

# Correlation between Frailty and Nutrition, Psychological, and Physical Function in Patients with Decompensated Cirrhosis: A Cross-Sectional Study Based on the Liver Frailty Index

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## Abstract

**Objective:** This study investigates the relationship between frailty (assessed via the Liver Frailty Index), nutrition, negative emotions (depression and anxiety), and physical function in decompensated cirrhosis patients, aiming to provide scientific evidence for clinical treatment strategies. **Methods:** A cross-sectional study was conducted involving 477 decompensated cirrhosis patients. Data were collected using general information questionnaires, the 2002 Nutrition Risk Screening Scale, the Self-Rating Anxiety Scale, the Self-Rating Depression Scale, the Daily Living Activity Rating Scale, the Morse Fall Risk Assessment Scale, and the Liver Frailty Index. A binary logistic regression model was employed to analyze the associations between frailty and nutritional status, negative emotions, and physical function. **Results:** The prevalence of frailty in cirrhosis patients was 21.38%. Univariate analysis revealed that patients with frailty showed significantly worse outcomes in age, gender, nutritional indicators (body mass index, potassium ion, prealbumin, albumin, hemoglobin), fall risk, activity capacity, nutritional risk, depression and anxiety scores, as well as complications (ascites, bleeding) compared to those without frailty. The binary logistic regression analysis further identified depression scores (OR = 1.03) and hemoglobin levels (OR = 0.98) as independent factors contributing to increased frailty. Higher depression scores were associated with significantly elevated risks of frailty. The lower the hemoglobin level, the greater the patient's frailty index. Additionally, aging, elevated Morse fall risk score, and decreased ADL (Activities of Daily Living) scores have been confirmed as independent correlates of frailty. **Conclusion:** Frailty in decompensated cirrhosis patients is significantly associated with ad-

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vanced age, depressive mood, nutritional anemia, high fall risk, and reduced baseline mobility. Therefore, clinical interventions should extend beyond conventional liver disease management by integrating psychological support (particularly for depression), targeted nutritional interventions (to correct anemia), fall prevention strategies, and functional rehabilitation exercises. This multidimensional approach aims to delay or reverse frailty progression and improve overall patient outcomes.

## Keywords

Decompensated Cirrhosis, Frailty, Nutrition, Psychological, Somatic Function

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## 1. Introduction

As a chronic progressive liver disease, liver cirrhosis in the decompensated stage is a critical phase of the disease, which is often accompanied by life-threatening complications such as ascites, gastrointestinal bleeding, and hepatic encephalopathy [1] [2]. In recent years, the concept of frailty—a syndrome reflecting declining physiological reserves and heightened stress vulnerability in the elderly—has garnered significant attention among patients with liver cirrhosis, particularly those in the decompensated stage. Frailty is not only closely associated with adverse clinical outcomes such as prolonged hospital stays, increased re-admission rates, and higher mortality, but also profoundly impacts patients' ability to live independently and their tolerance to treatment [3] [4]. However, the specific contributing factors and their interaction networks of hepatic frailty in decompensated cirrhosis patients require further elucidation. The Liver Frailty Index (LFI), a specialized assessment tool for liver disease populations, demonstrates strong reliability and validity, enabling precise quantification of hepatic frailty [5]. Therefore, this study applied the Liver Frailty Index (LFI) to evaluate the relationship between the multiple indicators of frailty, nutrition status, psychological and emotional state, and physical function in patients with decompensated cirrhosis, in order to provide theoretical reference and practical guidance for the establishment of an early identification and comprehensive intervention system.

## 2. Data and Methods

### 2.1. Research Subjects

This study employed a cross-sectional survey method, enrolling 477 hospitalized patients with decompensated cirrhosis from September 2021 to February 2022. Inclusion criteria were: (1) meeting diagnostic criteria for decompensated cirrhosis; (2) age  $\geq 18$  years; (3) voluntary participation with signed informed consent. Exclusion criteria included: (1) comorbidities such as malignancies or severe cardiovascular/cerebrovascular diseases; (2) cognitive impairment preventing assessment cooperation; (3) recent major surgeries or trauma; (4) incomplete clinical or

assessment data. With 16 independent variables, the study required a sample size 5 - 10 times larger than the variables, necessitating at least 160 participants. The protocol was approved by our hospital's ethics committee, and all participants provided written informed consent.

