Sarcopenia Is Associated with the Presence of Nonalcoholic Fatty Liver Disease in Zhejiang Province, China: A Cross-Sectional Observational Study.

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Research article

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Abstract

Background

Nowadays, both nonalcoholic fatty liver disease (NAFLD) and sarcopenia have attracted extensive attention in public health. However, the relationship between NAFLD and sarcopenia remains unclear. This study aimed to clarify the gender-specific association between sarcopenia and NAFLD according to the Asian Working Group for Sarcopenia (AWGS).

Methods

Dual-energy X-ray absorptiometry (DXA) and hepatic ultrasonography were measured in 578 participants (92 men and 486 women) during their annual health examinations. Multivariate logistic regression models were used to explore the association between NAFLD and sarcopenia with its two components.

Results

150 participants (30 men and 124 women) had NAFLD. The prevalence of sarcopenia was higher among the participants with NAFLD than those without (men: 20.0% vs. 9.7%, $P = 0.295$, women: 15.3% vs. 8.0%, $P = 0.019$). Low muscle mass (LMM) was independently associated with NAFLD in both men and women (men: odds ratio [OR], 2.88; 95% confidence interval [CI] 1.52–5.46; women: OR, 2.08; 95%CI 1.63–2.67). Whereas, low muscle strength (LMS) was independently associated with NAFLD only in men participants, with the OR of 1.15 (95%CI 1.02–1.28).

Conclusion

The occurrence of sarcopenia was associated with a higher risk of NAFLD, especially in men, as demonstrated by lower muscle mass and lower muscle strength.

Background

Nonalcoholic fatty liver disease (NAFLD) is currently the leading cause of chronic liver disease worldwide, ranging from simple steatosis, nonalcoholic steatohepatitis (NASH), advanced fibrosis, to end-stage liver diseases, such as cirrhosis and hepatocellular carcinoma [1]. The total NAFLD population in 2015 was estimated at 83.1 million cases with a prevalence rate of 30.0% among the population aged≥15 years and will increase to 33.5% by 2030 [2]. NAFLD has been proven to be closely associated with systemic diseases and has attracted extensive attention in public health [3]. The biological mechanisms, such as peripheral resistance to insulin, dyslipidemia, and the activation of inflammatory pathways associated with NAFLD are relevant to these systemic diseases [4].

Sarcopenia is a multifactorial geriatric syndrome with the overall concept of skeletal muscle failure or insufficiency. Skeletal muscle, as an active endocrine organ responsible for insulin-mediated glucose disposal, plays a significant role in glucose homeostasis, insulin resistance and inflammation [5]. Meanwhile, sarcopenia is associated with a sedentary lifestyle, which increases the risk for obesity, metabolic syndrome and NAFLD [6]. It has been reported that up to 60% of patients with end-stage liver disease accompanied with sarcopenia. And the presence of NASH in those patients was associated with a 6-fold increased risk of sarcopenic obesity [7, 8]. Thus, sarcopenia shares the common risk factors that contribute to NAFLD and have a plausible association with NAFLD.

Until now, all consensus agreed on two crucial components of sarcopenia definition: that sarcopenia involves both structural damage (low muscle mass [LMM]) and impaired function (low muscle strength [LMS]) [9]. Several studies have shown that sarcopenia including its two crucial components (LMM and LMS) is associated with the prevalence of NAFLD. Kim *et al.* found that skeletal muscle mass was positively correlated with the occurrence of NAFLD and negatively correlated with the resolution of existing NAFLD [10]. Another study showed that men and women with NAFLD had 7.3% and 7.9% lower handgrip strength (HGS) than controls in older adults [11]. Since the trajectories of muscle mass and muscle strength decline during ageing do not overlap, and muscle strength declines much more rapidly than muscle mass, it is essential to illustrate the association between NAFLD and sarcopenia with its two components in the same time [12, 13]. Furthermore, almost clinical researches were conducted in Korean populations and few were diagnosed according to the Asian Working Group for Sarcopenia (AWGS) consensus guidelines. Therefore, this study aimed to investigate the independent association of the two components of sarcopenia with NAFLD, stratified by gender, in the aged Chinese population, according to AWGS. Besides, we tried to assess which component could better predict NAFLD prevalence in different gender groups.

Methods

Study Population.

