

Diagnostic Accuracy of Quadriceps and Forearm Muscle Ultrasound Compared with CT-Derived Skeletal Muscle Index for Assessment of Sarcopenic Obesity in Patients with Cirrhosis

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Abstract

Background: Sarcopenic obesity is an under-recognised but clinically important complication of cirrhosis, and a simple bedside tool for muscle assessment may help identify patients with reduced muscle mass and systemic inflammation.

Objective: To compare the diagnostic accuracy of muscle ultrasound measurements, namely quadriceps muscle thickness and forearm muscle thickness, with CT-derived skeletal muscle index for the assessment of sarcopenic obesity in patients with cirrhosis.

Methods: This single-centre, hospital-based, analytical cross-sectional study was conducted among 75 patients with cirrhosis and sarcopenic obesity attending the OPD and/or inpatient wards of the Department of Medical Gastroenterology at a tertiary teaching healthcare facility in India from July 2025 to June 2026.

Results: CT-defined sarcopenic obesity was present in 34 (45.3%). Patients with CT-SO were older (55.1 ± 7.8 vs. 51.5 ± 7.5 years, $p=0.044$), had longer cirrhosis duration, and higher MELD-Na scores (16.8 ± 4.9 vs. 12.6 ± 2.6 , $p<0.001$). They also had lower mid-arm circumference, hand-grip strength, gait speed, albumin, and CT-SMI, with higher CRP (27.4 ± 11.4 vs. 13.4 ± 4.4 mg/L, $p<0.001$). Ultrasound-derived quadriceps and forearm thickness were significantly reduced in CT-SO and strongly correlated with CT-SMI. Quadriceps compression thickness showed the best diagnostic performance (AUC=0.960; sensitivity 91.2%; specificity 90.2%). CRP independently predicted lower CT-SMI after adjustment.

Conclusion: Muscle ultrasound, particularly quadriceps thickness, was a reliable bedside surrogate for CT-derived skeletal muscle index in identifying sarcopenic obesity in cirrhosis, and higher CRP levels were associated with greater muscle depletion.

Keywords: Cirrhosis, Sarcopenic obesity, Muscle ultrasound, Skeletal muscle index, Quadriceps thickness, C-reactive protein.

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Introduction

Cirrhosis is a major global health problem and represents the final common pathway of several chronic liver diseases, including alcohol-related liver disease, viral hepatitis, and metabolic dysfunction-associated steatotic liver disease. Asrani et al. reported that liver disease accounted for approximately two million deaths annually worldwide, including about one million deaths due to complications of cirrhosis, highlighting the continuing clinical and public health relevance of advanced chronic liver disease. [1] Beyond hepatic

dysfunction, cirrhosis is increasingly recognised as a systemic disorder characterized by metabolic derangements, malnutrition, inflammation, and progressive loss of skeletal muscle mass and function. Sarcopenia is one of the most clinically important extrahepatic complications of cirrhosis. Tantai et al., in a meta-analysis of patients with cirrhosis, reported an overall sarcopenia prevalence of 37.5%, with a higher prevalence among males, patients with alcohol-related liver disease, and those with advanced liver dysfunction. [2] The

prognostic importance of sarcopenia has also been well established, as Tantai et al. found that sarcopenia was independently associated with an approximately two-fold higher risk of mortality in cirrhosis. [2] Carey et al., in a multicentre study of patients with end-stage liver disease awaiting liver transplantation, proposed CT-derived L3 skeletal muscle index cut-offs of $<50 \text{ cm}^2/\text{m}^2$ in men and $<39 \text{ cm}^2/\text{m}^2$ in women for defining sarcopenia, thereby reinforcing the value of CT-based body composition assessment in this population. [3]

Sarcopenic obesity is a particularly important phenotype because excess adiposity may conceal loss of skeletal muscle on routine clinical examination and body mass index assessment. Montano-Loza et al. reported that sarcopenic obesity and myosteatosis were associated with higher mortality in patients with cirrhosis, with sarcopenia and myosteatosis increasing mortality risk by approximately 1.5- to 2-fold. [4] Carias et al. further showed that non-alcoholic steatohepatitis was associated with a six-fold increased risk of sarcopenic obesity among cirrhotic patients undergoing liver transplant evaluation. [5] Although CT-derived skeletal muscle index is an objective reference measure, it is not always practical for repeated bedside or outpatient assessment. Quadriceps muscle ultrasound has emerged as a portable, non-invasive tool for muscle assessment; Pardo et al. demonstrated good intra- and inter-observer reliability for quadriceps muscle thickness measurement, with intra-observer intraclass correlation coefficients up to 0.83 and inter-observer coefficients up to 0.81. [6] Systemic inflammation may further contribute to muscle depletion in chronic liver disease, as Kumar et al. described hyperammonemia, amino acid deprivation, hormonal imbalance, gut dysbiosis, insulin resistance, and chronic inflammation as key contributors to altered muscle protein turnover in chronic liver disease. [7] Against this background, the objectives of the present study were to compare the diagnostic accuracy of muscle ultrasound measurements, namely quadriceps muscle thickness and forearm muscle thickness, with CT-derived skeletal muscle index for the assessment of sarcopenic obesity in patients with cirrhosis, and to investigate the relationship between systemic inflammation, as measured by CRP levels, and muscle mass in cirrhotic patients.