## 2.2. Research Methods

This study employed an observational cross-sectional design. All data were collected and assessed by two specialized liver disease research nurses during the patients' hospitalization, who had undergone systematic and standardized training. To ensure data quality and consistency, the training covered standardized interpretation of various scales, proper use of assessment tools (e.g., grip strength testers), standardized testing procedures, and a unified data recording format. Data collection was conducted after the patients' admission, when their condition stabilized following initial treatment and they could cooperate with the assessments, typically within 48 to 72 hours post-admission.

### 2.2.1. General Data Collection Form

Researchers used a self-designed general data collection form for information gathering. The questionnaire primarily included: (1) Demographic data: age, gender, marital status; (2) Anthropometric measurements: height and weight were measured using calibrated scales, with body mass index (BMI, kg/m<sup>2</sup>) calculated; (3) Clinical data: cirrhosis etiology (e.g., viral or alcoholic) was identified through medical records, along with current complications (ascites, hepatic encephalopathy, upper gastrointestinal bleeding) and their severity; (4) Laboratory parameters: fasting venous blood samples were collected the morning after hospital admission, and the hospital laboratory analyzed serum prealbumin, albumin, hemoglobin, and potassium levels using standard methods.

### 2.2.2. Anxiety and Depression Scale

The Anxiety Self-Rating Scale (SAS) and Depression Self-Rating Scale (SDS) were used to assess patients' anxiety and depressive symptoms. Both SAS and SDS are self-administered scales comprising 20 items, requiring patients to select responses based on their actual feelings during the past week. Scoring criteria: A 4-point Likert scale was employed, with items primarily evaluating symptom frequency: 1 (none or very few), 2 (a few), 3 (quite a few), and 4 (most or all). Total scores were calculated by summing individual items, with a score > 40 or a standard score exceeding 60 indicating the presence of anxiety or depressive symptoms. Higher scores indicate more severe symptoms. The Chinese version of this scale has been validated in multiple chronic disease populations, including chronic liver disease, demonstrating good reliability and validity, with Cronbach's  $\alpha$  coefficients typically reported above 0.80 and strong construct validity [6].

### 2.2.3. The NRS2002 Scoring System

The NRS2002 scoring system utilizes the 2002 Nutrition Risk Screening Tool recommended by the European Society for Parenteral and Enteral Nutrition. This

tool evaluates three dimensions: (1) Disease severity score (reflecting increased nutritional demands); (2) Nutritional status impairment score (based on BMI, recent weight loss, and reduced dietary intake over the past week); (3) Age score (1 point added for patients aged  $\geq 70$ ). The total score ranges from 0 to 7, with a score of 3 or higher indicating nutritional risk and necessitating the development and implementation of a clinical nutrition support plan. NRS2002 has been validated through large-scale multicenter trials internationally, demonstrating high validity in predicting clinical outcomes, along with reliable reliability and clinical applicability [7].

#### **2.2.4. Morse Scoring System**

The Morse Fall Risk Assessment Scale is employed to predict fall risks during hospitalization. This scale comprises six key components: fall history, number of comorbidities, use of walking aids, current intravenous therapy, gait characteristics, and cognitive status. Each component has clear scoring criteria, with a total range of 0 - 125 points. According to widely adopted clinical thresholds, a total score of  $\geq 45$  points indicates a high fall risk. The Morse scale, validated through extensive clinical practice, demonstrates excellent sensitivity and specificity in fall prediction, making it a reliable tool for nursing assessments [8].

