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Original Article

The association between appendicular skeletal muscle index and liver steatosis and fibrosis: A cross-sectional study

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Abstract

Background: Skeletal muscle index (SMI) is a measure for evaluating skeletal muscle status. However, its specific association with liver steatosis and fibrosis remains unclear. The aim of this study was to investigate the association between SMI and liver steatosis and fibrosis.

Methods: We conducted a cross-sectional study with a random selection of 328 participants over the age of 18, with no history of alcohol consumption or liver disease, from a nutrition clinic in Iran. Waist circumference (WC) and hip circumference (HC) were measured using a tape measure according to standards. Total fat mass, SMI, and other body composition parameters were obtained via bioelectrical impedance analysis. Liver status was assessed in all participants using elastography (FibroScan®).

Results: The participants included 64.0% males and 36.0% females, with a median age of 41 (IQR:14) years. After adjusting for confounders, SMI had no significant association with liver steatosis (P=0.647). Indeed, body mass index (BMI) (P=0.028), WC (P=0.038), and HC (P=0.007) were the significant predictors of liver steatosis. Conversely, each unit increase in SMI value decreased the chance of liver fibrosis by 48% after controlling the confounders (aOR=0.519, 95%CI: 0.283-0.951, P=0.034). Additionally, BMI (P=001), WC (P=0.006), and HC (P=0.026) were other significant predictors of liver fibrosis.

Conclusion: In conclusion, while a higher SMI did not mitigate obesity-linked liver steatosis risk, it independently lowered the odds of fibrosis. Furthermore, increased waist circumference was a stronger predictor of both steatosis and fibrosis than increased trunk fat mass.

Keywords: Metabolic dysfunction-associated fatty liver disease, MAFLD, NAFLD, Metabolic dysfunction-associated steatotic liver disease, MASLD, Sarcopenia.

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Metabolic dysfunction-associated fatty liver disease (MAFLD) is a broad term for liver fat accumulation linked to obesity, diabetes, or at least two metabolic issues. These issues can include an enlarged waist, high blood pressure, prediabetes, high triglycerides, and low levels of high-density lipoprotein cholesterol (HDL-C) (1). MAFLD has become increasingly prevalent over the past three decades, affecting approximately one-third of adults (2). This rise is largely attributed to a combination of physical inactivity and unhealthy dietary habits (3, 4). The prevalence is even higher in obese adults, reaching approximately 60% (5). Liver fibrosis is the essential pathologic feature that predict liver related mortality and complications, such as liver failure, liver transplantation, and hepatocellular carcinoma (6). Direct fat storage during feeding, de novo lipogenesis, and free fatty acid release from visceral adipose tissue during fasting are key mechanisms of liver steatosis (7). A high-calorie diet and obesity, especially visceral obesity, are the main risk factors for the development and progression of MAFLD (8-11).

In contrast, physical activity and aerobic exercise guarantee metabolic health status and attenuation of liver steatosis, which is associated with higher relative appendicular skeletal muscle mass (12-14). Body composition refers to the proportional contributions of fat, muscle, bone, and water within an individual's body volume, and bioelectric impedance analysis (BIA) stands out as a rapid, noninvasive, and cost-effective technique suitable for bedside applications. BIA operates on the principles of impedance, which encompasses resistance indicative of the opposition to electrical current flow and reactance, which reflects the opposition to changes in current due to material capacitance. As an electrical current traverses the body, varying resistance levels are encountered: tissues with high water content, such as muscle, exhibit lower resistance due to their electrolyte-rich composition, while anhydrous tissues, such as fat, present higher resistance. Additionally, reactance correlates with cell density and membrane integrity, further characterizing the body's composition (15).