Materials and Methods

This was a single-centre, hospital-based, analytical cross-sectional study conducted in the outpatient department (OPD) and/or inpatient wards of the Department of Gastroenterology and Hepatology, in a tertiary teaching healthcare facility in India over a period of 12 months (July 2025–June 2026). The study was approved by the Institutional Human Ethics Committee (IHEC). Each participant, along

with their attendant when applicable, was provided with a Participant Information Sheet (PIS) translated into their local language. The information was also explained verbally to ensure clear understanding and voluntary agreement. Patients were included if they were aged 18 years or older, had a diagnosis of cirrhosis, had a body mass index (BMI) of $\geq 25 \text{ kg}/\text{m}^2$, and fulfilled the criteria for sarcopenia. Patients with hepatocellular carcinoma or other active malignancies, overt hepatic encephalopathy at the time of assessment, end-stage renal disease, cardiac failure, stroke, pregnancy, or lack of informed written consent were excluded.

The sample size for the present study was calculated using Dhariwal et al.; ultrasound-derived muscle indices showed a positive correlation with CT-derived skeletal muscle index, with correlation coefficients ranging from 0.40 to 0.58. (8) For the present study, a conservative expected correlation coefficient of 0.40 was considered. Assuming a two-sided alpha error of 5% and 90% power, and using Fisher's z transformation formula for correlation, the minimum required sample size was rounded off to 75 participants; enrolled using nonprobability sampling technique – convenience sampling/complete enumeration. Demographic details, including age and sex were recorded, followed by details related to cirrhosis, presenting symptoms, and relevant comorbidities, including diabetes mellitus, hypertension, dyslipidaemia, ischaemic heart disease, heart failure, chronic kidney disease, thyroid disease, autoimmune disease, reactive airway disease, malignancy, cerebrovascular disease, and other associated illnesses. A detailed clinical examination was performed. Anthropometric and nutritional assessment included measurement of height, dry weight, BMI, mid-arm circumference, and triceps skin-fold thickness.

Muscle strength was assessed using hand-grip strength measured with a handheld hydraulic dynamometer in the dominant hand, with the elbow maintained at 90° ; three readings were recorded, and the maximum value was used for analysis. Low hand-grip strength was defined as $<25.63 \text{ kg}$ in males and $<16.7 \text{ kg}$ in females. Muscle function was assessed using the gait speed test, and a gait speed of $<0.8 \text{ m/s}$ was considered reduced.

Muscle mass was assessed using computed tomography-derived skeletal muscle index, calculated from the skeletal muscle cross-sectional area at the third lumbar vertebral level and normalized for height squared, and low skeletal muscle index was defined as $<36.54 \text{ cm}^2/\text{m}^2$ in males and $<30.21 \text{ cm}^2/\text{m}^2$ in females. Sarcopenia was considered present when at least two of the three domains, namely reduced muscle strength, reduced muscle mass, and impaired muscle

function, were positive. Ultrasonographic assessment of muscle thickness was then performed using B-mode ultrasonography. Quadriceps muscle thickness was measured over the thigh at two standardized points, located at one-half and one-third of the distance from the superior border of the patella to the iliac crest. At each point, two readings were obtained, including a compression reading and a feather-weight reading, and the readings from both points were averaged. Forearm muscle thickness was measured on the dominant upper limb using B-mode ultrasonography with a high-frequency linear transducer of 7–14 MHz on a Philips IU22 machine. Measurements were obtained over the anterior forearm at one-third and two-thirds of the distance between the styloid process and the head of the radius. Blood investigations were performed for all participants and included complete hemogram, renal function tests, serum electrolytes, liver function tests, prothrombin time/international normalized ratio, CRP, HbA1c, thyroid-stimulating hormone and alpha-fetoprotein.