#### **2.2.5. Daily Living Activity Assessment**

The Barthel Index Scale is used to evaluate patients' daily living activity capabilities. This scale comprises 10 basic activities: feeding, bathing, personal hygiene, dressing, bowel control, bladder control, toilet use, transfer from bed to chair, walking on flat surfaces, and stair climbing. Each activity is scored based on the level of assistance required, with a total score ranging from 0 to 100. Higher scores indicate greater independence and better ADL performance. The Barthel Index is one of the most widely used ADL assessment tools in rehabilitation medicine and geriatric medicine. Its inter-rater reliability and criterion validity have been repeatedly validated, demonstrating high reliability and validity [9].

#### **2.2.6. Liver Frailty Index (LFI)**

This study employs a liver-related frailty index to evaluate patients' physical frailty. The index comprises three tests: grip strength, timed standing test, and balance ability, which respectively indicate nutritional status, lower limb strength, and motor balance function. (1) Grip Strength Test: A calibrated electronic grip strength meter measures the maximum grip strength of the patient's dominant hand. The patient sits with elbows flexed at  $90^\circ$  and forearms in a neutral position. Three consecutive measurements are taken at 30-second intervals, with the maximum value recorded for calculating the Liver Frailty Index (LFI). (2) Timed Standing Test: This evaluates lower limb muscle strength and endurance. The patient sits on a standard 43 cm-high armless chair with arms crossed in front of the chest. The time required to complete five consecutive "stand-sit" movements as quickly as possible upon hearing the "start" command is recorded. This test reflects lower limb explosive power and functional strength. (3) Balance Ability

Test: This assesses static balance. Patients are required to perform three progressively more challenging standing postures in sequence, recording the maximum duration they can maintain each posture without support. The three postures are: a) Standing with feet together; b) Half tandem stance (one heel touching the mid-foot of the other); c) Tandem stance (heel-to-toe alignment). Records are kept for the patient's performance. The ability to maintain each posture for seconds (0-10 seconds), with a total score range of 0-30 seconds. Substitute the three test results into the structured formula for frailty performance:  $LFI = (-0.330 \times \text{grip strength (kg)}) + (-2.529 \times \log_5 (\text{chair stands time (s)})) + (-0.040 \times \log_5 (\text{balance time (s)})) + 6$ . The final calculation determines the severity of the patient's frailty. Patients with  $LFI \geq 4.5$ ,  $3.2 - 4.5$  and  $<3.2$  are categorized as frail, pre-frail, and normal. This index is a well-validated tool for assessing frailty in cirrhosis patients [10].

### 2.2.7. Statistical Analysis Methods and Procedures

Statistical Analysis Statistical data were analyzed using SPSS 26.0 software. For quantitative data, normality testing was performed first. Data conforming to a normal distribution were described as mean  $\pm$  standard deviation, with an independent samples t-test used for inter-group comparisons. For non-normally distributed data, the median and quartiles were reported, and the Mann-Whitney U test was employed for inter-group comparisons. Categorical data were presented as frequency and percentage, with the chi-square test or Fisher's exact probability method used for inter-group comparisons. Spearman's rank correlation analysis was conducted to examine associations between frailty and continuous variables such as nutritional risk scores and depression scores. To identify independent factors affecting frailty, a binary logistic regression model was constructed using variables with  $P < 0.1$  from univariate analysis and clinically significant variables as independent variables, with frailty status as the dependent variable. The forward stepwise method (likelihood ratio test) was applied for variable selection, with alpha levels set at 0.05 for inclusion and 0.10 for exclusion. The odds ratios and 95% confidence intervals for selected independent variables were calculated. All statistical analyses were performed with two-tailed testing, and differences were considered statistically significant when  $P < 0.05$ . The binary logistic regression model was selected as the dependent variable (frailty status) was dichotomous (frail vs. non-frail), which is suitable for identifying factors associated with the presence or absence of an outcome.

