletal muscle volume was the strongest protective factor, with models achieving AUCs of 0.840 (≥ F3) and 0.827 (≥ F2). Regression-based scoring systems demonstrated smooth, incremental gradients across histologic categories. In survival analysis, lower skeletal muscle area (HR: 0.33, 95% CI: 0.14-0.80, P = 0.014) and higher spleen attenuation, and lower visceral fat attenuation were independently associated with increased risk of LREs. Notably, skeletal muscle attenuation declined only in early fibrosis stages, highlighting the pathophysiological distinction between myosteatosis and sarcopenia.

**Conclusion:** Automated CT-derived body composition metrics provide accurate, noninvasive indicators of MASLD severity and prognosis. By integrating imaging and clinical features within a single histology-confirmed cohort, we developed interpretable scoring systems for steatosis, fibrosis, and LRE risk stratification. These findings support the clinical utility of CT-based precision tools for scalable MASLD management across the disease spectrum.

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**Introduction**

The global burden of metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is rapidly increasing in parallel with rising rates of obesity and type 2 diabetes, with adult prevalence projected to exceed 55% by 2040 [1, 2]. MASLD encompasses a histological spectrum, ranging from simple hepatic steatosi, defined as fat accumulation in more than 5% of hepatocytes, to metabolic dysfunction-associated steatohepatitis (MASH), characterized by hepatocellular ballooning, inflammation, and varying degrees of fibrosis [3-6]. Disease progression may culminate in cirrhosis and hepatocellular carcinoma, underscoring the urgent need for early identification and risk stratification [7].

Liver biopsy remains the gold standard for assessing steatosis and fibrosis; however, its invasiveness, risk of complications, poor patient acceptance, and sampling variability limit its utility, especially in routine practice [8-14]. Moreover, biopsy-derived data may not adequately reflect the heterogeneity of fat distribution or fibrotic remodeling in the liver [15]. Among non-invasive alternatives, ultrasound imaging is commonly used for initial screening but is hampered by operator dependency and limited accuracy, particularly in patients with visceral adiposity [16, 17]. Magnetic resonance proton density fat fraction (MRI-PDFF) is accepted as a non-invasive reference standard for liver fat quantification, but its clinical use is limited by high cost and restricted accessibility [18, 19].

Computed tomography (CT) is widely accessible, cost-effective, and reproducible for assessing hepatic and abdominal body composition [20]. Traditional ROI-based or semiautomated methods are time-consuming, subject to interobserver variability in subtle radiological changes, and prone to missing early-stage changes [21-23]. Recent advances in deep learning have enabled rapid, automated extraction of features such as visceral fat mass, skeletal muscle mass, and liver–spleen volume and attenuation, eliminating the need for manual annotation. Unlike black-box classification models, segmentation provides interpretable, trustworthy, morphology-based metrics that reflect known structural changes in liver disease [24-26]. Also, volumetric measurements are less affected by image quality and have demonstrated value in both staging fibrosis and predicting clinical outcomes in chronic liver disease [27-29].

This study aims to address key gaps by leveraging CT-derived body composition biomarkers to non-invasively evaluate multiple MASLD phenotypes - steatosis severity, fibrosis stage, and prognosis - within a unified framework. Using histology-confirmed cases, we integrate imaging and clinical data across the full MASLD spectrum, including patients without steatosis, and develop interpretable scoring systems to support precision risk stratification and clinical decision-making in the context of precision medicine.

**Methods**

This retrospective study was approved by the Institutional Review Board (IRB) of Severance Hospital, Seoul, South Korea (IRB No), with a waiver of informed consent. The study was conducted in accordance with the principles of the Declaration of Helsinki.

**Patient Selection**

Patients who underwent abdominal computed tomography (CT) between March 2006 and July 2023 were considered for inclusion. Eligibility criteria required the availability of corresponding liver biopsy, laboratory, and transient elastography data within three months of the CT scan. Patients were excluded if they had liver disease attributable to non-metabolic etiologies, including viral hepatitis, autoimmune hepatitis, drug-induced liver injury, or HIV infection. Additional exclusion criteria included prior liver resection, space-occupying hepatic lesions, acute infections, or other significant comorbidities [30].

**Liver Histology**

Liver biopsy specimens were evaluated to determine steatosis grade and fibrosis stage according to the NASH Clinical Research Network (CRN) histological scoring system [31]. Steatosis grade was assigned based on the histologic hepatic fat fraction, with S0 (none) indicating <5%, S1 (low) indicating 5–33%, S2 (moderate) indicating 34–66%, and S3 (severe) indicating >66%. Fibrosis stage was determined according to the Brunt criteria: F0 represented no fibrosis; F1, perisinusoidal or periportal fibrosis; F2, perisinusoidal and portal/p eriportal fibrosis; F3, bridging fibrosis; and F4, cirrhosis [32].