This is a cross-sectional, observational study of 578 senior hospital staffs (92 men and 486 women) who attended to the annual health examinations at the First Affiliated Hospital, School of Medicine, Zhejiang University between January 2019 and December 2019. Participants who had full records of personal health history, anthropometric and biochemical data and results of hepatic ultrasonography were initially enrolled. Participants who had cancer, viral/drug-induced/autoimmune liver diseases, severe cardiopulmonary disorders, renal dysfunction, history of organ transplant, physical or cognitive impairment, excessive alcoholic consumption (male >140 g/week or female >70 g/week) were excluded [14]. This study was approved by the Ethics Committee of The First Affiliated Hospital, School of Medicine, Zhejiang University in accordance with the Helsinki Declaration. All participants gave written informed consent before participation.

Laboratory measurements

All participants underwent a thorough physical examination. Height (cm) and weight (kg) were measured using standardized protocols while the participants were dressed in light clothing, without shoes. Body mass index (BMI) (kg/m^2) was calculated according to the following formula: BMI = weight(kg)/height(m)^2. According to Asia-Pacific criteria, general obesity was defined as BMI≥25 kg/m^2 . After an overnight fast of ≥8 hours, blood samples were obtained from the peripheral vein of each participant. All laboratory measurements, including liver function tests (alanine aminotransferase [ALT], aspartate aminotransferase [AST], and gamma-glutamyltransferase [GGT]), lipid profile (triglyceride [TG], total cholesterol [TC], high-density lipoprotein cholesterol [HDL-C], and low-density lipoprotein cholesterol [LDL-C]), fasting plasma glucose (FPG), albumin (ALB), creatinine (Cr) and uric acid (UA), were measured by a Hitachi 7600 autoanalyzer (Hitachi, Tokyo, Japan) using standard protocols.

Diagnosis of sarcopenia and relative parameters

The participants who had completed dual-energy X-ray absorptiometry (DXA), tests for HGS and 4m walking speed were selected. The gait speed (GS)≤0.8m/s was defined as low GS, as recommended by AWGS. Both the left and right HGSs were measured with a Jamar Hydraulic Hand Dynamometer (Jamar Hydraulic Hand Dynamometer Model 5030J1; Sammons Preston, Bolingbrook, IL, USA) for three times and the maximum value was used, according to the recommendations of American Society of Hand Therapists (ASHT). LMS was defined as ≤26.0 kg in men and ≤18.0 kg in women, as recommended by AWGS. Skeletal muscle mass was estimated by the skeletal muscle index (SMI) using appendicular skeletal muscle mass (ASM) divided by squared body height (kg/m^2), which were measured by DXA using a Hologic Discovery^{TM} device (Hologic, Waltham, MA, USA). LMM was defined as ≤7.0 kg/m^2 in men and ≤5.4 kg/m^2 in women as recommended by AWGS. Sarcopenia was diagnosed according to the criteria of the AWGS [15] (**Figure 1**). Sarcopenic obesity was defined as the presence of both sarcopenia and obesity.

Diagnosis of NAFLD

NAFLD was determined based on the results of hepatic ultrasound examination following the exclusion of alcohol consumption, viral, or autoimmune liver disease. Hepatic ultrasonography (US; Acuson Sequoia 512, Siemens, Mountain View, CA, USA) was carried out by experienced ultrasonographers. The ultrasonographers were blinded to the study design and clinical data. The criteria for the ultrasonic diagnosis of fatty liver were based on those recommended by the Chinese Liver Disease Association [16].

Statistical analysis

Continuous variables were presented as mean ± standard deviation (SD), and categorical variables were presented as frequency (percentage). The statistical significance of the differences in clinical and biochemical values between participants with and without NAFLD was analyzed by gender using Student's t-test for continuous variables and Chi-squared test for categorical variables. The Pearson correlation coefficient was calculated to assess the associations between muscle mass or muscle

strength and the relative parameters of NAFLD. Multivariate logistic regression models were performed to calculate the adjusted OR and 95%CI for exploring the associations of NAFLD with muscle mass and muscle strength. The following factors were considered independent variables for multivariate logistic regression analysis: Model 1: age and weight; Model 2: model 1 covariates plus BMI, TG, and ALT. A receiver operating characteristic (ROC) curve of muscle mass was developed to predict the presence of NAFLD both in men and women. All calculations were performed using IBM SPSS Statistics ver. 22.0 (IBM Co., Armonk, NY, USA) and the associated results were plotted using GraphPad Prism6 (GraphPad, San Diego, California, USA). Two-sided *P*-values <0.05 were considered statistically significant.

