Dear Reviewers and Editors

Thank you for your helpful corrections. We have corrected the paper according to your comments. Our replies to the questions raised by the reviewers are as follows.

For the first reviewer:

1. Wha is meant by (0 < CONUT score < 4)? very bad presentation of the score

Regarding the presentation "mean of $0 \le \text{CONUT score} \le 4$ ", which indicated that the patient CONUT score was ≤ 4 (0, 1, 2, 3, or 4), we have revised the presentation: patients were divided into low, medium, and high CONUT score groups (low score group: score ≤ 4 ; medium score group: $5 \le \text{score} \le 8$; high score group: score $9 \le \text{score} \le 12$).

2. Is your data was parametric or not parametric???

The mean \pm SD is reported for normally distributed data, and the independent sample t test was used for intergroup comparisons. The median (quartile) is presented for nonnormally distributed data, and the Mann-Whitney U or Kruskal-Wallis test (non-parametric test) was used for the intergroup comparisons.

3. This sentence (It has been reported that the PMTH is correlated with the sex and mortality of cirrhosis patients [6]. The ROC curves of males and females were generated according to postoperative patient mortality, and PMTH cutoff values were selected as those that yielded the optimal Youden Index) is unclear it is result or discussion or what?? what is Youden Index????

We wanted to say that because the PMTH is correlated with sex, the cutoff values for sarcopenia in men and women are different, so we had to generate ROC curves to determine the cutoff values for men and women separately. The PMTH is correlated with the mortality of cirrhosis patients. Therefore, ROC curves to assess postoperative patient mortality were generated.

The Youden index= sensitivity+ specificity- 1. It was used to detect true and false positives. The maximum Youden index is the point at which the screening effect is maximal, and the cutoff value corresponds to the optimal (maximum) Youden index.

4. where grade I and II in Clavien-Dindo classification ???

Grade I :Any deviation from the normal postoperative course without the need for pharmacological treatment or surgical, endoscopic, and radiological interventions Allowed therapeutic regimens are: drugs as antiemetics, antipyretics, analgetics, diuretics, electrolytes, and physiotherapy. This grade also includes wound infections opened at the bedside

Grade II :Requiring pharmacological treatment with drugs other than such allowed for grade I complications Blood transfusions and total parenteral nutrition are also included

We think grade I and grade II complications are not the typical after LT. We aimed to focus on complications that require surgical, endoscopic or radiological intervention and higher grade of complications.

5. How to consider low CONUT and low PMTH as normal although original score consider them low malnourished?

We did not sufficiently consider a low CONUT score, which should indicate a low risk of malnutrition. We aimed to combine the two tools to predict prognosis more accurately. However, both reviewers expressed doubts regarding this idea. After repeated consideration, we decided to delete this part of the study for the following reasons: 1. the number of patients included in the last comparison (64 vs 44) was too small to draw definite conclusions. 2. There was no significant difference between the malnutrition (high CONUT+low PMTH) and low PMTH groups in the incidence of grade V complications (20.6% vs 17.3%, P=0.668). Therefore, this combined consideration of the CONUT score and PMTH did not perform as well as we expected. We hope to include this analysis after expanding the sample size.

6.It does not discuss the paper's scientific significance and/or relevance to clinical practice sufficiently?

We have added this discussion.

For the second reviewer:

1.the MELD score was higher in the low CONUT group (18 vs. 14). A comment

on this point may be valuable

Our data show that the MELD score was **lower** in the low CONUT group than in the high CONUT group (14 vs. 18). We believe that this result is related to the progression of disease. Patients with a longer course of disease have more severely impaired liver function, resulting in an increased bilirubin and INR.

2. Why the Authors decided to rule out "marginal donors"?

Because many study shows that marginal donors result in a poor prognosis for liver transplantation recipients[1-5]. Although the use of marginal donors has increased this year[6-8], it remains controversial. So in order to minimize the uncontrollability factor, we decided to rule out "marginal donors".

3.indeed, serum cholesterol may be increased in patients with PBC or PSC, whereas alcohol is associated with worse nutritional status.

There are our limitations. In the early stage of primary biliary cirrhosis and primary sclerosing cholangitis, patients have dyslipidemia that leads to hypercholesterolemia, which then gradually decreases as the disease progresses. This affects the CONUT score in some patients. In addition, studies have shown that patients with alcoholic cirrhosis have a higher incidence of malnutrition than patients with nonalcoholic cirrhosis, probably because ethanol and its metabolites may affect protein synthesis and skeletal muscle autophagy, but whether this leads to sarcopenia needs to be further validated by expanding the number of cases (added in our paper with references)

4. Can we assume that sarcopenia was the driver in survival, or that CONUT score was not able to predict the degree of nutrition in cirrhosis?

There is no doubt that many studies have shown an association between sarcopenia and recipient mortality. The CONUT score predicts mortality after hepatectomy for hepatocellular carcinoma. However, our data show that the CONUT score cannot predict mortality after LT, as was found in another study. We think this inconsistency in results is because LT does feature some improvement in nutrition-related indices, especially in terms of the elevation of albumin. This may avoid the most severe complications (death), but it cannot improve sarcopenia over a short time. (added in our paper with references)

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5. A comment about anemia may be of interest. What is the clinical significance of anemia? Shall we consider it a surrogate marker of malnutrition or a marker of portal hypertension?

We have added the discussion of anemia in our paper.

References:

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