**Imaging and Clinical Parameters**

Non-contrast torso CT images were analyzed using a commercially available deep learning-based body composition analysis software (DeepCatch, v1.1.8.0, MEDICALIP Co., Ltd., Seoul, South Korea) [33-35]. Non-contrast CT scans were used to avoid contrast-induced variability in attenuation values, which are influenced by hepatic blood flow, injection rate, and scan timing which can impair accurate steatosis quantification [36, 37]. The software computed CT-derived body composition parameters, including total, visceral, and subcutaneous fat volumes (cm³); liver and spleen volumes (cm³); and their respective attenuation values in Hounsfield units (HU). Visceral fat attenuation and subcutaneous fat attenuation were measured at the abdominal waist level (between the 12th rib and iliac crest), while skeletal muscle area (cm²) and attenuation (HU) were assessed at the L3 vertebral level. [38, 39].

To assess hepatic steatosis, body mass index (BMI), controlled attenuation parameter (CAP), and waist-to-height ratio were evaluated. For fibrosis severity, liver stiffness measurement (LSM), the fibrosis-4 index (FIB-4), and the NAFLD fibrosis score (NFS) were calculated for comparison [40, 41].

**Endpoints**

The study evaluated three primary outcomes: steatosis severity, fibrosis severity, and event-free survival (EFS), defined as the time to the first liver-related event (LRE), including ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome, or liver transplantation [42-44]. Survival time was defined as the interval between the date of CT imaging and either the first liver-related event or the date of the last follow-up in the absence of an event.

Analyses were performed across multiple clinically relevant thresholds to reflect varying disease severities, including ≥ S2 (moderate-to-severe steatosis), ≥ S1 (low-to-severe steatosis), ≥ F3 (advanced fibrosis), and ≥ F2 (significant fibrosis).

**Model Training and Evaluation**

Variables found to be significant in univariable logistic regression were entered into multivariable models using backward stepwise selection. Model performance was assessed using the area under the receiver operating characteristic curve (AUC). The 95% confidence intervals (CIs) for the AUC were estimated through bootstrap resampling (1,000 iterations), using the 2.5th and 97.5th percentiles [45]. Sensitivity, specificity, positive predictive value and negative predictive value were calculated for the models. Youden index was used to determine the optimal cut-off value for these.

For each outcome, we developed a unique scoring system derived from the final logistic regression model. Based on prior methodology, each predictor was assigned a score proportional to its regression coefficient, enabling the development of a composite risk score specific to each outcome [46].

**Statistical Analyses**

All analyses were performed using Python (version 3.13.3). Data are presented as median (interquartile range [IQR]) or as counts with percentages (n [%]). Differences between groups were analyzed using the Mann-Whitney U test for continuous variables and the chi-square or Fisher’s exact test for categorical variables. Odds ratios (ORs) and hazard ratios (HRs) were calculated using logistic regression and Cox proportional hazards models, respectively. Kaplan–Meier method was utilized to generate survival curves for EFS, with group comparisons performed using the log-rank test.

**Results**

**Patients**

A total of 292 patients who underwent abdominal CT imaging and evaluation using the DeepCatch program were initially screened. Seven patients were excluded due to the absence of definitive liver biopsy results. Of the remaining 235 patients, 63 were excluded due to liver disease of non-metabolic etiology, including injury-related, chronic viral, or cholestatic hepatitis. 30 patients were additionally excluded due to absent ultrasonography or serum test results. Ultimately, 229 patients with complete body composition metrics, liver biopsy, imaging, and laboratory data were included in the final analysis , as shown in **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 61.8 months (IQR: 44.9–96.0 months). Baseline characteristics for the overall cohort and stratified comparisons between advanced and non-advanced fibrosis groups are summarized in **Table 1**. Comparisons between none-to-mild and moderate-to-severe steatosis groups are summarized in **Supplementary** **Table 1**.

**Distribution of Traditional and Body Composition Metrics by Steatosis Score and Fibrosis Stage**

For steatosis, liver-to-spleen attenuation ratio values significantly declined with each increasing steatosis score (P < 0.05), while liver volume and total fat index progressively increased (P < 0.05). CAP and waist-to-height ratio also showed significant differences across scores. However, BMI did not distinguish advanced steatosis stages, as illustrated in **Figure 2**. For fibrosis, LSM, FIB-4, and NFS increased steadily with higher fibrosis stages, confirming their relevance as fibrosis markers. Skeletal muscle volume consistently declined across stages, whereas skeletal muscle attenuation and area showed a significant drop only in the early stages, as shown in **Figure 3**.