Result

Basic laboratory and clinical characteristics

Basic laboratory and clinical characteristics of the 578 participants (92 men and 486 women) with and without NAFLD enrolled in this study are shown in Table 1. Thirty (32.6%) men and 124 (25.5%) women participants had NAFLD. Among the men, the participants with NAFLD were younger and had higher body weight, BMI, serum TG, ALT compared with those without NAFLD, but there were no differences in height, serum TC, HDL-C, LDL-C, AST, GGT, ALB, Cr, FPG and UA between the two groups. Among the women, the participants with NAFLD were older and had higher weight, BMI, TG, serum AST, ALT, GGT, ALB, FPG, and UA, and lower HDL-C compared with those without NAFLD. However, there were no differences in height, serum TC, LDL-C and Cr between the two groups. As illustrated in Table 1, the levels of GS were lower in NAFLD patients, compared with NAFLD-free participants both in men and women (men: 1.1 ± 0.3 *vs*. 1.3 ± 0.3 kg/m^2, *P* = 0.008; women: 1.1 ± 0.3 *vs*. 1.2 ± 0.2 kg/m^2, *P* = 0.008). The levels of muscle mass were also significantly lower in NAFLD patients, compared with NAFLD-free participants (men: 6.6 ± 1.2 *vs*. 7.2 ± 1.3 kg/m^2, *P* = 0.032; women: 5.2 ± 1.3 *vs*. 5.6 ± 1.1 kg/m^2, *P* = 0.011). Whereas, there was no statistical difference in the levels of HGS between the two groups (men: 35.8 ± 8.6 *vs*. 37.6 ± 9.7, *P* = 0.404, women: 23.4 ± 5.8 *vs*. 24.5 ± 5.1 kg, *P* = 0.053). The prevalence of sarcopenia was higher among the participants with NAFLD (men: 20.0% *vs*. 9.7%, *P* = 0.295, women: 15.3% *vs*. 8.0%, *P* = 0.019). Moreover, the prevalence of sarcopenic obesity was also higher in the NAFLD group both in men and women, but without statistical difference (men: 10.0% *vs*. 3.2%, *P* = 0.394, women: 3.2% *vs*. 0.8%, *P* = 0.134).

Associations of muscle mass and anthropometric, biochemical variables of NAFLD

We performed Pearson correlation analysis to determine the correlations between muscle mass and anthropometric, biochemical variables of NAFLD. The correlation analyses between anthropometric, biochemical variables and muscle mass in participants were shown in Table 2. We found that muscle mass was positively correlated with body weight (r = 0.292, *P* = 0.005), BMI (r = 0.291, *P* = 0.005), and HGS (r = 0.315, *P* = 0.002), while negatively correlated with age (r = − 0.244, *P* = 0.019) and FPG (r = − 0.251, *P* = 0.016) among men participants. Meanwhile, muscle mass was positively correlated with age (r = 0.111, *P* = 0.015), body weight (r = 0.295, *P*<0.001), BMI (r = 0.326, *P*<0.001), serum ALT (r = 0.139, *P* = 0.002), UA (r = 0.142, *P* = 0.002), and FPG (r = 0.111, *P* = 0.015) among women participants (Table 2).

Associations of HGS and anthropometric, biochemical variables of NAFLD

We found that HGS was positively correlated with body weight (r = 0.255, P = 0.014), height (r = 0.51, P<0.001), GS (r = 0.408, P<0.001) and muscle mass (r = 0.315, P = 0.002), while negatively correlated with age (r = − 0.504, P<0.001) among men participants. Meanwhile, HGS was positively correlated with body weight (r = 0.206, P<0.001), GS (r = 0.538, P<0.001) and height (r = 0.315, P<0.001), while negatively correlated with age (r = − 0.533, P<0.001), serum AST (r = − 0.391, P<0.001), TG (r = − 0.144, P = 0.002), UA (r = − 0.193, P<0.001), and FPG (r = − 0.177, P<0.001) among women participants. (Table 3)

Independent impact of muscle mass and muscle strength on the presence of NAFLD

A logistic regression model was conducted to evaluate the gender-specific relationship between the components of sarcopenia (LMM or LMS) and NAFLD risk (models 1–2, Table 4). The relationship between LMM and NAFLD was statistically significant in both models. In model 1, the OR with 95% CI for NAFLD were 2.91 (95%CI, 1.58–5.35) and 1.89 (95%CI 1.51–2.38) in men and women, respectively. Further, the fully adjusted model (model 2) showed LMM was still associated with an increased risk of NAFLD with OR of 2.88 (95%CI 1.52–5.46) in men and 2.08 (95%CI 1.63–2.67) in women. Participants with LMS showed significantly high odds of NAFLD with OR of 1.15 (95% CI, 1.04–1.26) and 1.15 (95%CI 1.02–1.28) in men after adjusting in Model 1 and Model 2. However, there was no statistical significance in muscle strength in women after adjusting in Model 1 and Model 2, with OR of 1.05 (95%CI 0.99–1.11) and 1.01 (95%CI 0.95–1.07), respectively.