Skeletal muscle percentage is a muscle index that has been used frequently for investigating the role of skeletal muscle mass on severity of MAFLD. Numerous studies have shown that a lower skeletal muscle percentage is associated with a higher risk of liver steatosis and fibrosis (16-20). Despite frequent application of this muscle index in literature, a significant concern arises regarding its independence as a distinct feature. Muscle mass and fat mass are two major components of body weight; when one increases, it often leads to a decrease in the proportion of the other. For instance, an obese individual with a notably higher skeletal mass may actually exhibit a much lower percentage of skeletal muscle due to an excess of fat mass. This index is heavily influenced by total fat mass, which complicates the assessment of the independent impact of muscle mass on conditions such as liver steatosis and fibrosis. Few studies have adjusted skeletal muscle mass or area for height in examining the relationship between skeletal muscle mass and MAFLD to address the impact of fat mass on skeletal muscle percentage. A recent study by *Jin, et al.* (21) found that an increase of one unit in the skeletal muscle index (SMI), which is defined as the division of skeletal muscle mass by the height squared, escalated the risk of MAFLD by 70%. Also, the skeletal muscle area index at the L3 level showed a positive correlation with steatosis severity (22). These findings challenge earlier research suggesting that skeletal muscle status has a protective effect against MAFLD. This discrepancy arises from the limitations of surrogate indexes used to compare skeletal muscle status across individuals. Therefore, we designed this study to explore the relationship between SMI, liver steatosis, and liver fibrosis, aiming to clarify the conflicting findings of recent research.

Methods

Design and participants: In the current study, we selected participants from a nutrition clinic in the south of Iran (Bandar Abbas City) in 2023. Based on a previous study (23) with a reported MAFLD prevalence of P=0.33, and given α=0.05 ($Z\_(\alpha\ /\ 2) = 1.96$), β=0.2 ($Z_{1-\beta} = 0.84$), and d=0.05, the required sample size was calculated as 340. We therefore enrolled 342 participants. Of the 342 individuals, over the age of 18 in the study, 14 individuals were excluded due to alcohol consumption more than twice a week, a history of viral hepatitis or other chronic liver diseases, administration of corticosteroids, steroids, contraceptives, antiepileptics, chemotherapy regimens, pregnancy, and recent cancer. This study has been approved by the Ethics Committee of Hormozgan University of Medical Sciences (IR.HUMS.REC.1403.105) and has been conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Data collection: An internalist performed a thorough history taking and physical examination. An expert nutritionist measured the height, waist circumference (WC), and hip circumference (HC) according to standards (11). All the participants were gone under bioelectrical impedance analysis (Inbody270, south Korea) to measure weight, total fat mass, total fat percent, trunk fat mass, fat-free mass, bone mineral content, ASM, and SMI. SMI was defined as division of ASM by height squared. Skeletal muscle percentage is the percentage of body weight that is occupied by ASM. Controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) were measured by FibroScan® (Echosens 504, Paris, France). M or XL probes was selected based on the distance of skin to liver capsule. A reliable LSM was defined as the median liver stiffness of ten measurements (24). We considered a CAP score of 238 and above as liver steatosis (25). Additionally, we considered an LSM of 7.6 and higher as significant fibrosis (26).

Statistical analysis: All statistical analyses were performed using IBM SPSS Version 26.0. The normality of the data was tested using the Kolmogorov–Smirnov test. We reported the categorical variables as numbers (n) and percentages (%), and the continuous variables as medians and interquartile ranges (IQR). We compared the variables between the two groups using the Mann-Whitney U test.

The Spearman correlation test was performed to measure the correlation between variables. The Kruskal-Wallis test was applied to assess the impact of SMI quartiles on the severity of liver steatosis and liver fibrosis. Univariate (crude analysis) and multiple logistic regression analyses (adjusted analysis) were performed to evaluate the association of variables with liver steatosis and significant fibrosis. The goodness of fit for the model was assessed using the Hosmer-Lemeshow test, where a p-value greater than 0.05 indicates a satisfactory fit of the model to the observed data.

Results

Descriptive data: We enrolled 328 individuals in this study, including 210 (64.0%) males and 118 (36.0%) females, with a median age of 41 (14.0) years. The number of individuals involved with liver steatosis and significant liver fibrosis were 245 (74.7%), and 73 (22.3%), respectively. The number of overweight and obese participants were 142 (43.3%), and 133 (40.5%), respectively. The analysis also revealed that weight, BMI, WC, HC, WHR, total fat mass, and trunk fat mass were higher in individuals with liver steatosis or significant liver fibrosis than individuals without these conditions, for more details, refer to table 1.

SMI and liver steatosis: The comparison analysis showed that SMI was higher in individuals with liver steatosis (table 1), and it had positive correlation with CAP score (rho=0.170, P=0.002) (figure 1). Furthermore, the CAP score (P=0.001) gradually increased by going toward higher quartiles of SMI (figure 2).

