ORIGINAL ARTICLE

Hand Grip Strength as a Functional Marker of Sarcopenia in Liver Cirrhosis: Evidence from an Indian Cohort

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Abstract

Background: Sarcopenia is a frequent and prognostically significant complication of liver cirrhosis. Hand grip strength (HGS) has emerged as a simple, noninvasive tool for assessing muscle function, yet limited data exist on its utility in Indian cirrhotic populations.

Aim: To evaluate the association of HGS with established prognostic scores and biochemical parameters in Indian patients with cirrhosis.

Materials and methods: In this cross-sectional observational study, 100 adult cirrhotic patients were assessed between August 2022 and December 2023. HGS was measured using a validated hand-held dynamometer. Correlations between HGS and clinical scores of severity of cirrhosis [Child–Turcotte–Pugh (CTP), Model for End-Stage Liver Disease (MELD)] and biochemical markers were analyzed using appropriate statistical methods.

Results: Mean patient age was 59.2 ± 8.46 years; 85% were male. The most common etiologies were alcohol (46%) and viral hepatitis (26%). HGS declined significantly with increasing liver disease severity: CTP A (34.0 ± 1.48 kg), B (21.63 ± 1.07 kg), and C (13.5 ± 2.87 kg) ($p < 0.0001$). HGS was inversely correlated with MELD score ($r = -0.820$) and showed strong positive correlations with serum albumin ($r = +0.872$) and hemoglobin ($r = +0.59$). Age, international normalized ratio (INR), and bilirubin were negatively correlated with HGS.

Conclusion: HGS is strongly associated with liver disease severity and key biochemical indicators. As a bedside, radiation-free tool, it offers a practical method for assessing sarcopenia in cirrhosis, especially in resource-limited settings.

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Introduction

Cirrhosis signifies the end stage of chronic liver disease, characterized by progressive hepatocellular dysfunction, portal hypertension, and multisystem complications impacting patient morbidity and mortality.[1] The global burden of cirrhosis continues to escalate, with an estimated 1.16 million deaths annually attributable to cirrhosis and its complications, making it the 11th leading cause of death worldwide.[2] In India, the prevalence of chronic liver disease has increased substantially over the past 2 decades. As per 2021 World Health Organization data, India accounts for the highest number of deaths attributable to cirrhosis globally.[3]

Sarcopenia in Cirrhosis

Sarcopenia, defined as progressive loss of skeletal muscle mass and function, is one of the most clinically significant complications of cirrhosis.[4] Its prevalence in cirrhosis ranges from 20 to 70%, depending on the diagnostic criteria employed and disease severity.[5,6] This wide variation in prevalence estimates underscores the challenges in establishing standardized diagnostic approaches for sarcopenia assessment in clinical practice.

The pathophysiology of sarcopenia involves complex interactions between altered protein metabolism, chronic inflammation, hormonal imbalances, and nutritional deficiencies.[7] Hepatic dysfunction leads to impaired albumin synthesis, altered amino acid metabolism, and increased protein catabolism, resulting in progressive muscle wasting. Additionally, portal-systemic shunting causes hyperammonemia, which impairs muscle protein synthesis and promotes muscle proteolysis *via* the ubiquitin-proteasome pathway.[8] Multiple studies have demonstrated that sarcopenia independently predicts mortality, increases the risk of hepatic encephalopathy, prolongs hospital stay, and adversely affects posttransplant outcomes.[9]

Traditional methods for assessing sarcopenia include bioelectrical impedance analysis, dual-energy X-ray absorptiometry, and cross-sectional imaging techniques such as computed tomography (CT) or magnetic resonance imaging.[10] While these modalities provide accurate measurements of muscle mass and composition, their widespread implementation is limited by cost, availability, technical expertise requirements, and radiation exposure concerns, particularly in resource-constrained settings.

Hand Grip Strength: An Emerging Tool

Hand grip strength (HGS) has emerged as a simple, reproducible, and clinically meaningful measure of muscle function that correlates strongly with overall muscle strength and physical performance.[11] The simplicity of HGS measurement using handheld dynamometry, combined with its strong prognostic value, positions it as an attractive option for routine clinical evaluation. Several studies have explored the relationship between HGS and cirrhosis. Tandon et al. demonstrated that reduced HGS independently predicted mortality in cirrhotic patients awaiting liver transplantation, with comparable prognostic accuracy to established scoring systems.[12] Similarly, Sinclair and colleagues showed that HGS provided additional prognostic value beyond the Model for End-Stage Liver Disease (MELD) score in male cirrhotics.[13]

Study Rationale and Objectives

Despite growing evidence supporting HGS as a valuable assessment tool, limited data exist on its application in Indian cirrhotic patients. This study was designed to evaluate the feasibility of HGS testing in routine clinical practice in a cohort of Indian cirrhotic patients of varied etiology. Our primary objective was to determine the correlation between HGS and established prognostic scores of cirrhosis. Secondary objectives included assessment of the relationship between HGS and individual biochemical parameters.