ROC curve of muscle mass

The ROC curves of muscle mass plotted for the diagnoses of NAFLD by gender were shown in **Fig. 2**. The cut-off value of muscle mass was 8.0 kg/m^2 in men and 4.9 kg/m^2 in women, with the sensitivity of 33.9% and 68.8%, and the specificity of 90.0% and 47.6%, respectively. The areas under ROC for NAFLD were 0.624 (95% CI 0.501–0.748, P< 0.063) and 0.592 (95% CI 0.531–0.653, P < 0.031) in men and women, respectively.

Discussion

NAFLD is a spectrum of liver disease that now becomes the most common cause of chronic liver disease in adults of all ethnicities. The risk of NAFLD increases with age [17]. Sarcopenia refers to involuntary loss of muscle mass, muscle strength and muscle performance that occurs with ageing. And it has been considered as a new geriatric syndrome that closely associated with metabolic disorders [18]. In this cross-sectional sample of Chinese study, we aimed at examining the gender-specific association between NAFLD and sarcopenia with its two crucial components, using hepatic imaging and DXA scans, according to the AWGS criteria. Our data indicated that men and women with NAFLD both had markedly lower muscle mass, and were more likely to have lower muscle strength than controls. And the status of sarcopenia and sarcopenic obesity lead to an increased prevalence of NAFLD. Moreover, LMM appeared

to be a better predictor for NAFLD prevalence than LMS. Further multivariable analysis identified that LMM had statistically higher odds of suffering from NAFLD compared to LMS both in men and women.

Recent clinical studies have already demonstrated a positive relationship between sarcopenia and the prevalence of NAFLD [6, 11, 19, 20]. Hong *et al.* firstly found the OR for NAFLD risk was 5.16 (95%CI 1.63–16.33) in the lowest quartile of SMI compared to the highest quartile [6]. Then, Kim *et al.* showed that men and women with NAFLD had markedly lower HGS and were more likely to have LMS than controls [11]. However, most of these studies focused on the single component of sarcopenia, which was not sufficient to fully understand the relationship between muscle status and NAFLD. It is unclear whether LMM or LMS is independently associated with NAFLD in the same model. In the present study, we found that men and women with NAFLD had markedly lower SMI and were more likely to have LMM than controls. LMM was associated with an increased risk of NAFLD with OR of 2.88 (95%CI 1.52–5.46) in men and 2.08 (95%CI 1.63–2.67) in women. However, participants with LMS showed a slightly higher odd of NAFLD with OR of 1.15 (95%CI 1.02–1.28) in men only.

Previous studies also attempted to elucidate the mechanism of sarcopenia development in patients with NAFLD. Nowadays, growing attention has been paid in gender-specific differences in the development of sarcopenia and NAFLD. In this study, men with LMM had increased risk of suffering from NAFLD with the higher odds after adjustment compared to women participants. When it comes to muscle strength, it indicated that low HGS was associated with an increased NAFLD prevalence in men only. Yang *et al.* found that some metabolic syndromes may put men more prone to have sarcopenia, likely due to the low levels of physical activity associated with such conditions [21]. A small cross-sectional study from Japan revealed that the SMI had a negative association with hepatic steatosis only in men with type 2 diabetes [22]. Then, a more extensive population-based study involving 4210 men and women also suggested that sarcopenia was independently associated with NAFLD in men with type 2 diabetes, while no significant difference was found in women [23]. Some studies have confirmed that dysregulated sexual hormone disorders are involved in the pathogenesis of NAFLD and sarcopenia. Testosterone, as a potent anabolic hormone, can promote muscle protein synthesis [24]. Sarcopenia, which is related to the reduction of physical activity, the lack of anabolic hormones, and the decrease of pro-inflammatory cytokines, has been associated with NAFLD independent of metabolic syndrome features [5, 25]. Extrapolating from these findings, the regulation of sex hormones may be involved in the mechanism of the gender-specific differences in the development of sarcopenia and NAFLD.

Several epidemiological and experimental studies have shown that insulin resistance may have an essential role in sarcopenia. The skeletal muscle is recognized as a tissue that primary responsible for peripheral insulin-mediated glucose disposal. Insulin resistance may reduce the stimulation of protein synthesis pathway, and at the same time, increase the activation of protein degradation pathway that might eventually lead to muscle loss [18, 26]. Indeed, the prevalence of sarcopenic obesity is higher in the NAFLD participants in our study. Meanwhile, Pearson correlation coefficient analyses confirmed that the level of FPG was inversely associated with muscle mass in men and HGS in women. And the level of serum of TG was also negatively correlated with HGS in women. In this context, our current research also

implied that both LMM and LMS might participate in the progress of NAFLD through dyslipidemia and insulin resistance.