Although SMI had a significant association with liver steatosis in univariate analysis (cOR=1.357, 95%CI: 1.075-1.713, P=0.010), BMI, WC and HC were the independent predictors of liver steatosis in multiple logistic regression (BMI: aOR=1.345, 95%CI: 1.033-1.752, P=0.028, WC: aOR=1.068, 95%CI: 1.004-1.137, P=0.038, and HC: aOR=0.893 95%CI: 0.823-0.969, P=0.007) (table 2).

SMI and liver fibrosis: The comparison analysis showed that SMI was higher in individuals with significant fibrosis compared to individuals without these conditions (table 1). Moreover, SMI had positive correlation with LSM (rho=0.240, P<0.001) (figure 1). Furthermore, the LSM (p<0.001) gradually increased by going toward higher quartiles of SMI (figure 2).

SMI had also positive association with significant fibrosis in univariate analysis (cOR=1.419, 95%CI: 1.111-1.811, P=0.005), but the association changed to negative nature in multiple logistic regression. SMI, BMI, WC, and HC were the independent predictors of significant fibrosis in multiple logistic regression (SMI: aOR=0.519, 95%CI: 0.283-0.951, P=0.034; BMI: aOR =1.568, 95%CI: 1.202-2.045, P=0.001, WC: aOR =1.095, 95%CI: 1.026-1.168, P=0.006, HC: aOR=0.923, 95%CI=0.859-0.990, P=0.026) (table 2).

Table 1. Comparisons. Differences of body composition parameters between liver steatosis or significant fibrosis subjects and individuals without the condition

Variable	Steatosis			fibrosis Significant		
	No n (%)	Yes n (%)	P-value	No n (%)	Yes n (%)	P-value
Age	39 (13.0)	41 (15.0)	0.082	40.0 (15.0)	42.0 (16.0)	0.67
Gender			0.586			0.934
Male	52 (66.7%)	155 (63.3%)		159 (63.9%)	47 (64.4%)	
Female	26 (33.3%)	90 (36.7%)		90 (36.1%)	26 (35.6%)	
Weight	76.8 (21.3)	85. 3 (18.1)	<0.001	81.4 (17.5)	93.1 (21.7)	<0.001
Height	171.0 (14.0)	170.0 (16.0)	0.213	170.0 (14.5)	169 (20.0)	0.436
BMI	26.1 (5.2)	29.8 (5.8)	<0.001	28.3 (5.0)	33.3 (6.5)	<0.001
WC	91.0 (16.0)	100.0 (13.5)	<0.001	96.0 (13)	106.0 (16.0)	<0.001
HC	105.0 (11.0)	108.0 (11.0)	0.001	106.0 (9.0)	111.0 (12.0)	<0.001
WHR	0.92 (0.09)	0.96 (0.1)	<0.001	0.94 (0.09)	0.99 (0.09)	<0.001
TFM	21.7 (8.6)	29.3 (11.6)	<0.001	25.8 (10.9)	34.6 (16.0)	<0.001

Variable	Steatosis			fibrosis Significant		
	No n (%)	Yes n (%)	P-value	No n (%)	Yes n (%)	P-value
TFP	29.1 (12.6)	35.0 (12.9)	<0.001	32.5 (12.1)	36.3 (13.0)	<0.001
TrFM	11.5 (4.7)	15.4 (5.6)	<0.001	13.7 (5.6)	18.2 (6.8)	<0.001
FFM	54.8 (15.9)	56.2 (17.5)	0.246	55.0 (16.2)	57.9 (20.3)	0.048
BMC	3.75 (1.04)	3.80 (1.1)	0.371	3.76 (1.0)	3.89 (1.3)	0.140
ASM	23.1 (7.2)	24.1 (7.7)	0.354	23.6 (7.4)	24.5 (8.4)	0.202
SMI	8.0 (1.3)	8.3 (1.5)	0.007	8.1 (1.5)	8.6 (1.7)	0.004
SMP	29.5 (6.0)	27.7 (6.2)	<0.001	28.6 (6.4)	26.5 (5.7)	<0.001
CAP	205.0 (32.0)	293.0 (54.0)	<0.001	268.0 (72.5)	304.0 (55.0)	<0.001
LSM	4.6 (1.2)	6.0 (2.8)	<0.001	5.1 (1.6)	10.1 (3.5)	<0.001

Abbreviations: BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio: TFM, total fat mass; TFP, total fat percent; TrFM, trunk fat mass; FFM, free fat mass; BMC, bone mineral content; ASM, appendicular skeletal muscle mass; SMI, skeletal muscle index; SMP, skeletal muscle percent; CAP, controlled attenuated parameter; LSM, liver stiffness measurement.