## 3. Results

### 3.1. Basic Characteristics and Frailty Status of Patients with Decompensated Cirrhosis

This study ultimately enrolled 477 patients with decompensated cirrhosis who met the inclusion criteria. The cohort comprised 387 males (81.13%) and 90 females (18.87%), with an age range of 22 to 83 years and a median age of 51.00 years. According to the standardized Liver Frailty Index (LFI) assessment, among

the 477 patients, 74 (15.51%) were categorized as normal (LFI < 3.2), 301 (63.10%) as pre-frail ( $3.2 \leq \text{LFI} < 4.5$ ), and 102 (21.38%) as frail (LFI  $\geq 4.5$ ). The overall prevalence of frailty was 21.38%. For subsequent regression analysis, patients were dichotomized into a frail group (n = 102) and a non-frail group (n = 375, combining normal and pre-frail categories).

### 3.2. Single-Factor Analysis of Frailty in Patients with Decompensated Cirrhosis

The study involved 477 participants. The frailty group and non-frailty group showed statistically significant differences in age, body mass index (BMI), prealbumin, albumin, hemoglobin, serum potassium, Morse score, NRS2002 score, ADL score, depression score, anxiety score, gender, ascites, and bleeding ( $P < 0.05$ ). However, marital status and hepatic encephalopathy showed no statistically significant differences ( $P > 0.05$ ), as shown in **Table 1**.

**Table 1.** Single factor analysis of frailty in patients with decompensated cirrhosis.

project	Total (n = 477)	No frailty group (n = 375)	weakened group (n = 102)	statistic	P
Age [years, M (Q1, Q3)]	51.00 (41.00, 59.00)	49.00 (40.00, 57.00)	59.50 (46.75, 68.75)	Z = -5.68	<0.001
Body mass index [kg/m <sup>2</sup> , M (Q1, Q3)]	22.72 (20.42, 25.39)	23.03 (20.58, 25.64)	21.71 (19.86, 23.88)	Z = -3.17	0.002
prealbumin [g/L, M (Q1, Q3)]	69.00 (46.00, 113.00)	74.00 (48.00, 116.00)	64.50 (43.00, 96.00)	Z = -2.05	0.040
Albumin [g/L, M (Q1, Q3)]	34.40 (31.00, 37.90)	34.70 (31.50, 38.10)	32.85 (30.02, 36.62)	Z = -2.95	0.003
Hemoglobin [g/L, M (Q1, Q3)]	112.00 (95.00, 127.00)	115.00 (99.50, 129.00)	100.00 (83.25, 117.00)	Z = -4.94	<0.001
K[mmol/L, M (Q1, Q3)]	3.83 (3.54, 4.16)	3.86 (3.57, 4.17)	3.67 (3.48, 4.11)	Z = -2.34	0.020
Morse score [points, M (Q1, Q3)]	35.00 (15.00, 45.00)	30.00 (15.00, 45.00)	45.00 (35.00, 45.00)	Z = -7.47	<0.001
ADL score [points, M (Q1, Q3)]	100.00 (85.00, 100.00)	100.00 (90.00, 100.00)	80.00 (60.00, 98.75)	Z = -8.81	<0.001
NRS2002 score [points, M (Q1, Q3)]	1.00 (0.00, 2.00)	1.00 (0.00, 2.00)	2.00 (0.00, 3.00)	Z = -3.59	<0.001
Depression score (points, M (Q1, Q3))	42.50 (35.00, 53.75)	41.25 (33.75, 51.25)	47.50 (41.25, 58.75)	Z = -4.85	<0.001
Anxiety score (points, M (Q1, Q3))	40.00 (33.75, 45.00)	38.75 (32.50, 45.00)	43.75 (37.50, 48.75)	Z = -4.54	<0.001
sex, n (%)				$\chi^2 = 7.75$	0.005
man	387 (81.13)	314 (83.73)	73 (71.57)		
woman	90 (18.87)	61 (16.27)	29 (28.43)		
Marital status, n (%):				$\chi^2 = 3.77$	0.152
unmarried	34 (7.13)	31 (8.27)	3 (2.94)		
married	429 (89.94)	334 (89.07)	95 (93.14)		
divorced or widowed	14 (2.94)	10 (2.67)	4 (3.92)		
ascites, n (%)				$\chi^2 = 10.60$	0.014
not have	231 (48.43)	194 (51.73)	37 (36.27)		
a small amount	100 (20.96)	77 (20.53)	23 (22.55)		