**Prediction of Steatosis Severity**

For moderate-to-severe steatosis (score ≥ 2), multivariable logistic regression with backward stepwise selection identified five independent predictors: Liver-to-spleen attenuation ratio, total fat index, liver volume, PT INR, and sex. The final model achieved an AUC of 0.847 (95% CI: 0.786-0.901) with a sensitivity of 0.824 and specificity of 0.790, as shown in **Figure 4A**. Higher liver-to-spleen attenuation ratio (OR = 0.16, 95% CI: 0.07–0.35, P < 0.001), lower liver volume (OR: 2.84, 95% CI: 1.31-6.17, P = 0.009), and lower total fat index (OR: 3.01, 95% CI: 1.34-6.76, P = 0.008) was significantly protective as shown in **Supplementary Table 2**. The scoring system derived from our regression model demonstrated a smooth, incremental gradient that closely aligned with increasing steatosis scores, as shown in **Figure 4B**.

For mild-to-severe steatosis (score ≥ 1), the final model included three independent predictors: Liver-to-spleen attenuation ratio, waist-to-height ratio, PT INR. The final model achieved an AUC of 0.860 (95% CI: 0.797-0.920) with a sensitivity of 0.669 and specificity of 0.870, as shown in **Supplementary Figure 1A**. Higher liver-to-spleen attenuation ratio (OR = 0.08, 95% CI: 0.02–0.38, P = 0.001) was significantly protective, as described in **Supplementary Table 3**.

**Prediction of Fibrosis Severity**

For advanced fibrosis (stage ≥ 3), the final model retained five independent predictors: Sketeal muscle volume, waist-to-height ratio, AST ALT, and diabetes status. This model yielded strong discriminatory performance with an AUC of 0.840 (95% CI: 0.767-0.900) with a sensitivity of 0.812 and specificity of 0.736, as shown in **Figure 4C**. Higher sketeal muscle volume (OR: 0.13, 95% CI: 0.05–0.31, P < 0.001), absence of diabetes (OR: 2.56, 95% CI: 1.10-5.98, P = 0.030) was significantly protective as shown in **Supplementary Table 4**. The scoring system derived from our regression model demonstrated a smooth, incremental gradient that closely aligned with increasing fibrosis stages, as shown in **Figure 4D**.

For significant fibrosis (stage ≥ 2), the final model incldued six independent predictors: Skeletel muscle volume, waist-to-height ratio, AST, PLT, age, and diabetes status. The final model achieved an AUC of 0.827 (95% CI: 0.758-0.884) with a sensitivity of 0.771 and specificity of 0.746, as shown in **Supplementary Figure 1B**. Higher skeletal muscle volume (OR: 0.29, 95% CI: 0.13-0.62, P = 0.002) and absence of diabetes (OR: 3.39, 95% CI: 1.54-7.46, P = 0.002) were protective, as shown in **Supplementary Table 5**.

**Prognostic Value for LRE-free Survival**

Kaplan–Meier analysis revealed significant LRE-free survival differences stratified by high and low skeletal muscle area, as shown in **Supplementary** **Figure 2**. Higher skelete muscle area (HR: 0.33, 95% CI: 0.14-0.80, P = 0.014), higher spleen attenuation (OR: 0.38, 95% CI: 0.17-0.89, P = 0.025), and lower visceral fat attenuation (OR: 2.87, 95% CI: 1.19-6.91, P = 0.019) were significantly associated with lower LRE risk, as shown in **Supplementary Table 6**.

**Discussion**

In this study, we evaluated the utility of non-contrast CT-derived body composition metrics in diagnosing and prognosticating MASLD phenotypes, including steatosis severity, fibrosis stage, and EFS. Liver-to-spleen attenuation ratio emerged as the strongest imaging-based marker for steatosis, consistently declining across increasing histologic steatosis scores and achieving high diagnostic performance for both moderate-to-severe (AUC = 0.847) and mild-to-severe steatosis (AUC = 0.860). For fibrosis, skeletal muscle volume was the most protective factor, significantly declining across fibrosis stages and showing strongest discriminatory power for both advanced (AUC = 0.840) and significant fibrosis (AUC = 0.827). Additionally, lower skeletal muscle area was associated with higher risk of liver-related events, as demonstrated by survival analysis. Across both steatosis and fibrosis models, scoring systems derived from logistic regression coefficients demonstrated smooth, stepwise increases aligned with histologic severity, supporting their potential as interpretable, noninvasive risk stratification tools in MASLD.