Statistical analysis: Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for distribution and were expressed as mean and standard deviation when normally distributed, while categorical variables were expressed as frequency and percentage. Patients were classified according to the presence or absence of CT-defined sarcopenic obesity, and comparisons between the two groups were performed using the independent-samples t-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. The relationship between ultrasound-derived muscle measurements and CT-derived skeletal muscle index was evaluated using Pearson's correlation coefficient. The diagnostic performance of quadriceps and forearm muscle thickness measurements for identifying CT-defined sarcopenic obesity was assessed using receiver operating characteristic curve analysis, and the area under the curve, optimal cut-off value, sensitivity, specificity, positive predictive value, negative predictive value, and overall diagnostic accuracy were calculated. Multivariable linear regression analysis was performed with CT-derived skeletal muscle index as the dependent variable to identify independent predictors after adjusting for relevant covariates, including CRP, age, sex, BMI, and MELD-Na score. A p value of <0.05 was considered statistically significant.

Results

Among the 75 patients included in the study, CT-defined sarcopenic obesity (CT-SO) was present in 34 patients and absent in 41 patients. The mean age

was significantly higher in the CT-SO group than in the non-CT-SO group (55.1 ± 7.8 vs. 51.5 ± 7.5 years, $p=0.044$), while gender distribution was comparable. Patients with CT-SO had a significantly longer duration of cirrhosis (4.9 ± 2.6 vs. 3.8 ± 2.0 years, $p=0.049$) and a higher MELD-Na score (16.8 ± 4.9 vs. 12.6 ± 2.6 , $p<0.001$), indicating more advanced liver disease. Alcohol-related cirrhosis was the most common etiology overall (42.7%), followed by MASLD/NASH (29.3%), with no significant difference between groups. Fatigue and abdominal distension/ascites symptoms were significantly more frequent in patients with CT-SO (82.4% vs. 46.3%, $p=0.002$; and 64.7% vs. 31.7%, $p=0.006$, respectively), while most comorbidities, including diabetes mellitus, hypertension, dyslipidaemia, chronic kidney disease, and thyroid disease, were comparable between groups. On clinical and functional assessment, patients with CT-SO had significantly lower mid-arm circumference (25.1 ± 2.0 vs. 28.1 ± 2.9 cm, $p<0.001$), hand-grip strength (21.0 ± 5.5 vs. 26.5 ± 5.9 kg, $p<0.001$), and gait speed (0.71 ± 0.11 vs. 0.90 ± 0.16 m/s, $p<0.001$). Low hand-grip strength and reduced gait speed were also significantly more common in the CT-SO group (70.6% vs. 12.2% and 82.4% vs. 19.5%, respectively; both $p<0.001$). Pedal edema on examination was more frequent among patients with CT-SO (55.9% vs. 22.0%, $p=0.004$). Laboratory evaluation showed that CT-SO was associated with higher total leukocyte count, ESR, urea, direct bilirubin, AST, PT/INR, and CRP, along with lower platelet count, sodium, total protein, and albumin. Notably, CRP was markedly higher in the CT-SO group than in the non-CT-SO group (27.4 ± 11.4 vs. 13.4 ± 4.4 mg/L, $p<0.001$), while albumin was significantly lower (2.84 ± 0.46 vs. 3.31 ± 0.47 g/dL, $p<0.001$).

CT-derived skeletal muscle index (CT-SMI) was significantly lower among patients with CT-SO than among those without CT-SO (31.41 ± 2.86 vs. 37.47 ± 3.92 cm²/m², $p<0.001$). Similarly, all ultrasound-derived muscle measurements were significantly reduced in the CT-SO group, including quadriceps feather-weight thickness (1.22 ± 0.21 vs. 1.79 ± 0.25 cm), quadriceps compression thickness (1.01 ± 0.19 vs. 1.47 ± 0.20 cm), mean quadriceps muscle thickness (1.12 ± 0.20 vs. 1.63 ± 0.22 cm), and forearm muscle thickness (1.06 ± 0.13 vs. 1.31 ± 0.19 cm) (all $p<0.001$). Ultrasound measurements demonstrated strong positive correlations with CT-SMI, with the highest correlation observed for quadriceps feather-weight thickness ($r=0.835$), followed by mean quadriceps muscle thickness ($r=0.830$), quadriceps compression thickness ($r=0.818$), and forearm muscle thickness ($r=0.724$) (all $p<0.001$). ROC analysis showed excellent diagnostic performance of quadriceps ultrasound measurements for

identifying CT-defined sarcopenic obesity. Quadriceps compression thickness achieved the highest diagnostic accuracy (AUC=0.960, 95% CI: 0.916–0.991), with an optimal cut-off of ≤ 1.21 cm, sensitivity of 91.2%, specificity of 90.2%, and overall accuracy of 90.7%. Mean quadriceps muscle thickness and quadriceps feather-weight thickness also demonstrated excellent discrimination (AUC=0.956 and 0.955, respectively), whereas forearm muscle thickness showed good diagnostic performance (AUC=0.847, 95% CI: 0.752–0.925). Correlation analysis further demonstrated that increasing CRP levels were significantly associated with lower CT-SMI ($r=-0.655$, $p<0.001$), lower mean quadriceps muscle