## Continued

Medium	55 (11.53)	36 (9.60)	19 (18.63)		
a large number	91 (19.08)	68 (18.13)	23 (22.55)		
Hepatic encephalopathy, n (%)				$\chi^2 = 0.42$	0.519
not have	429 (89.94)	339 (90.40)	90 (88.24)		
have	48 (10.06)	36 (9.60)	12 (11.76)		
haemorrhage, n (%)				$\chi^2 = 6.60$	0.010
not have	447 (93.71)	357 (95.20)	90 (88.24)		
have	30 (6.29)	18 (4.80)	12 (11.76)		

### 3.3. Correlation Analysis of Frailty and Nutritional Risk, and Depression in Patients with Decompensated Cirrhosis

The Spearman rank correlation analysis showed that the LFI value, a continuous variable representing the degree of frailty, was significantly positively correlated with the self-rated depression score and significantly negatively correlated with the hemoglobin level ( $P < 0.001$ ). Additionally, there was a significant positive correlation between the depression score and the NRS2002 nutritional risk score ( $P < 0.001$ ).

### 3.4. Binary Regression Analysis of Frailty in Patients with Decompensated Cirrhosis

A logistic regression analysis was conducted with frailty status as the dependent variable and age, gender, body mass index (BMI), NRS2002 score, anxiety score, depression score, prealbumin, albumin, hemoglobin, serum potassium, Morse score, ADL score, bleeding, and ascites as independent variables. The interpretation of the odds ratios (ORs) provides clinical insights. For the depression score, the OR of 1.03 (95% CI: 1.01 - 1.07) indicates that for each one-point increase in the depression score, the odds of being frail increase by approximately 3%, holding other factors constant. For hemoglobin, the OR of 0.98 (95% CI: 0.97 - 0.99) suggests that each one-unit (g/L) increase in hemoglobin level is associated with a 2% decrease in the odds of frailty. Similarly, each additional year of age increases the odds of frailty by about 4% (OR = 1.04), each one-point increase in Morse score increases the odds by about 2% (OR = 1.02), and each one-point increase in ADL score decreases the odds by about 3% (OR = 0.97). Depression score, hemoglobin level, age, Morse fall risk score, and ADL score were identified as independent factors affecting frailty status in decompensated cirrhosis patients. See **Table 2**.

**Table 2.** Binary regression analysis of frailty in patients with decompensated cirrhosis.

Project	$\beta$	S.E	Z	P	OR (95%CI)
nodal increment	1.95	2.07	0.95	0.344	7.05 (0.12 - 404.73)
age	0.03	0.01	3.13	0.002	1.04 (1.01 - 1.06)

**Continued**

constitutional index	-0.05	0.04	-1.32	0.188	0.95 (0.88 - 1.03)
K	-0.41	0.28	-1.48	0.140	0.66 (0.38 - 1.15)
albumin (g/L)	-0.02	0.03	-0.90	0.369	0.98 (0.93 - 1.03)
prealbumin	0.00	0.00	0.48	0.628	1.00 (1.00 - 1.01)
hemoglobin	-0.02	0.01	-2.27	0.023	0.98 (0.97 - 0.99)
ADL grade	-0.03	0.01	-4.41	<0.001	0.97 (0.95 - 0.98)
NRS2002 score	-0.09	0.11	-0.82	0.414	0.91 (0.74 - 1.13)
Morse grade	0.02	0.01	2.16	0.031	1.02 (1.01 - 1.03)
Depression score	0.03	0.02	1.98	0.047	1.03 (1.01 - 1.07)
Anxiety score	0.02	0.02	0.88	0.381	1.02 (0.98 - 1.06)
sex					
1					1.00
2	0.30	0.33	0.93	0.355	1.36 (0.71 - 2.59)
ascites					
0					1.00
1	0.04	0.37	0.11	0.909	1.04 (0.51 - 2.15)
2	0.07	0.43	0.16	0.870	1.07 (0.46 - 2.48)
3	-0.26	0.39	-0.66	0.507	0.77 (0.36 - 1.66)
haemorrhage					
0					1.00
1	0.72	0.49	1.47	0.140	2.05 (0.79 - 5.30)