Liver biopsy remains the diagnostic gold standard for assessing steatosis and fibrosis, but its invasiveness, cost, and reliance on sampling from the right hepaticl lobe restrict its routine use, particularly for screening or monitoring. Despite the availability of noninvasive alternatives, fewer than 5% of primary care physicians currently utilize them for MASLD risk stratification [47]. Ultrasound-based techniques, including transient elastography (FibroScan), are widely used but have limitations such as operator dependency, poor reproducibility in obese individuals, and reduced accuracy in grading steatosis or detecting precirrhotic fibrosis [48-50]. Serologic tools like FIB-4 and the NFS help identify ≥ F3 fibrosis but lack precision for detecting ≥ F2, which is critical for timely intervention [40, 41, 51]. MRI-based methods such as PDFF and MRE offer excellent accuracy but remain costly and less accessible for widespread clinical adoption.

Non-contrast CT offers a widely accessible, cost-effective, and reproducible method for hepatic steatosis assessment. CT attenuation values directly reflect hepatic fat content based on the difference in X-ray absorption between triglycerides and normal liver parenchyma [52]. Absolute liver attenuation and the liver–spleen attenuation index (LAI), are well-validated indicators, with reported AUCs of ~0.7 for detecting > 5% steatosis and ~0.9 for > 33% [53, 54]. While traditionally measured via region-of-interest (ROI) methods, volumetric CT attenuation measurements may offer a more comprehensive assessment, particularly in cases of heterogeneous steatosism, though data on their accuracy using robust histologic reference standards remain limited [55-57], Recently, deep learning–based automated segmentation has emerged as a scalable alternative, enabling rapid and reproducible extraction of volumetric metrics with strong concordance to ROI-based assessments [58-60].

Traditional indices such as BMI and waist-to-height ratio have been used to predict MASLD presence [61]. However, BMI does not capture fat distribution, while waist-to-height ratio cannot distinguish between visceral and subcutaneous fat, which limit their utility when evaluating histologic severity [62-65]. Among steatosis-related metrics, the liver-to-spleen attenuation ratio emerged as the most significant predictor, demonstrating the lowest regression coefficient in models predicting both ≥ S2 and S3 steatosis. This aligns with known principles of CT attenuation, where increased hepatic fat leads to reduced radiodensity due to lower X-ray absorption of triglyceride-rich tissues. Volumetric attenuation analysis, particularly when normalized to spleen density, provides a more comprehensive and reproducible estimate of steatosis burden than labor-extensive ROI methods, especially in cases of heterogeneous fat distribution [55-57].

Sarcopenia (low muscle mass, assessed by volume or area) and myosteatosis (poor muscle quality, measured by reduced attenuation) are independently linked to increased risks of fibrosis progression, cirrhosis, and liver-related events [66-69]. Transcriptomic analyses have linked sarcopenia to heightened inflammation and impaired muscle growth, while myosteatosis is associated with disrupted oxidative phosphorylation and lipid accumulation, which are key drivers of advanced MASLD [70, 71]. Sarcopenia increases MALSD risk fourfold, and its coexistence elevates the likelihood of advanced fibrosis by 1.8 times [72, 73]. Additionally, aging and diabetes further amplify fibrosis risk and remain well-established predictors of disease progression [74-79].

Our findings align with this evidence, as both lower muscle area, and higher age, diabetes prevalence were linked to higher fibrosis stage and poorer event-free survival. Skeletal muscle mass declined with advancing fibrosis, while attenuation decreased only in early stages, consistent with reports that myosteatosis precedes muscle loss in MASLD progression [80, 81]. This may reflect the limitations of mean attenuation, which cannot clearly differentiate fat and muscle compartments in advanced stages. Future studies should separately evaluate low- and normal-quality muscle, as only low-quality muscle has been shown to independently predict fibrosis progression over time [82].

This study has several limitations. Although conducted at a high-volume tertiary center, it may be subject to referral bias and limited generalizability. While liver biopsy served as the histologic reference, variability in interpretation and sampling may still lead to misclassification bias despite our efforts to use standardized scoring systems (NASH CRN). Additionally, emerging genomics and longitudinal data were not incorporated, and the retrospective design precludes causal inference. Future prospective, multicenter studies are warranted.