thickness ($r=-0.665$, $p<0.001$), lower forearm muscle thickness ($r=-0.512$, $p<0.001$), reduced hand-grip strength ($r=-0.461$, $p<0.001$), slower gait speed ($r=-0.253$, $p=0.028$), and lower serum albumin levels ($r=-0.290$, $p=0.012$). On multivariable linear regression analysis, higher CRP levels remained independently associated with lower CT-SMI ($\beta=-1.72$ per 10 mg/L increase, 95% CI: -2.36 to -1.09; $p<0.001$), while male sex ($\beta=5.04$, $p<0.001$) and BMI ($\beta=0.24$, $p=0.036$) were independently associated with higher CT-SMI, whereas increasing MELD-Na score was independently associated with lower CT-SMI ($\beta=-0.22$, $p=0.006$). Age was not an independent predictor of CT-SMI after adjustment ($p=0.072$).

Table 1: Sociodemographic, cirrhosis-related, symptom, and comorbidity profile

		Overall (N=75)	CT-SO present (n=34)	CT-SO absent (n=41)	P value
Age (years), Mean (SD)		53.1 (7.8)	55.1 (7.8)	51.5 (7.5)	0.044*
Gender	Male	47 (62.7)	22 (64.7)	25 (61.0)	0.926
	Female	28 (37.3)	12 (35.3)	16 (39.0)	
Duration of cirrhosis (years), Mean (SD)		4.3 (2.3)	4.9 (2.6)	3.8 (2.0)	0.049*
Etiology of cirrhosis	Alcohol-related	32 (42.7)	15 (44.1)	17 (41.5)	0.945
	MASLD/NASH	22 (29.3)	10 (29.4)	12 (29.3)	
	Viral hepatitis	11 (14.7)	5 (14.7)	6 (14.6)	
	Autoimmune	4 (5.3)	1 (2.9)	3 (7.3)	
	Cryptogenic/others	6 (8.0)	3 (8.8)	3 (7.3)	
Child-Pugh class	A	20 (26.7)	5 (14.7)	15 (36.6)	0.054
	B	36 (48.0)	17 (50.0)	19 (46.3)	
	C	19 (25.3)	12 (35.3)	7 (17.1)	
MELD-Na score, Mean (SD)		14.5 (4.3)	16.8 (4.9)	12.6 (2.6)	<0.001*
Fatigue		47 (62.7)	28 (82.4)	19 (46.3)	0.002*
Abdominal distension/ascites symptoms		35 (46.7)	22 (64.7)	13 (31.7)	0.006*
Pedal edema symptoms		26 (34.7)	16 (47.1)	10 (24.4)	0.053
Jaundice symptoms		37 (49.3)	19 (55.9)	18 (43.9)	0.357
History of gastrointestinal bleeding		10 (13.3)	6 (17.6)	4 (9.8)	0.497
Diabetes mellitus		16 (21.3)	9 (26.5)	7 (17.1)	0.400
Hypertension		18 (24.0)	7 (20.6)	11 (26.8)	0.595
Dyslipidemia		19 (25.3)	10 (29.4)	9 (22.0)	0.595
Ischemic heart disease		1 (1.3)	0 (0.0)	1 (2.4)	1.000
Heart failure		0 (0.0)	0 (0.0)	0 (0.0)	-
Chronic kidney disease (not ESRD)		5 (6.7)	2 (5.9)	3 (7.3)	1.000
Thyroid disease		8 (10.7)	5 (14.7)	3 (7.3)	0.456
Autoimmune disease		2 (2.7)	2 (5.9)	0 (0.0)	0.202
Reactive airway disease		8 (10.7)	6 (17.6)	2 (4.9)	0.130
Active/previous malignancy		0 (0.0)	0 (0.0)	0 (0.0)	-
Cerebrovascular disease/stroke		0 (0.0)	0 (0.0)	0 (0.0)	-