It is also worth mentioning that compared to LMS, LMM had higher odds of increasing the risk of NAFLD in our study. And in women, sarcopenia was only found to have a statistically significant relationship with NAFLD when defined in terms of muscle mass alone, which was consistent with the early research focused on the relationship between sarcopenia and metabolic syndrome. This might suggest that muscle mass, to some extent, was more significant than muscle strength in the context of NAFLD.

However, several limitations should also be considered when interpreting the results. First, the diagnosis of NAFLD was based on hepatic ultrasonography, and histologic confirmation of NAFLD by liver biopsy was not available. Second, the study limited to verify causality due to the cross-sectional design. Third, the information regarding the past medical history was self-reported, which might have led to recall bias. Then, the number of subjects with sarcopenic obesity was low, which further need an enlargement of the sample size to allow an adequate statistical comparison. At last, our study population was exclusively Chinese, so the results might not be generalizable to other populations.

Conclusion

This work is among the few studies to examine the independent association of the two crucial components of sarcopenia with NAFLD, stratified by gender according to AWGS. LMM was consistently associated with NAFLD in both men and women, while LMS was associated with NAFLD only in men, after adjustment for potential confounders. Moreover, compared to muscle strength, muscle mass was a better predictor for the presence of NAFLD in both genders. Given that the understanding of the close relationship between NAFLD and sarcopenia is of great interest in this era of the ageing population, further more well-designed studies should focus on the common therapeutic strategies to prevent from muscle wasting as well as NAFLD.

Abbreviations

ALB: albumin; ALT: alanine aminotransferase; ASM: appendicular skeletal muscle mass; AST: aspartate aminotransferase; AWGS: Asian Working Group for Sarcopenia; CI: confidence intervals; Cr: creatinine; DXA: dual-energy X-ray absorptiometry; FPG: fasting plasma glucose; GGT: gamma-glutamyltransferase; GS：gait speed; HDL-C: high-density lipoprotein cholesterol; HGS: handgrip strength; LDL-C: low-density lipoprotein cholesterol; LMM: low muscle mass; LMS: low muscle strength; NAFLD: nonalcoholic fatty liver disease; NASH: nonalcoholic steatohepatitis; OR: odds ratio; SMI: skeletal muscle index; TC: total cholesterol; TG: triglyceride.

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The research ethics committee of the First Affiliated Hospital, School of Medicine, Zhejiang University approved the study. The reference number was 2008-1106. Written informed consent was obtained from all participants included in the study.

Consent to publish

Not Applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Y.M Yang and Y.M Wang conceived and designed the study. D.F Deng, Y.C Yang and W.W Lu collected patient samples; W.J Zhou and J Xu proofed the data; K.F Zhu and W.J Zhou performance the statistical analysis under the guidance of Q Zhang. Y.M Wang wrote the first draft of the manuscript. K.F Zhu and W.J Zhou mostly involved in several revisions of the manuscript. Y.M Yang provided critical comments and joined other authors in revising the manuscript and approving the final submission. All authors have read and approved the manuscript in its current state.

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Tables

Table 1. Baseline characteristics of the study participants, categorized according to the presence or absence of NAFLD