Table 2. Logistic regression. The association of skeletal muscle index, anthropometric and body composition parameters with liver steatosis and significant liver fibrosis

Independent Variables	Dependent Variables	Univariate			Multiple		
		OR	95%CI	P-value	OR	95%CI	P-value
Age	**Steatosis**	1.021	0.997-1.046	0.089	0.993	0.962-1.024	0.650
Gender		1.161	0.678-1.988	0.586	1.241	0.426-3.613	0.693
BMI		1.235	1.148-1.328	<0.001	1.345	1.033-1.752	0.028
TFM		1.092	1.056-1.130	<0.001	0.881	0.705-1.100	0.264
TrFM		1.207	1.130-1.290	<0.001	1.276	0.865-1.883	0.219
WC		1.092	1.060-1.125	<0.001	1.068	1.004-1.137	0.038
HC		1.051	1.019-1.083	0.002	0.893	0.823-0.969	0.007
SMI		1.357	1.075-1.713	0.010	0.870	0.479-1.579	0.647
Age	**Fibrosis**	1.025	1.001-1.050	0.042	1.001	0.970-1.034	0.936
Gender		0.977	0.567-1.684	0.934	0.406	0.136-1.212	0.106
BMI		1.248	1.169-1.332	<0.001	1.568	1.202-2.045	0.001
TFM		1.091	1.061-1.122	<0.001	0.880	0.733-1.057	0.172
TrFM		1.221	1.147-1.301	<0.001	1.096	0.802-1.500	0.565
WC		1.108	1.075-1.143	<0.001	1.095	1.026-1.168	0.006
HC		1.063	1.032-1.095	<0.001	0.923	0.859-0.990	0.026
SMI		1.419	1.111-1.811	0.005	0.519	0.283-0.951	0.034

According Hosmer-Lemeshow test, there is no difference between the observed and model-predicted values in both steatosis (P=0.871) and fibrosis (P=0.134), implying that the model's estimates fit the data at an acceptable level. Abbreviations: BMI, body mass index; WC, waist circumference; HC, hip circumference; TFM, total fat mass; TrFM, trunk fat mass; SMI, skeletal muscle index.