Table 2: Clinical examination, anthropometry, and functional muscle assessment

	Overall (N=75)	CT-SO present (n=34)	CT-SO absent (n=41)	P value
Pulse rate (beats/min)	85.0 (10.3)	87.2 (11.0)	83.2 (9.4)	0.100
Systolic blood pressure (mmHg)	119.1 (11.6)	116.4 (8.7)	121.4 (13.2)	0.052
Diastolic blood pressure (mmHg)	73.0 (9.5)	72.8 (9.2)	73.1 (9.8)	0.904
SpO ₂ (%)	97.6 (1.2)	97.2 (1.2)	98.0 (1.0)	0.005*
Height (cm)	163.8 (7.6)	163.6 (7.3)	164.0 (7.8)	0.827
Dry weight (kg)	80.7 (9.6)	79.3 (10.5)	81.9 (8.8)	0.237
BMI (kg/m ²)	30.1 (2.6)	29.5 (2.3)	30.5 (2.8)	0.096
Mid-arm circumference (cm)	26.7 (2.9)	25.1 (2.0)	28.1 (2.9)	<0.001*
Triceps skin-fold thickness (mm)	20.9 (4.8)	19.9 (4.7)	21.6 (4.8)	0.133
Hand-grip strength (kg)	24.0 (6.3)	21.0 (5.5)	26.5 (5.9)	<0.001*
Gait speed (m/s)	0.81 (0.17)	0.71 (0.11)	0.90 (0.16)	<0.001*
Pallor	22 (29.3)	14 (41.2)	8 (19.5)	0.046*
Icterus	30 (40.0)	17 (50.0)	13 (31.7)	0.155
Pedal edema on examination	28 (37.3)	19 (55.9)	9 (22.0)	0.004*
Raised jugular venous pressure	4 (5.3)	2 (5.9)	2 (4.9)	1.000
Low hand-grip strength	29 (38.7)	24 (70.6)	5 (12.2)	<0.001*
Reduced gait speed (<0.8 m/s)	36 (48.0)	28 (82.4)	8 (19.5)	<0.001*

Table 3: Laboratory investigations

	Overall (N=75)	CT-SO present (n=34)	CT-SO absent (n=41)	P value
Hemoglobin (g/dL)	11.3 (1.5)	11.1 (1.5)	11.5 (1.4)	0.181
Total leukocyte count (/mm ³ ×10 ³)	8.1 (2.6)	9.2 (2.5)	7.1 (2.2)	<0.001*
Neutrophils/polymorphs (%)	64.8 (9.0)	67.1 (10.0)	63.0 (7.7)	0.052
Lymphocytes (%)	26.2 (9.3)	23.8 (10.5)	28.1 (7.8)	0.052
Monocytes (%)	6.0 (1.5)	5.7 (1.6)	6.4 (1.4)	0.048*
Eosinophils (%)	2.7 (1.1)	2.8 (1.0)	2.6 (1.2)	0.473
Platelet count (/mm ³ ×10 ³)	115.5 (41.7)	105.1 (33.4)	124.2 (46.1)	0.041*
ESR (mm/hr)	31.3 (14.4)	37.5 (14.9)	26.1 (11.8)	<0.001*
MCV (fL)	86.3 (7.9)	86.2 (9.0)	86.4 (6.9)	0.889
Urea (mg/dL)	36.2 (13.6)	42.0 (14.7)	31.4 (10.7)	<0.001*
Creatinine (mg/dL)	0.99 (0.26)	1.05 (0.28)	0.94 (0.22)	0.067
Sodium (mEq/L)	135.6 (3.7)	134.2 (3.5)	136.7 (3.5)	0.002*
Potassium (mEq/L)	4.12 (0.42)	4.17 (0.39)	4.09 (0.45)	0.428
Magnesium (mEq/L)	1.88 (0.24)	1.87 (0.24)	1.89 (0.24)	0.749
Total bilirubin (mg/dL)	2.53 (1.37)	2.78 (1.49)	2.33 (1.25)	0.160
Direct bilirubin (mg/dL)	1.20 (0.71)	1.39 (0.83)	1.05 (0.57)	0.045*
Indirect bilirubin (mg/dL)	1.33 (0.75)	1.39 (0.74)	1.28 (0.76)	0.516
SGOT/AST (U/L)	65.3 (29.5)	75.9 (35.9)	56.4 (19.3)	0.006*
SGPT/ALT (U/L)	53.2 (21.6)	56.4 (22.0)	50.6 (21.2)	0.255
Alkaline phosphatase (U/L)	159.0 (58.5)	169.2 (63.2)	150.6 (53.5)	0.179
Total protein (g/dL)	6.54 (0.82)	6.11 (0.80)	6.89 (0.66)	<0.001*
Albumin (g/dL)	3.10 (0.52)	2.84 (0.46)	3.31 (0.47)	<0.001*
Globulin (g/dL)	3.44 (0.85)	3.27 (0.92)	3.57 (0.76)	0.137
PT/INR	1.48 (0.30)	1.64 (0.27)	1.34 (0.26)	<0.001*
CRP (mg/L)	19.7 (10.9)	27.4 (11.4)	13.4 (4.4)	<0.001*
HbA1c (%)	6.0 (1.1)	6.0 (1.1)	6.1 (1.2)	0.937
TSH (mIU/L)	3.07 (1.49)	2.99 (1.36)	3.14 (1.61)	0.657
AFP (ng/mL)	5.5 (3.0)	6.2 (3.2)	4.9 (2.9)	0.086