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Materials and Methods

This cross-sectional observational study included patients aged ≥18 years diagnosed with cirrhosis attending the Gastroenterology and Medicine departments (inpatient and outpatient) between August 2022 and December 2023. Cirrhosis was diagnosed using history and clinical examination, biochemical, endoscopic, imaging, and elastography parameters. Decompensation encompassed ascites, variceal bleed, and hepatic encephalopathy. Patients with hepatocellular carcinoma, other organ malignancy, severe extrahepatic disorders, and connective tissue disorders were excluded, as well as patients with inability to perform grip strength testing due to hand or arm pathology.

Demographics, history, and clinical findings were recorded. Body mass index (BMI) was calculated using height and weight adjusted for ascites and pedal edema.[12] Baseline hemogram, liver and renal function tests, and workup for cirrhosis etiology were done. Child–Turcotte–Pugh (CTP) and MELD scores were calculated.

HGS was assessed using a digital hand-grip dynamometer Camry®: Trailite Hand-Dynamometer LSC100 (Zhongshan Camry Electronic Co. Ltd., Zhongshan, China). It has been previously validated in multiple populations, including Indians.[14] Patients were seated with shoulders adducted and in neutral position, elbows flexed at 90°, forearms neutral, and wrists dorsiflexed between 0 and 30°. Three readings were taken on the dominant hand, and the mean value was used for analysis.

Data Analysis

All statistical analyses were done with Statistical Package for the Social Sciences (SPSS) v25 (IBM, Armonk, New York, United States of America). Descriptive statistics were presented as means and standard deviations for continuous variables and frequencies with percentages for categorical variables. Comparisons were made using *t*-tests or Mann–Whitney *U* tests for continuous variables. Chi-squared or Fisher's exact test was used for categorical variables. Multiple regression assessed associations between HGS and various parameters. Significance was set at $p < 0.05$.

Results

Demographic and Clinical Characteristics

A total of 100 patients with liver cirrhosis were recruited during the study period. The mean age was 59.2 ± 8.46 years, with 85% males. The most common etiologies were alcohol (46%) and viral hepatitis (26%). Most patients presented with abdominal distension and had advanced liver disease, with 79% classified as CTP B or C (Table 1). The mean MELD score was 21.81 ± 6.73, which showed strong correlation with CTP score (Table 2 and Fig. 1).

Hand Grip Strength across Disease Severity

There was a significant decline in HGS with increasing severity of cirrhosis, as determined by both CTP class and MELD score.

Table 2: MELD score statistics by CTP class

CTP class (n)	*MELD (mean ± SD)*
CTP A (21)	13.49 ± 2.06
CTP B (19)	17.59 ± 1.98
CTP C (60)	26.06 ± 4.97

Table 1: Baseline characteristics of study participants

Characteristic	*Subgroup*	*Number (n)*	*Percentage (%)*
Gender	Male	85	85
	Female	15	15
Age-group	40–49 years	16	16
	50–59 years	32	32
	60–70 years	52	52
CTP class	CTP-A	21	21
	CTP-B	19	19
	CTP-C	60	60
Etiology	Alcohol	46	46
	Hepatitis B	20	20
	MAFLD	15	15
	Hepatitis C	6	6
	Auto-immune	6	6
	Idiopathic	6	6
	Wilson disease	1	1

Child–Turcotte–Pugh Class Comparison

Mean HGS in CTP A, B, and C were 34.0 ± 1.48 kg, 21.63 ± 1.07 kg, and 13.5 ± 2.87 kg, respectively (Table 3 and Fig. 2). Pairwise comparisons using Mann–Whitney *U* tests with Bonferroni correction showed statistically significant differences between all groups ($p < 0.0001$). Cohen's *d* effect sizes were very large: A vs B = 9.50, A vs C = 7.92, B vs C = 3.17.

Model for End-Stage Liver Disease Score Comparison

Patients with MELD >25 had significantly lower HGS (15.14 ± 7.03 kg) than those with MELD ≤15 (33.35 ± 2.98 kg), with a clear inverse trend (Table 4 and Fig. 3). Patients with low HGS are clustered in the CTP-C, high MELD quadrant (Fig. 4).