In conclusion, this study highlights the utility of automated, CT-derived body composition metrics as reliable, noninvasive biomarkers for the comprehensive assessment of MASLD. By simultaneously evaluating steatosis severity, fibrosis stage, and liver-related prognosis within a histology-confirmed cohort, we capture the full clinical spectrum of MASLD. Liver-to-spleen attenuation ratio emerged as the most robust imaging-based marker for steatosis, while skeletal muscle mass served as a key protective factor against fibrosis and adverse outcomes. Notably, our models incorporated both imaging and clinical variables—including patients without steatosis—to develop interpretable, regression-based scoring systems that closely align with histologic severity. These results underscore the potential of CT-based tools to support individualized, scalable risk stratification and enhance precision management of MASLD in everyday clinical settings.

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**Figure 1.** Study Cohort Flowchart

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**Figure 2.** Boxplot Analysis of Traditional Metrics and Deepcatch-Derived Metrics Across Steatosis Scores

(A) BMI

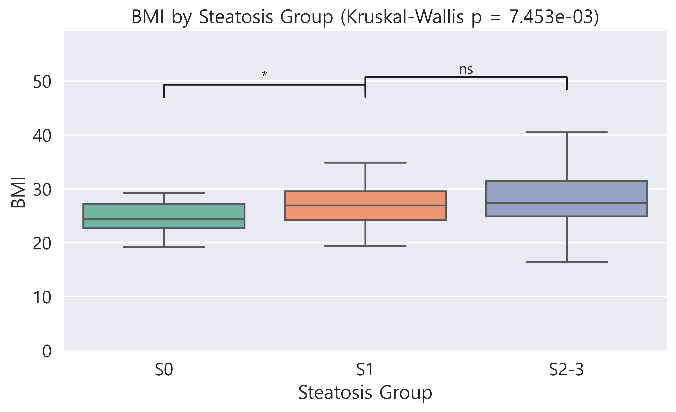
(B) TFI

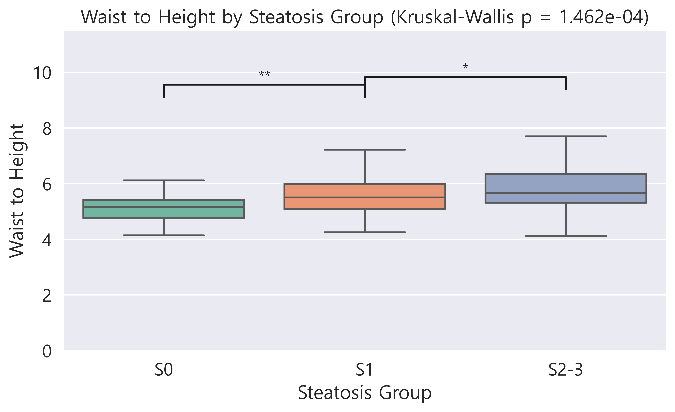
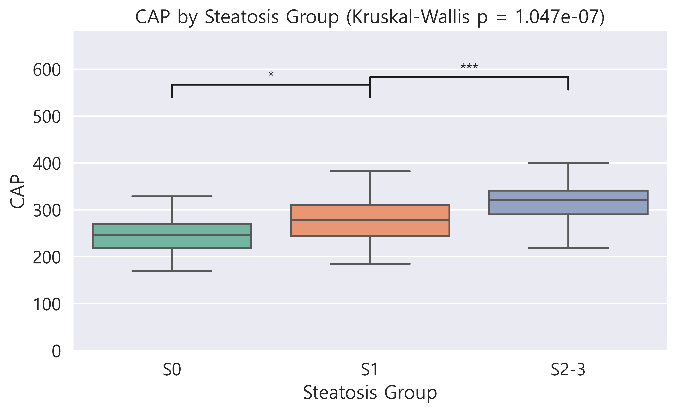
(C) CAP

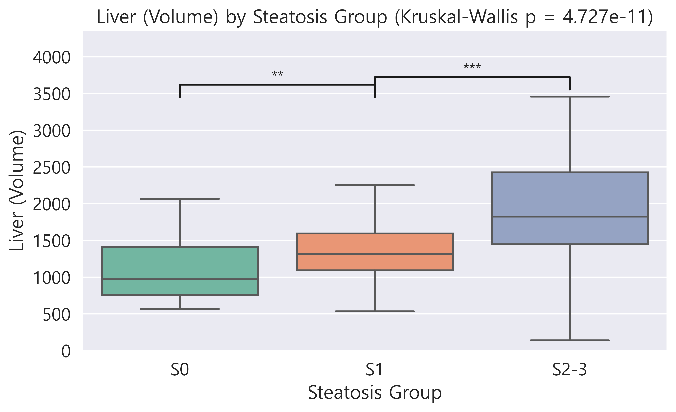
(D) Waist-to-Height Ratio

(E) Liver-to-Spleen Attenuation Ratio

(F) Liver Volume

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**Figure 3.** Boxplot Analysis of Traditional Metrics and Deepcatch-Derived Metrics Across Fibrosis Stages