**4. Discussion**

This study employed a disease-specific liver frailty index as an assessment tool, revealing a staggering 21.38% prevalence of frailty status among hospitalized patients with decompensated cirrhosis. These findings underscore the pervasive and severe nature of frailty in such patients, which has evolved beyond being merely a comorbid condition to becoming a critical factor influencing clinical progression and prognosis. Furthermore, through a multivariate regression model controlling for multiple confounding factors, the study identified depressive mood, anemia, advanced age, high fall risk, and reduced daily living activities as independent risk factors for frailty development. This breakthrough deconstructs the previously ambiguous functional decline often attributed to “advanced liver disease” into multiple measurable and partially intervenable clinical dimensions, providing clear intervention pathways for developing precision-oriented and integrated management strategies.

**4.1. Relationship between Nutritional Status and Frailty in Patients with Decompensated Cirrhosis**

Nutritional assessment often focuses on albumin and prealbumin, which primar-

ily reflect liver synthesis function and short-term nutritional changes [11] [12]. This study revealed that hemoglobin levels demonstrated greater independent predictive significance in multiple regression analysis. These findings suggest that the mechanisms by which nutrition affects frailty may be more complex in the specific pathological context of decompensated cirrhosis. Cirrhotic anemia results from multiple interacting factors, including portal hypertension-induced hypersplenism accelerating erythrocyte destruction, potential or overt gastrointestinal bleeding, deficiencies in hematopoietic substrates such as iron, folic acid, and vitamin B12 due to impaired metabolism or absorption, “chronic disease anemia” under chronic inflammatory conditions, and reduced erythropoietin responsiveness in hepatorenal syndrome [13] [14]. Hemoglobin levels, a key indicator of nutritional status, show that lower levels correlate with higher frailty indices, indicating poorer nutritional conditions. Patients with liver cirrhosis often experience complications like portal hypertension, which may cause nausea and vomiting, impairing digestion and absorption and ultimately leading to malnutrition [15]. Long-term malnutrition weakens patients’ physical strength, thereby exacerbating their frailty. Therefore, in clinical practice, nutritional support strategies should go beyond mere calorie and protein supplementation. They should involve systematic screening and intervention for various causes of anemia, improving patients’ nutritional status, and ultimately slowing the progression of frailty.

#### **4.2. Effect of Depressive Mood on Frailty in Patients with Decompensated Cirrhosis**

The results of this study confirm that depression is the strongest psychological predictor of frailty. Patients with decompensated cirrhosis are deeply troubled by disease symptoms, social function regression, economic burden, and fear of liver transplantation or death, and the prevalence of depression is much higher than that of the general population [16] [17]. Depression not only affects patients’ mental health but also exacerbates physical frailty through multiple pathways. Specifically, depression-induced symptoms such as appetite loss, sleep disturbances, and poor treatment adherence worsen malnutrition and metabolic disorders, creating a vicious cycle of depression, malnutrition, and physical decline [18] [19]. On the other hand, depressive mood may also reduce patients’ treatment adherence, impair therapeutic outcomes, and consequently accelerate the progression of frailty [20] [21]. Therefore, recognizing depression as a treatable complication with clear biological consequences is essential. Integrating routine psychological screening into liver disease management pathways and establishing collaborative referral mechanisms with psychiatry departments—through psychological interventions, social support, and safe pharmacotherapy—may be a highly cost-effective strategy to halt the progression of frailty and enhance overall treatment outcomes.