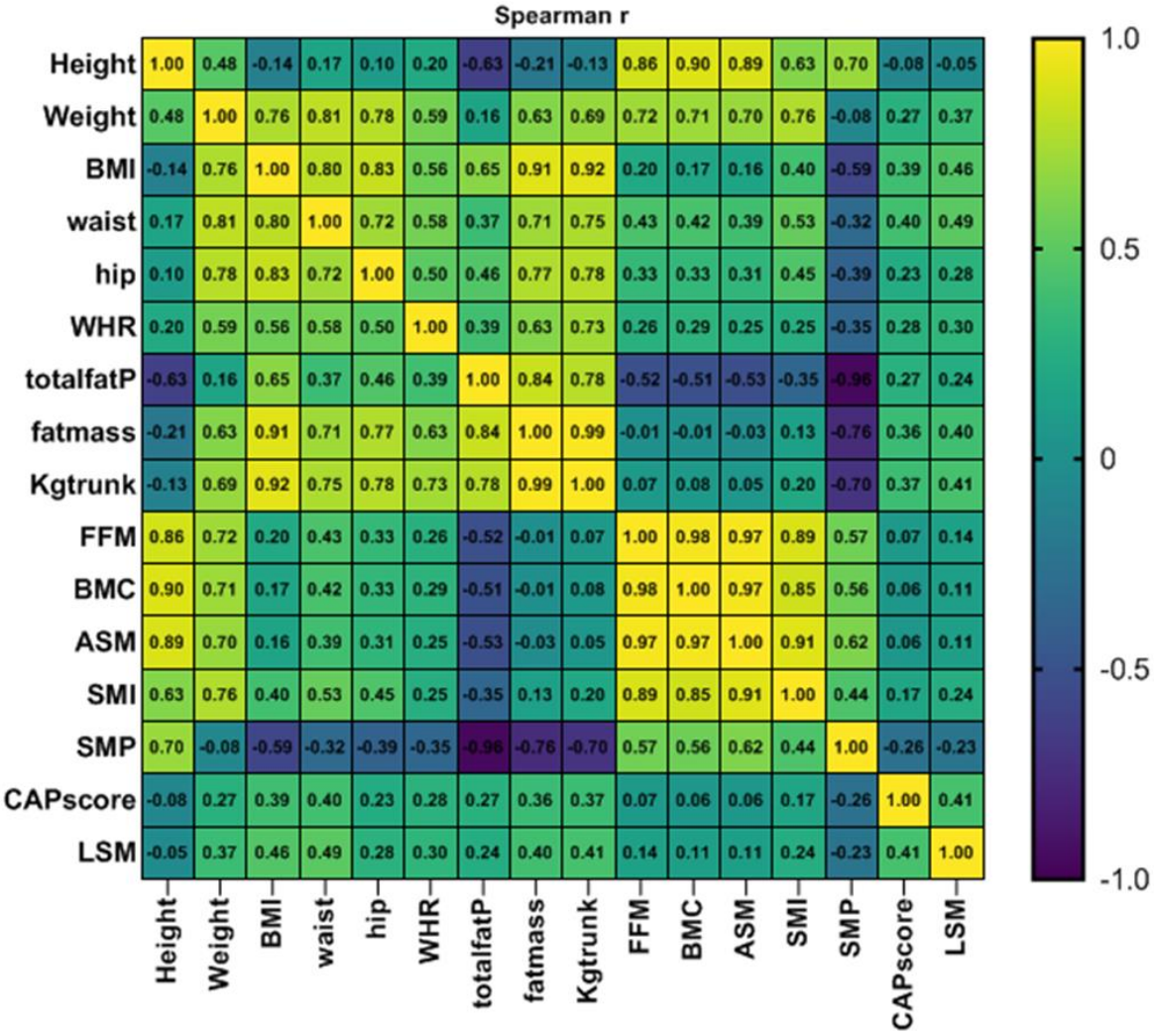

Figure 1. The correlation matrix.

Abbreviations: BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-to-hip ratio: TFM, total fat mass; TFP, total fat percent; TrFM, trunk fat mass; FFM, free fat mass; BMC, bone mineral content; ASM, appendicular skeletal muscle mass; SMI, skeletal muscle index; SMP, skeletal muscle percent; CAP, controlled attenuated parameter; LSM, liver stiffness measurement.

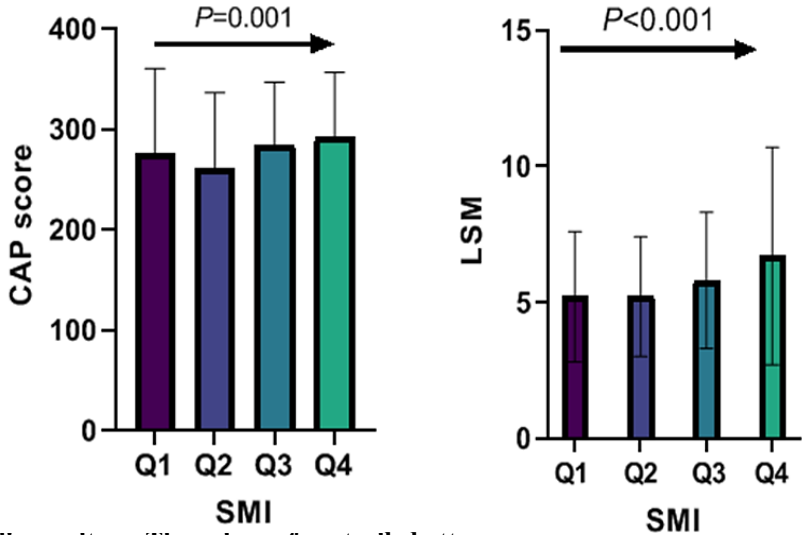

Figure 2. Kruskal-Wallis results. a. The values of controlled attenuation parameter gradually increase by going toward higher quartiles of skeletal muscle index (SMI). b. The values of liver stiffness measurements gradually increase by going toward higher quartiles of SMI.