(A) Skeletal Muscle Volume

(B) Skeletal Muscle Area

(C) Skeletal Muscle Attenuation

(D) LSM

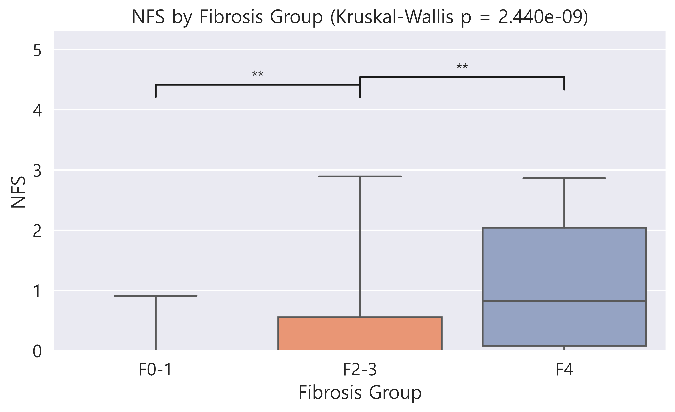
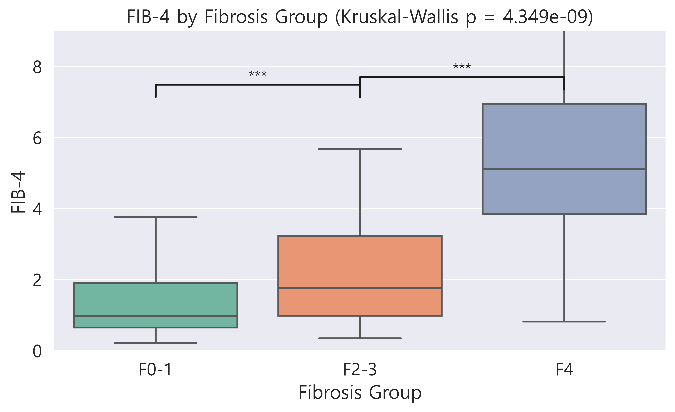
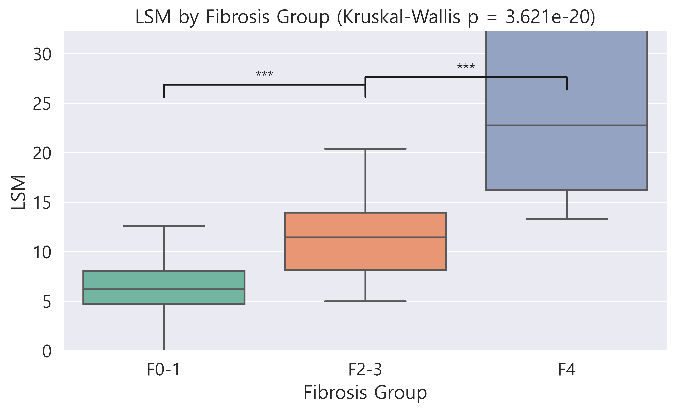
(E) FIB-4

(F) NFS

텍스트, 스크린샷, 도표, 직사각형이(가) 표시된 사진

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**Figure 4.** ROC Curves and Risk Scores Based on Logistic Regression Models for Assessing Steatosis Severity, and Fibrosis Stage

(A) ROC Curve: Discriminating Moderate-to-Severe Steatosis (Score ≥ 2)

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

(C) ROC Curve: Predicting Advanced Fibrosis (Stage ≥ 3)

(D) Risk Score: Predicting Advanced Fibrosis (Stage ≥ 3)