Variable	Men（n=92)			Women（n=486）		
	Without NAFLD (n=62)	With NAFLD (n=30)	*P*	Without NAFLD (n=362)	With NAFLD (n=124)	*P*
Ages(years)	**72.9±9.2**	**68.9±5.8**	**0.029**	**62.9±12.1**	**67.5±10.1**	**<0.001**
Weight(Kg)	**67.3±9.6**	**75.3±12.2**	**<0.001**	**55.8±7.0**	**61.5±8.3**	**<0.001**
Height(cm)	170.7±6.1	169.0±6.4	0.239	157.3±9.4	157.7±4.9	0.587
Body mass index(Kg/m2)	**23.6±3.0**	**25.7±3.3**	**0.002**	**22.5±2.6**	**24.7±2.8**	**<0.001**
TC (mmol/L)	4.4±0.9	4.6±0.9	0.312	5.0±0.9	5.1±1.1	0.711
TG (mmol/L)	**0.8±0.5**	**1.8±0.7**	**<0.001**	**1.2±0.7**	**1.6±1.0**	**<0.001**
HDL-C (mmol/L)	1.3±0.3	1.2±0.4	0.065	**1.5±0.4**	**1.3±0.3**	**<0.001**
LDL-C (mmol/L)	2.5±0.7	2.6±0.8	0.426	2.8±0.7	2.9±0.9	0.321
AST (IU/L)	23.0±3.1	23.6±4.4	0.665	**21.3±6.0**	**26.1±7.2**	**0.003**
ALT (IU/L)	**19.3±7.4**	**25.5±14.0**	**0.007**	**17.6±10.2**	**24.5±14.6**	**<0.001**
GGT (IU/L	28.0±17.6	29.5±13.7	0.696	**21.2±16.8**	**28.4±18.4**	**<0.001**
ALB(g/L) albumin	46.6±2.4	47.6±2.0	0.061	**46.9±2.4**	**47.9±2.5**	**<0.001**
Cr (umol/L) creatinine	89.1±15.3	87.2±12.3	0.555	64.9±11.0	64.6±11.1	0.789
FPG (mmol/L)	5.6±1.4	5.9±1.1	0.236	**5.2±1.0**	**5.8±1.2**	**<0.001**
UA (μmol/L)	357.5±84.2	371.0±77.5	0.461	**267.3±63.4**	**305.4±71.0**	**<0.001**
Gait speed (m/s)	**1.3±0.3**	**1.1±0.3**	**0.008**	**1.2±0.2**	**1.1±0.3**	**0.008**
Muscle strength (kg)	37.6±9.7	35.8±8.6	0.404	24.5±5.1	23.4±5.8	0.053
Muscle mass (kg/m^2)	**7.2±1.3**	**6.6±1.2**	**0.032**	**5.6±1.1**	**5.23±1.3**	**0.011**
Sarcopenia	6/62 (9.7%)	6/30 (20.0%)	0.295	**29/362(8.0%)**	**19/124(15.3%)**	**0.019**
Sarcopenic obesity	2/62 (3.2%)	3/30 (10.0%)	0.394	3/362 (0.8%)	4/124 (3.2%)	0.134

Values are presented as mean ± standard deviation, unless otherwise specified. Bold numbers indicate statistically significant values. NAFLD, non-alcoholic fatty liver disease; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; ALB, albumin; Cr, creatinine; FPG, fasting plasma glucose; UA, uric acid.

Table 2. Pearson correlation coefficients between muscle mass and patient characteristics at baseline by gender (for men and women).

Variable	Muscle mass (kg/m^2)			
	men		women	
	r	*P*	r	*P*
Age (years)	-0.244	0.019	0.111	0.015
Body weight (kg)	0.292	0.005	0.295	<0.001
Height (cm)	0.098	0.355	-0.078	0.088
BMI (kg/m^2)	0.291	0.005	0.326	<0.001
ALT (IU/L)	0.093	0.378	0.139	0.002
AST (IU/L)	0.243	0.167	0.177	0.101
TG (mmol/L)	-0.025	0.815	-0.01	0.834
TC (mmol/L)	-0.108	0.304	-0.055	0.232
ALB (g/L)	0.232	0.026	-0.003	0.943
Cr (μmol/L)	0.101	0.339	0.193	<0.001
UA (μmol/L)	0.179	0.087	0.142	0.002
FPG (mmol/L)	-0.251	0.016	0.111	0.015
Gait speed (m/s)	0.153	0.145	-0.026	0.568
Muscle strength (kg)	0.315	0.002	-0.041	0.364

Table 3. Pearson correlation coefficients between muscle strength and patient characteristics at baseline by gender (for men and women).

Variable	Muscle strength (kg)			
	men		women	
	r	*P*	r	*P*
Age (years)	-0.504	<0.001	-0.533	<0.001
Body weight (kg)	0.255	0.014	0.206	<0.001
Height (cm)	0.51	<0.001	0.315	<0.001
BMI (kg/m^2)	0.02	0.848	-0.018	0.692
ALT (IU/L)	0.062	0.554	-0.065	0.156
AST (IU/L)	0.08	0.651	-0.391	<0.001
TG (mmol/L)	0.15	0.152	-0.144	0.002
TC (mmol/L)	0.019	0.854	0.011	0.804
ALB (g/L)	0.184	0.079	0.142	0.002
Cr (μmol/L)	0.013	0.901	-0.155	0.001
UA (μmol/L)	0.059	0.578	-0.193	<0.001
FPG (mmol/L)	-0.195	0.062	-0.177	<0.001
Gait speed (m/s)	0.408	<0.001	0.538	<0.001
Muscle mass (kg/m^2)	0.315	0.002	-0.041	0.364