Biochemical and Anthropometric Correlations

Pearson correlation analysis demonstrated a strong positive association between HGS and serum albumin and hemoglobin. Moderate positive correlations were observed with serum sodium and platelet count. Significant negative correlations were noted with CTP class, MELD score, and age (Table 5).

Table 3: Hand grip strength across CTP classes

HGS	*Mean ± SD (kg)*	*Range (kg)*
CTP A	34 ± 1.48	30–36
CTP B	21.63 ± 1.07	20–24
CTP C	13.5 ± 2.87	10–20

Table 4: HGS across MELD categories

MELD (n = 100)	*Mean ± SD*
>25 (35)	15.14 ± 7.03
16–25 (48)	17.46 ± 5.18
≤15 (17)	33.35 ± 2.98

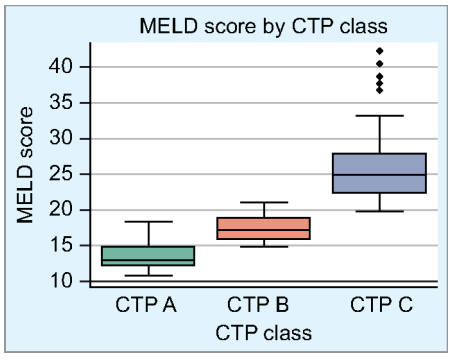

Fig. 1: Boxplot showing distribution of MELD scores across CTP classes

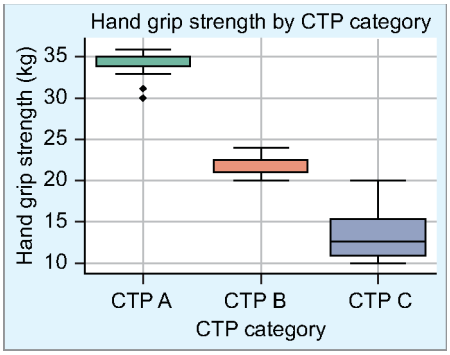

Fig. 2: Boxplot showing decline in HGS across CTP classes

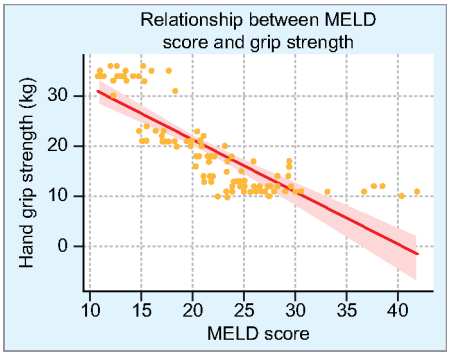

Fig. 3: Scatter plot visualizing inverse relationship between MELD and HGS

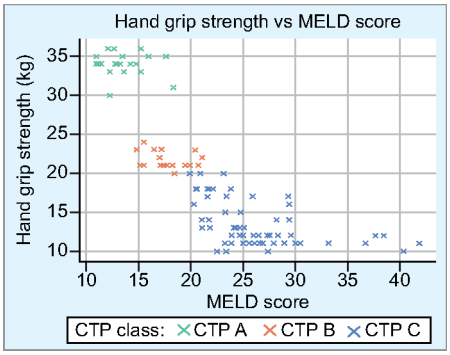

Fig. 4: Clustering of low HGS with CTP-C and high MELD

Discussion

The European Working Group on Sarcopenia in Older People-2 and the recently released South Asian Working Action Group on Sarcopenia (SWAG-SARCO) guidelines emphasize the need for both a functional and quantitative assessment for defining sarcopenia.[15,16] Sarcopenia diagnosis should follow a hierarchical model: probable sarcopenia is identified by reduced muscle strength, confirmed by demonstrating reduced muscle mass, and considered severe when accompanied by impaired physical performance. The SWAG-SARCO consensus reinforces the importance of muscle strength testing as the initial step in sarcopenia assessment, especially in low-resource settings.

Among the various methods to detect sarcopenia, the current gold standard is CT-based skeletal muscle index (SMI). Various society guidelines have advocated using HGS for measurement of muscle strength.[15–18] In a recent study, HGS was shown to be a better predictor of mortality in cirrhotic patients compared to SMI.[13] Since HGS measures muscle function directly, it possibly is a better indicator of physical activity and functional status than markers of muscle mass.