Table 4: CT-derived skeletal muscle index and ultrasound-derived muscle measurements

	Overall (N=75)	CT-SO present (n=34)	CT-SO absent (n=41)	P value	Correlation with CT-SMI
CT-derived skeletal muscle index (cm ² /m ²)	34.72 (4.60)	31.41 (2.86)	37.47 (3.92)	<0.001*	Reference
Quadriceps thickness, feather-weight reading (cm)	1.53 (0.37)	1.22 (0.21)	1.79 (0.25)	<0.001*	r=0.835; <0.001*
Quadriceps thickness, compression reading (cm)	1.26 (0.30)	1.01 (0.19)	1.47 (0.20)	<0.001*	r=0.818; <0.001*
Mean quadriceps muscle thickness (cm)	1.40 (0.33)	1.12 (0.20)	1.63 (0.22)	<0.001*	r=0.830; <0.001*
Forearm muscle thickness (cm)	1.19 (0.21)	1.06 (0.13)	1.31 (0.19)	<0.001*	r=0.724; <0.001*

Table 5: Diagnostic accuracy of ultrasound-derived muscle thickness for CT-defined sarcopenic obesity

Ultrasound parameter	Optimal cut-off (cm)	AUC (95% CI)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)	P value
Mean quadriceps muscle thickness	≤1.41	0.956 (0.909-0.990)	97.1	82.9	82.5	97.1	89.3	<0.001*
Quadriceps feather-weight thickness	≤1.52	0.955 (0.907-0.991)	97.1	82.9	82.5	97.1	89.3	<0.001*
Quadriceps compression thickness	≤1.21	0.960 (0.916-0.991)	91.2	90.2	88.6	92.5	90.7	<0.001*
Forearm muscle thickness	≤1.23	0.847 (0.752-0.925)	91.2	65.9	68.9	90.0	77.3	<0.001*

Table 6: Correlation of CRP with muscle and functional parameters

	Pearson r	P value
CT-derived skeletal muscle index (cm ² /m ²)	-0.655	<0.001*
Mean quadriceps muscle thickness (cm)	-0.665	<0.001*
Forearm muscle thickness (cm)	-0.512	<0.001*
Hand-grip strength (kg)	-0.461	<0.001*
Gait speed (m/s)	-0.253	0.028*
Serum albumin (g/dL)	-0.290	0.012*

Table 7: Multivariable linear regression with CT-derived skeletal muscle index as the dependent variable

Predictor	Regression coefficient (β)	95% CI	P value
CRP per 10 mg/L	-1.72	-2.36 to -1.09	<0.001*
Age per 10 years	-0.69	-1.44 to 0.06	0.072
Male sex	5.04	3.77 to 6.31	<0.001*
BMI (kg/m ²)	0.24	0.02 to 0.47	0.036*
MELD-Na score	-0.22	-0.37 to -0.06	0.006*

Regression coefficients represent the adjusted change in CT-SMI (cm²/m²) for each predictor

Discussion

The present study demonstrated that CT-SO was frequent among patients with cirrhosis and obesity, being identified in 34 of 75 participants (45.3%). This burden is clinically important because sarcopenia in cirrhosis represents more than reduced muscle bulk; it reflects altered protein metabolism, chronic inflammation, impaired ammonia detoxification, reduced physical reserve,

and progressive hepatic decompensation. [9] The finding that patients with CT-SO were older than those without CT-SO (55.1 ± 7.8 vs. 51.5 ± 7.5 years, p=0.044) was biologically plausible, as ageing is associated with anabolic resistance, reduced motor-unit recruitment, mitochondrial dysfunction, and progressive type II muscle fibre loss.[10] In cirrhosis, these age-related changes are amplified by hyperammonaemia, systemic

inflammation, reduced oral intake, altered branched-chain amino acid metabolism, insulin resistance, and reduced physical activity, which together accelerate skeletal muscle wasting. EASL guidelines also recognise sarcopenia as a major component of malnutrition in chronic liver disease and note that direct quantification of skeletal muscle requires cross-sectional imaging, with CT analysis at the L3 vertebral level being a widely accepted method for quantifying muscle loss.[11]