텍스트, 스크린샷, 라인, 도표이(가) 표시된 사진

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

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **Total Patients (n=192)** | **Advanced Fibrosis Patients a (n=48)** | **Non-Advanced Fibrosis Patients b (n=144)** | ***P*** |
| **Age, yrs** | 49.5 (38.8-62.0) | 61.0 (44.8-67.0) | 47.0 (36.0-58.0) | 0.000 |
| **Weight, kg** | 72.0 (64.0-82.0) | 67.9 (59.5-75.3) | 73.0 (64.9-84.8) | 0.006 |
| **Height, cm** | 165.0 (157.9-173.0) | 159.5 (153.7-166.2) | 166.8 (158.8-174.0) | 0.000 |
| **Waist to Height Ratio** | 5.5 (5.2-6.0) | 5.7 (5.3-6.1) | 5.5 (5.1-6.0) | 0.303 |
| **BMI, kg/m2** | 910.9 (841.1-983.8) | 910.8 (835.8-960.8) | 910.9 (847.7-999.6) | 0.482 |
| **VFI, cm3/m2** | 26.8 (24.2-29.8) | 26.5 (24.4-29.0) | 26.9 (24.2-29.8) | 0.652 |
| **SFI, cm3/m2** | 381.8 (250.8-543.0) | 359.0 (212.4-545.6) | 399.5 (263.5-538.8) | 0.316 |
| **TFI, cm3/m2** | 541.0 (409.7-777.5) | 575.4 (332.6-720.5) | 535.4 (414.4-806.1) | 0.458 |
| **SMI, cm3/m2** | 943.5 (689.2-1325.2) | 973.6 (633.3-1267.1) | 935.0 (702.0-1415.8) | 0.244 |
| **VFV, cm3** | 365.8 (298.7-442.9) | 299.0 (266.9-370.2) | 385.6 (316.1-446.0) | 0.000 |
| **SFV, cm3** | 1031.1 (682.0-1433.4) | 908.8 (560.1-1270.0) | 1064.5 (746.4-1486.5) | 0.067 |
| **Spleen Volume, cm3** | 1498.3 (1096.9-2010.0) | 1327.9 (852.2-1795.6) | 1507.2 (1111.2-2111.5) | 0.096 |
| **VFA (Area), cm2** | 195.8 (143.5-295.0) | 210.7 (142.0-313.3) | 193.2 (145.4-290.4) | 0.620 |
| **SFA (Area), cm2** | 147.9 (107.5-189.1) | 139.0 (106.3-180.0) | 148.9 (108.2-191.3) | 0.450 |
| **SMA (Area), cm2** | 184.0 (143.5-261.8) | 188.3 (142.0-261.8) | 182.3 (145.6-260.4) | 0.970 |
| **VFA (Attenuation), HU** | 130.7 (107.1-158.7) | 115.0 (101.5-135.3) | 137.8 (111.0-162.7) | 0.004 |
| **SFA (Attenuation), HU** | -97.0 (-103.8--90.0) | -95.7 (-103.5--86.0) | -98.1 (-103.9--91.8) | 0.140 |
| **SMA (Attenuation), HU** | -102.5 (-109.2--95.9) | -102.9 (-108.7--96.5) | -102.5 (-109.3--95.9) | 0.680 |
| **Liver-to-Spleen Volume** | 35.9 (29.1-41.9) | 35.2 (25.5-40.4) | 35.9 (29.8-43.3) | 0.119 |
| **Spleen Volume, cm3** | 7.6 (5.4-10.2) | 7.2 (4.2-9.3) | 7.7 (5.8-10.6) | 0.246 |
| **Liver-to-Spleen Attenuation** | 0.9 (0.7-1.1) | 1.0 (0.8-1.1) | 0.9 (0.7-1.1) | 0.054 |
| **Liver Attenuation, HU** | 46.3 (36.4-56.4) | 47.7 (42.6-56.5) | 45.1 (33.8-56.0) | 0.217 |
| **Spleen Attenuation, HU** | 46.8 (43.2-52.2) | 46.5 (42.6-51.2) | 47.0 (43.2-52.3) | 0.580 |
| **Liver (PDFF), %** | 11.2 (5.6-16.8) | 9.3 (5.3-13.7) | 11.6 (6.2-18.3) | 0.196 |
| **LSM, kPa** | 7.6 (5.2-12.2) | 14.7 (11.9-22.2) | 6.8 (4.8-8.6) | 0.000 |
| **CAP, dB/m** | 288.0 (253.2-327.0) | 273.0 (229.0-319.2) | 294.5 (256.0-330.5) | 0.071 |
| **AST, IU/L** | 47.0 (30.0-83.2) | 56.0 (42.2-80.0) | 43.0 (27.8-83.2) | 0.030 |
| **ALT, IU/L** | 57.5 (29.5-99.5) | 48.5 (18.8-80.8) | 61.0 (32.0-115.0) | 0.129 |
| **T.bil, mg/dL** | 0.7 (0.5-1.1) | 0.8 (0.6-1.3) | 0.7 (0.5-1.0) | 0.029 |