Considering the European and Indian National Association for Study of the Liver suggested cutoffs for HGS (<27 kg for men, <16 kg for women), prevalence of sarcopenia in our cohort would be 79% for males and 40% for females.[17,18] None of the patients in CTP A had HGS <27 kg. Taking the cutoff of ≤31 kg, as suggested by a recent study in Indian male patients, prevalence would be similar (81%).[19] The earlier reported incidence in Indian patients varies from 20 to 80%[19,20] and 50 to 70% in other Asian countries.[21] Most of these studies have used CT-based SMI for assessment of sarcopenia. Moreover, the higher prevalence in our cohort is attributable to more advanced liver disease in the cohort (79% CTP B and C).

The decrease in HGS as we move toward more advanced liver disease was highly significant between the CTP and MELD groups. The mean HGS in patients of CTP C and MELD >25 was less than half of those of CTP A and MELD ≤15, respectively. The substantial effect sizes observed (Cohen's d >0.8) indicate clinically meaningful differences beyond statistical significance. The exceptionally strong correlation between HGS and CTP score suggests that HGS can serve as an accurate predictor of functional liver reserve and disease prognosis. To the best of our knowledge, ours is the first study to demonstrate progressively declining HGS with increasing severity of liver disease.

Analyzing the factors influencing HGS, a particularly strong positive correlation was observed between HGS and serum albumin, indicating that hepatic synthetic capacity directly impacts muscle function. Albumin is not only a marker of liver synthetic function but also reflects systemic protein availability, a crucial component in muscle integrity. Other markers such as hemoglobin, serum sodium, and platelet count also showed significant positive correlations, suggesting that reduced HGS mirrors systemic derangements associated with advanced liver disease. However, unlike these biochemical markers, which may be influenced by acute fluctuations or laboratory variability, HGS provides a direct measure of muscle functional capacity that reflects the cumulative impact of chronic liver disease on patient wellbeing. Notably, etiology of cirrhosis did not influence HGS, suggesting that severity of liver dysfunction, rather than its cause, is the major determinant of muscle dysfunction in cirrhosis.

The age-related decline in HGS observed in our study highlights the compounding effects of chronological aging and cirrhotic

Table 5: Correlation between HGS and other parameters

Parameter	*Pearson correlation*	*95% confidence interval*	*p-value (2-tailed)*
CTP	−0.951	−0.969 to −0.924	<0.0001
MELD	−0.820	−0.877 to −0.747	<0.0001
INR	−0.818	−0.875 to −0.745	<0.0001
Albumin (gm/dL)	+0.872	+0.818 to +0.913	<0.0001
Age (years)	−0.777	−0.845 to −0.693	<0.0001
Bilirubin (mg/dL)	−0.674	−0.764 to −0.562	<0.0001
Hemoglobin (gm/dL)	+0.590	+0.442 to +0.709	<0.001
Sodium (mmol/L)	+0.370	+0.185 to +0.537	0.0100
Platelet count ($\times 10^9$/L)	+0.340	+0.152 to +0.511	0.0200
Weight (kg)	+0.225	+0.029 to +0.409	0.0242
Height (m)	+0.196	−0.001 to +0.384	0.0511
Creatinine (mg/dL)	−0.161	−0.349 to +0.038	0.1090
BMI (kg/m^2)	+0.115	−0.084 to +0.307	0.2544

muscle wasting. This is particularly relevant for elderly cirrhotic patients, who may experience accelerated functional decline and hence require more intensive monitoring and early intervention.

Our study is limited by its cross-sectional design, which precludes assessment of causality or serial changes in muscle function. The majority of patients were male and from a single center, which may limit generalizability. Additionally, incorporating comparative measures of sarcopenia assessment like SMI would have provided a more comprehensive picture of muscle function. While reduced HGS is a key indicator of probable sarcopenia, definitive diagnosis requires confirmation of reduced muscle mass per current consensus guidelines (e.g., EWGSOP2, SWAG-SARCO). This study focused on the functional aspect of sarcopenia through HGS due to feasibility and resource constraints.

In conclusion, the noninvasive nature, ease of use, and strong correlation of HGS with critical clinical parameters position it as a valuable adjunct in the comprehensive assessment of patients with liver cirrhosis. Hand-held dynamometers are readily available, require minimal training for proper use, and provide immediate results. It is especially relevant for resource-limited settings where advanced imaging modalities may not be readily available. As we continue to recognize the importance of sarcopenia in liver disease outcomes, HGS assessment offers a practical approach to identifying and monitoring this critical complication, ultimately supporting improved clinical outcomes.

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