Table 4. ORs and 95% CIs of muscle mass and muscle strength for NAFLD

	Men			
Variable	Model 1		Model 2	
	OR (95%CI)	*P*	OR (95%CI)	*P*
Muscle mass, per SD decrease	**2.91 (1.58, 5.35)**	**0.001**	**2.88 (1.52, 5.46)**	**0.001**
Muscle strength, per SD decrease	**1.15 (1.04, 1.26)**	**0.004**	**1.15 (1.02, 1.28)**	**0.021**
	Women			
Variable	Model 1		Model 2	
	OR (95%CI)	*P*	OR (95%CI)	*P*
Muscle mass, per SD decrease	**1.89（1.51, 2.38）**	**＜0.001**	**2.08 (1.63, 2.67)**	**＜0.001**
Muscle strength, per SD decrease	1.05（0.99, 1.11）	0.058	1.01 (0.95, 1.07)	0.716

Results are given as OR (95% CI) for NAFLD as outcome stratified by gender. Results in bold reflect significant findings with a P value <0.05. Model 1:

adjusted for age and weight. Model 2: adjusted for age, weight, BMI, TG, ALT. NAFLD, nonalcoholic fatty liver disease; BMI, body mass index; TG, triglyceride; ALT, alanine aminotransferase.

Figures

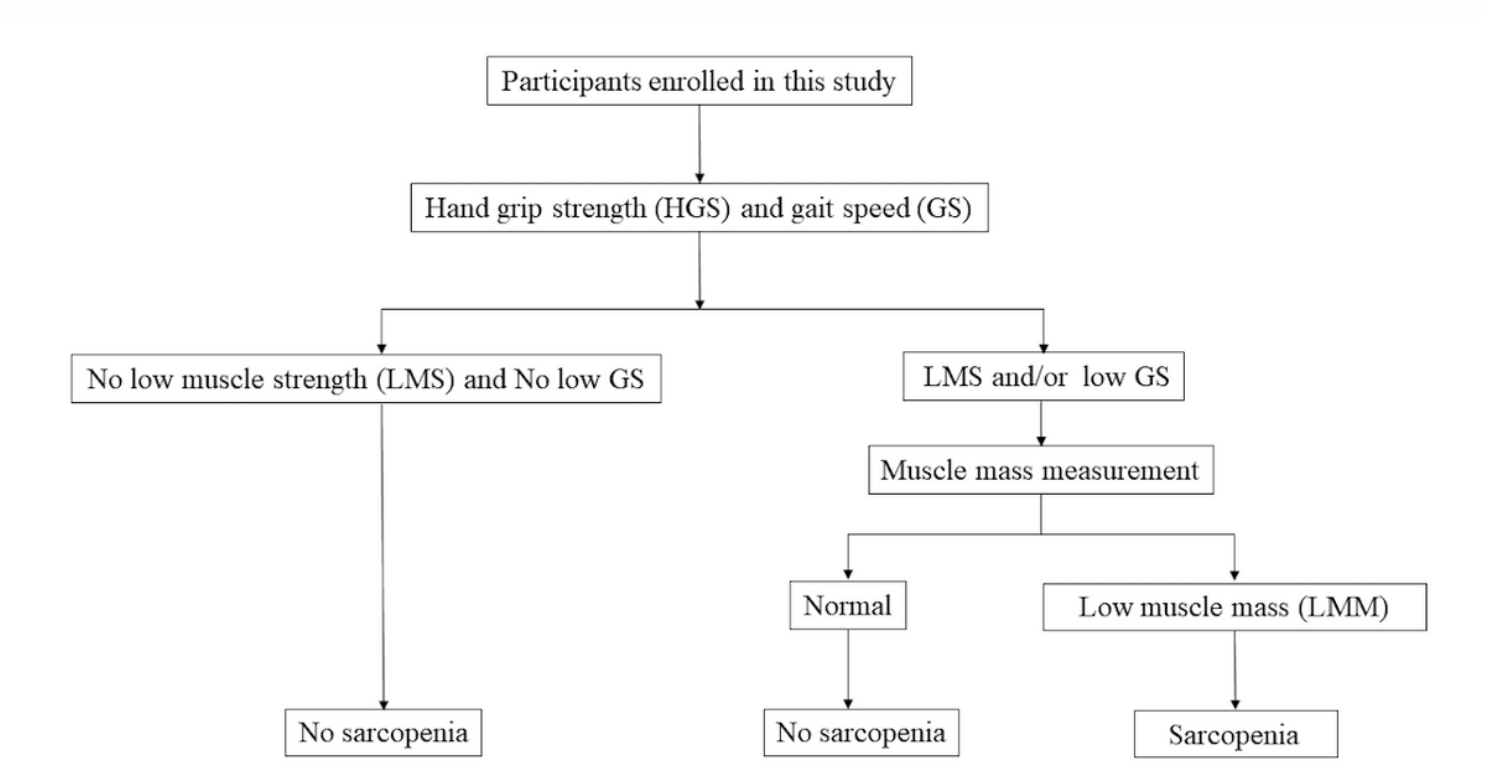

Figure 1

Recommended diagnostic algorithm of Asian Working Group for Sarcopenia (AWGS).