Discussion

To clarify the conflicting findings of recent research with previous studies (20-22), this study aimed to investigate the association between SMI and liver steatosis and fibrosis. The challenging finding was the positive correlation of SMI with severity of liver steatosis and liver fibrosis. The comparison analysis also suggested higher values of SMI in individuals with liver steatosis or fibrosis compared to healthy subjects. However, multiple logistic regression revealed that BMI, WC and HC were the independent predictors of liver steatosis, and SMI had no independent association with liver steatosis. In contrast, SMI played an independent role alongside BMI, WC and HC in predicting significant fibrosis. Multiple regression analysis revealed a protective role for SMI against liver fibrosis, independent of other obesity indices (BMI, WC, HC). Similarly, a ten-year follow-up of more than four thousand participants showed that an increase in fat depots increases the risk of developing MAFLD in obese individuals; however, a decrease in ASM had no association with MAFLD in obese individuals (27). Inconsistent with our findings, Jin, et al. (21) discovered that SMI and hemoglobin A1C are two independent risk factors for MAFLD, so each unit increase in SMI values, increases the odds of liver steatosis by 72%. The differences in the parameters included in the multiple regression model may have contributed to the conflicting results between the current study and theirs.

A significant aspect of our study is that SMI is associated with an increased risk of liver steatosis or significant fibrosis in univariate analysis and has a positive correlation with the severity of liver steatosis and liver fibrosis. This may be due to the positive correlation of SMI with obesity parameters such as BMI, WC, HC, and WHR (figure 1). Indeed, these obesity-related parameters had the highest correlation with the severity of liver steatosis and liver fibrosis. Consequently, SMI showed a positive correlation with liver steatosis and fibrosis in the positive direction. In the study by Kang, et al. (22), skeletal muscle area index (SMAI) showed a positive correlation with the severity of liver steatosis but a negative correlation with lobular inflammation. Moreover, they similarly discovered that SMAI had a positive correlation with visceral fat area. These results indicate that obese individuals necessarily do not possess worse skeletal muscle status than non-obese subjects. The SMI value was higher in MAFLD patients than in healthy individuals, which seems paradoxical since higher ASM is typically considered a sign of exercise and good cardiometabolic health (13). Indeed, the process of fatty liver begins when the amount of triglycerides received or produced exceeds the amount that is exported (28). High levels of insulin are secreted in individuals consuming a high-calorie diet, characterized by high levels of sugar, fat, and protein consumption (29). Insulin not only promotes glycogen synthesis and de novo lipogenesis but also stimulates protein synthesis in body tissues, particularly in muscles (30). This may explain why MAFLD patients have higher SMI than healthy individuals. Notably, SMI demonstrated a stronger correlation with ASM compared to skeletal muscle percentage, while exhibiting a weaker correlation with obesity-related parameters (figure 1). Thus, using SMI allowed us to better isolate and evaluate the independent contribution of ASM. The main limitation of the study was the sample size, which restricts the power of statistical analysis to detect subtle associations between body composition parameters and MAFLD. Additionally, the cross-sectional design of the study prevents us from drawing causal conclusions. We recommend that other researchers conduct a longitudinal study investigating the association of body composition indices with the risk of developing and progressing MAFLD, controlling for total caloric intake and expenditure, to clarify whether fat distribution and skeletal muscle status exert their influence on the MAFLD independent of malnutrition and calorie imbalance. In summary, after controlling for confounders, BMI, WC, and HC were significant predictors of both liver steatosis and fibrosis. However, unlike steatosis, liver fibrosis was negatively associated with SMI; each unit increase in SMI decreased the likelihood of liver fibrosis by 48%. These findings indicate that a higher skeletal muscle mass (or better muscle status) may not mitigate the adverse effects of obesity on steatosis, but it significantly reduces the odds of fibrosis. Additionally, an increase in waist circumference (a marker of abdominal size) plays a more significant role in increasing the risk of steatosis and fibrosis than an increase in trunk fat mass.

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Ethics approval: This study has been approved by the ethics committee of Hormozgan University of Medical Sciences (IR.HUMS.REC.1403.105) and has been conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Conflict of interests: None declared.

Authors' contribution: AB and FR contributed to the data collection, as well as the conceptualization and design of the

study. AB, EE, and AK were responsible for drafting the manuscript. SH conducted the statistical analysis and interpreted the results. FR and SA provided revisions to the manuscript. All authors reviewed the final version and confirmed the accuracy and consistency of the content.

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