#### **4.3. The Role of Somatic Function in the Frailty of Patients with Decompensated Cirrhosis**

The findings of this study demonstrate that ADL capacity and fall risk represent

two distinct yet complementary manifestations of physical frailty. A decline in ADL ability indicates that a patient's physiological reserves required to maintain basic independence are nearing depletion or have been depleted, serving as a functional outcome of frailty. The elevated fall risk, however, represents a more advanced stage, revealing increased vulnerability in patients when facing dynamic environmental challenges. This serves as an early warning signal for frail individuals who are about to or have already been exposed to severe adverse events. In cirrhosis patients, this functional decline is influenced by multiple factors: portal hypertension-related hypermetabolism, hormonal imbalances, and chronic inflammation collectively contribute to cirrhosis-associated sarcopenia, which forms the biological basis of physical frailty [22] [23]. At the same time, mild coordination disorders and postural control abnormalities caused by hepatic encephalopathy, as well as side effects of medications (such as diuretics and sedatives), further increase the risk of falls [24] [25]. Clinical interventions must adopt a dual approach: first, through individualized, supervised exercise rehabilitation (combining aerobic, resistance, and balance training) to reverse or delay muscle loss and improve neuromuscular function; second, through standardized fall prevention protocols involving multidisciplinary teams to systematically identify and manage risks, protecting vulnerable patients from secondary injuries.

#### **4.4. Effect of Age on Frailty in Patients with Decompensated Cirrhosis**

Aging is the result of the decline of physiological reserve [26]. The results of this study also demonstrate that age remains a robust predictor of frailty. Aging is characterized by a natural decline in anabolic hormone levels, mitochondrial dysfunction, diminished stem cell regenerative capacity, and chronic inflammatory dysregulation in the immune system [27] [28]. When the natural physiological decline coincides with cirrhosis—a severe catabolic disorder—these factors create a synergistic effect that accelerates multi-system dysfunction. This means clinicians treating elderly decompensated cirrhosis patients must exercise heightened premature frailty awareness. Even when a patient's Child-Pugh liver function score resembles that of younger counterparts, their functional reserves and rehabilitation potential may differ substantially. Consequently, treatment goals and supportive strategies should be tailored to individual needs with a conservative approach.

### **5. Limitations**

This study has several limitations. First, the cross-sectional design cannot determine causality between the observed associations (e.g., depression and frailty), only their correlations. Second, the study sample was drawn from hospitalized patients at a single center, which may introduce selection bias, and the results may not be generalizable to outpatients or community-dwelling patients with cirrhosis. Future multicenter, prospective cohort studies are needed to validate these findings and explore causal pathways.

## 6. Conclusion

Frailty in decompensated cirrhosis patients constitutes a clinical syndrome involving multiple dimensions: psychological depression, nutritional anemia, age-related decline, reduced functional capacity, and elevated fall risk. This finding underscores the need to transition from a disease-focused approach emphasizing complication management to an integrated model prioritizing overall functional preservation and quality of life. Clinical practice should implement standardized screening protocols for frailty, supported by multidisciplinary teams comprising hepatologists, nurses, clinical dietitians, psychiatrists/psychologists, and physiotherapists. These teams should deliver tailored interventions. For instance, a concrete example of a tailored nutritional intervention could involve not only calorie-protein optimization but also systematic screening and correction of iron, folate, or vitamin B12 deficiencies based on individual laboratory profiles to address anemia-specific mechanisms contributing to frailty. Future research should explore specific nutritional and psychological intervention strategies with validated assessment methods, conduct prospective studies to establish causal relationships between these factors and clinical outcomes, and design evidence-based “psycho-social-nutritional-motor” multimodal interventions. Additionally, developing accessible screening tools will facilitate deeper integration of frailty management into comprehensive cirrhosis care, ultimately improving patient prognosis.

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## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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