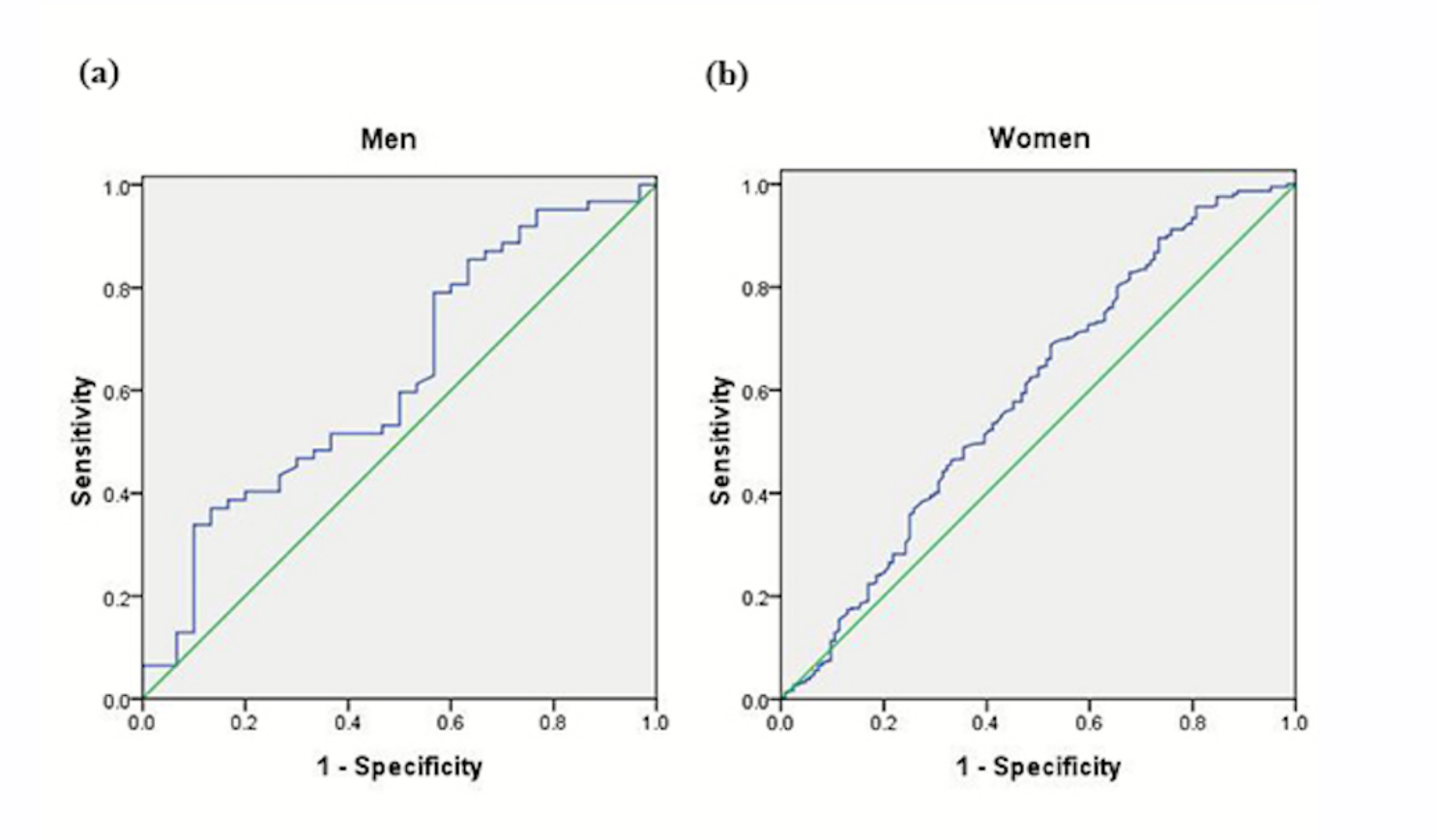

Figure 2

ROC curves of muscle mass to predict the presence of NAFLD by gender. (a) Men (AUC = 62.4%, P <0.063); (b) Women (AUC = 59.2%, P<0.031).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- supplement1.xlsx