The association of CT-SO with longer duration of cirrhosis (4.9 ± 2.6 vs. 3.8 ± 2.0 years, $p=0.049$) and higher MELD-Na score (16.8 ± 4.9 vs. 12.6 ± 2.6 , $p<0.001$) supports the concept that sarcopenic obesity is linked to disease progression rather than body mass alone. [12] In the present study, BMI did not differ significantly between groups, whereas CT-SMI, muscle strength, gait speed, albumin, CRP, sodium, INR, and MELD-Na did, indicating that conventional anthropometry alone was insufficient to identify clinically relevant muscle depletion in obese cirrhotic patients. This is particularly important in cirrhosis, where ascites and oedema can mask tissue loss and falsely preserve body weight. Lai et al. emphasised in the AASLD Practice Guidance that malnutrition, frailty, and sarcopenia are common but under-recognised in cirrhosis and should be assessed using objective measures rather than clinical impression alone. [13] The symptom and examination profile further supported the clinical relevance of CT-SO. Fatigue was reported in 82.4% of patients with CT-SO compared with 46.3% without CT-SO ($p=0.002$), and abdominal distension or ascites-related symptoms were also more frequent in the CT-SO group (64.7% vs. 31.7%, $p=0.006$). Pedal oedema on examination was observed in 55.9% of CT-SO patients compared with 22.0% of non-CT-SO patients ($p=0.004$). These findings are consistent with the interaction between worsening portal hypertension, fluid retention, reduced mobility, and muscle catabolism. The higher proportion of Child-Pugh class C disease among patients with CT-SO (35.3% vs. 17.1%) approached statistical significance and was concordant with the markedly higher MELD-Na score, suggesting that the sarcopenic-obese phenotype clustered with more advanced liver dysfunction. [14,15]

Functional muscle impairment was prominent in patients with CT-SO. Mean hand-grip strength was significantly lower in the CT-SO group (21.0 ± 5.5 vs. 26.5 ± 5.9 kg, $p<0.001$), and low hand-grip strength was present in 70.6% compared with 12.2% of patients without CT-SO ($p<0.001$). Gait speed showed an even stronger difference, with CT-SO patients walking more slowly than non-CT-SO patients (0.71 ± 0.11 vs. 0.90 ± 0.16 m/s, $p<0.001$), and reduced gait speed was present in

82.4% versus 19.5% ($p<0.001$). These results indicate that CT-defined muscle depletion translated into measurable functional compromise. This is relevant because EWGSOP2 placed strong emphasis on muscle strength as a key marker of sarcopenia and advised a gait-speed cut-off of ≤ 0.8 m/s as an indicator of poor physical performance or severe sarcopenia. [16] In the cirrhosis setting, reduced muscle function also has implications for hepatic encephalopathy risk, hospitalization, transplant wait-list outcomes, and survival. [17]

The laboratory profile suggested that CT-SO was associated with both hepatic synthetic dysfunction and systemic inflammation. Patients with CT-SO had lower albumin (2.84 ± 0.46 vs. 3.31 ± 0.47 g/dL, $p<0.001$), lower total protein, higher PT/INR (1.64 ± 0.27 vs. 1.34 ± 0.26 , $p<0.001$), higher AST, lower sodium, and higher urea. The markedly higher CRP level in the CT-SO group (27.4 ± 11.4 vs. 13.4 ± 4.4 mg/L, $p<0.001$) was particularly important because the study objective included evaluation of systemic inflammation in relation to muscle mass. CRP showed significant inverse correlations with CT-SMI ($r=-0.655$, $p<0.001$), mean quadriceps thickness ($r=-0.665$, $p<0.001$), forearm muscle thickness ($r=-0.512$, $p<0.001$), hand-grip strength ($r=-0.461$, $p<0.001$), gait speed ($r=-0.253$, $p=0.028$), and albumin ($r=-0.290$, $p=0.012$). These findings support an inflammatory sarcopenia phenotype in which higher inflammatory burden is associated not only with reduced CT-measured muscle mass but also with impaired muscle quality and physical performance. Lin et al. reported an inverse association between CRP concentrations and sarcopenia and proposed CRP as a potential biomarker of sarcopenia, [18] while van Ancum et al. found that higher CRP and lower albumin were associated with longitudinal decline in skeletal muscle mass index, hand-grip strength, and gait speed. [19]

The strongest technical finding of the present study was the close agreement between ultrasound-derived muscle thickness and CT-SMI. CT-SMI was substantially lower in patients with CT-SO than in those without CT-SO (31.41 ± 2.86 vs. 37.47 ± 3.92 cm²/m², $p<0.001$). In parallel, mean quadriceps thickness was lower by approximately 0.51 cm (1.12 ± 0.20 vs. 1.63 ± 0.22 cm, $p<0.001$), and forearm muscle thickness was lower by 0.25 cm (1.06 ± 0.13 vs. 1.31 ± 0.19 cm, $p<0.001$). The correlation with CT-SMI was strongest for quadriceps feather-weight thickness ($r=0.835$), mean quadriceps thickness ($r=0.830$), and quadriceps compression thickness ($r=0.818$), while forearm thickness also showed a strong positive correlation ($r=0.724$). This pattern suggests that lower-limb ultrasound may better reflect whole-body skeletal muscle reserve than forearm measurement in this population, possibly because