| **PLT, x 103/mm3** | 227.5 (176.8-287.2) | 170.0 (116.5-239.2) | 241.0 (199.0-301.2) | 0.000 |
| **PT INR** | 1.0 (0.9-1.1) | 1.0 (1.0-1.1) | 1.0 (0.9-1.0) | 0.005 |
| **Albumin, g/dL** | 4.4 (3.8-4.7) | 4.2 (3.7-4.7) | 4.4 (3.8-4.7) | 0.304 |
| **Glucose, mg/dL** | 106.0 (95.0-133.2) | 109.5 (96.5-137.8) | 104.5 (94.8-132.2) | 0.557 |
| **HbA1c, %** | 4.4 (4.4-6.0) | 4.4 (4.4-6.5) | 4.4 (4.4-5.9) | 0.455 |
| **eGFR, mL/min** | 98.4 (84.9-122.5) | 95.0 (83.9-116.4) | 101.8 (86.5-123.5) | 0.271 |
| **T.chol, mg/dL** | 169.0 (133.8-202.0) | 163.0 (126.8-192.2) | 171.5 (137.0-202.5) | 0.089 |
| **HDL, mg/dL** | 38.0 (9.0-47.0) | 29.5 (9.0-46.0) | 39.0 (9.0-47.0) | 0.288 |
| **LDL, mg/dL** | 24.0 (24.0-78.0) | 24.0 (24.0-24.0) | 24.0 (24.0-104.5) | 0.000 |
| **TG, mg/dL** | 113.5 (37.0-165.2) | 108.0 (42.2-140.2) | 116.0 (37.0-174.5) | 0.408 |
| **SBP, mmHg** | 126.0 (116.0-134.0) | 128.0 (116.2-134.0) | 124.7 (116.0-134.0) | 0.481 |
| **DBP, mmHg** | 79.0 (71.8-87.0) | 80.5 (72.8-89.0) | 79.0 (70.8-86.2) | 0.642 |
| **FIB-4** | 1.3 (0.7-3.0) | 3.4 (1.5-5.4) | 1.0 (0.7-2.1) | 0.000 |
| **Sex** |  |  |  | 0.029 |
| Male | 84 (43.8) | 14 (29.2) | 70 (48.6) |  |
| Female | 108 (56.2) | 34 (70.8) | 74 (51.4) |  |
| **MASLD Type c** |  |  |  | 0.000 |
| None | 21 (10.9) | 0 (0.0) | 21 (14.6) |  |
| MASLD | 89 (46.3) | 0 (0.0) | 89 (61.8) |  |
| MASH | 60 (31.2) | 26 (54.2) | 34 (23.6) |  |
| Cirrhosis | 22 (11.5) | 22 (45.8) | 0 (0.0) |  |
| **Steatosis Score d** |  |  |  | 0.003 |
| 0 | 23 (12.0) | 1 (2.1) | 22 (15.3) |  |
| 1 | 101 (52.6) | 35 (72.9) | 66 (45.8) |  |
| 2 | 52 (27.1) | 7 (14.6) | 45 (31.2) |  |
| 3 | 16 (8.3) | 5 (10.4) | 11 (7.6) |  |
| **Smoking** |  |  |  | 0.823 |
| Yes | 32 (16.7) | 7 (14.6) | 25 (17.4) |  |
| No | 160 (83.3) | 41 (85.4) | 119 (82.6) |  |
| **Liver-Related Event e** |  |  |  | 0.023 |
| Yes | 27 (14.1) | 12 (25.0) | 15 (10.4) |  |
| No | 165 (85.9) | 36 (75.0) | 129 (89.6) |  |
| **Diabetes/Prediabetes Status** |  |  |  | 0.041 |
| Diabetes | 43 (22.4) | 16 (33.3) | 27 (18.8) |  |
| Prediabetes | 10 (5.2) | 4 (8.3) | 6 (4.2) |  |
| No | 139 (72.4) | 28 (58.3) | 111 (77.1) |  |
| **Hypertension Status** |  |  |  | 0.045 |
| Yes | 49 (25.5) | 18 (37.5) | 31 (21.5) |  |
| No | 143 (74.5) | 30 (62.5) | 113 (78.5) |  |
| **Dyslipidemia Status** |  |  |  | 0.001 |
| Yes | 48 (25.0) | 21 (43.8) | 27 (18.8) |  |
| No | 144 (75.0) | 27 (56.2) | 117 (81.2) |  |
| **Ischemic Heart Disease Status** |  |  |  | 0.024 |
| Yes | 10 (5.2) | 6 (12.5) | 4 (2.8) |  |
| No | 182 (94.8) | 42 (87.5) | 140 (97.2) |  |
| **Cerebrovascular Disease Status** |  |  |  | 0.211 |
| Yes | 8 (4.2) | 4 (8.3) | 4 (2.8) |  |
| No | 184 (95.8) | 44 (91.7) | 140 (97.2) |  |
| **Nephropathy Status** |  |  |  | 0.134 |
| Yes | 10 (5.2) | 5 (10.4) | 5 (3.5) |  |
| No | 182 (94.8) | 43 (89.6) | 139 (96.5) |  |

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.