quadriceps muscle is more sensitive to inactivity, frailty, and protein-energy wasting. The diagnostic accuracy findings further support muscle ultrasound as a practical surrogate for CT-SMI in cirrhotic patients with obesity. Quadriceps compression thickness achieved the highest AUC of 0.960, with a cut-off of ≤ 1.21 cm, sensitivity of 91.2%, specificity of 90.2%, and overall accuracy of 90.7%. Mean quadriceps thickness and quadriceps feather-weight thickness had similarly excellent AUC values of 0.956 and 0.955, respectively. Forearm muscle thickness showed good but comparatively lower diagnostic performance (AUC=0.847). These results are closely aligned with the AMUSE Study by Dhariwal et al., in which 52 patients with cirrhosis and obesity underwent CT-SMI and ultrasound assessment; quadriceps muscle thickness showed very high diagnostic accuracy for CT-defined sarcopenia, with reported AUC values around 0.98 for quadriceps muscle thickness and 0.85 for forearm muscle thickness. Dhariwal et al. also reported significant positive correlations between ultrasound indices and CT-SMI, supporting the use of ultrasound as a point-of-care method for sarcopenia assessment in cirrhosis with obesity. [8]

The multivariable regression analysis strengthened the interpretation that inflammation was independently related to lower muscle mass. After adjustment for age, sex, BMI, and MELD-Na score, each 10 mg/L rise in CRP was associated with a 1.72 cm²/m² reduction in CT-SMI (95% CI: -2.36 to -1.09, $p < 0.001$). MELD-Na was also independently associated with lower CT-SMI ($\beta = -0.22$, $p = 0.006$), indicating that hepatic disease severity and inflammation contributed additively to muscle depletion.

Male sex remained strongly associated with higher CT-SMI ($\beta = 5.04$, $p < 0.001$), reflecting expected sex-related differences in skeletal muscle mass, while BMI showed a modest positive association with CT-SMI ($\beta = 0.24$, $p = 0.036$). The absence of an independent age effect after adjustment ($p = 0.072$) suggests that, in this cohort, inflammatory activity and liver disease severity were more proximate determinants of muscle depletion than chronological age alone.

The present study had certain limitations. First, it was a single-centre, hospital-based study with a relatively small sample size of 75 patients, which may have limited the generalisability of the findings to other populations and healthcare settings. Second, the cross-sectional design allowed assessment of associations between CT-derived skeletal muscle index, ultrasound-derived muscle measurements, functional parameters, and inflammatory markers, but did not permit evaluation of temporal or causal relationships.

Third, although CT-derived skeletal muscle index was used as the reference standard for muscle mass assessment, the study did not include longitudinal clinical outcomes such as hospitalization, hepatic decompensation, quality of life, transplant-related outcomes, or mortality. Fourth, muscle ultrasound measurements were potentially operator-dependent, and inter-observer or intra-observer variability was not assessed in detail. Fifth, systemic inflammation was evaluated using CRP measured at a single time point, which may not have fully reflected chronic inflammatory status or fluctuations related to intercurrent infection or recent decompensation.

Conclusion

The present study showed that CT-defined sarcopenic obesity was common among patients with cirrhosis and was associated with older age, longer duration of cirrhosis, higher MELD-Na score, greater symptom burden, poorer functional muscle status, lower albumin levels, and higher systemic inflammatory activity.

Patients with CT-defined sarcopenic obesity had significantly reduced CT-derived skeletal muscle index, lower quadriceps and forearm muscle thickness on ultrasound, reduced hand-grip strength, and slower gait speed. Ultrasound-derived muscle measurements, particularly quadriceps muscle thickness, showed strong positive correlations with CT-derived skeletal muscle index and demonstrated excellent diagnostic accuracy for identifying CT-defined sarcopenic obesity. Quadriceps compression thickness provided the highest diagnostic performance, suggesting that bedside muscle ultrasound may serve as a practical, non-invasive, and reliable tool for screening and assessment of sarcopenic obesity in patients with cirrhosis.

The study also demonstrated a significant inverse relationship between CRP and muscle mass, muscle thickness, hand-grip strength, gait speed, and serum albumin, indicating that systemic inflammation was closely associated with muscle depletion and functional impairment.

Overall, the findings supported the use of muscle ultrasound as a clinically useful surrogate for CT-based muscle assessment and highlighted the role of inflammation in the pathophysiology of sarcopenic obesity among cirrhotic patients.

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