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PEER-REVIEW REPORT

Name of journal: *World Journal of Clinical Cases*

Manuscript NO: 75338

Title: Analysis of short-term prognostic factors of hepatitis B virus-related acute-on-chronic liver failure

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 00052947
Position: Peer Reviewer
Academic degree: MD

Professional title: Director, Professor

Reviewer's Country/Territory: Germany

Author's Country/Territory: China

Manuscript submission date: 2022-01-24

Reviewer chosen by: AI Technique

Reviewer accepted review: 2022-01-28 17:03

Reviewer performed review: 2022-02-01 17:10

Review time: 4 Days

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [ ] Grade C: Good [Y] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [ ] Minor revision [Y] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No

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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

General comment I. The accurate prognosis of HBV-related ACLF is an important topic, particularly in China. The authors emphasize the usefulness of GP73 as a prognostic marker for survival and the necessity of a liver transplantation. They present a table with a large number of prognostic markers but they somehow fail to prove that GP73 is really superior to many other markers (point 15) except for the well-known old-fashioned MELD score. II. The discussion of HBV markers is almost completely neglected. More importantly, the role of previous or ongoing antiviral therapy of the patients is kept secret (point 8 and 12). III. Methological data are highly incomplete. IV. The discussion of the data as well of the relevant literature is superficial. V. Furthermore, the text contains many mistakes as pointed below. Specific points 1. Replace HBeAb and HBcAb by antiHBe and antiHBc throughout the text. 2. L45. Abstract and later. Replace "Golgi apparatus 73" by "Golgi protein 73" (GP73). 3. L50. Abstract and later. What is lgHBsAg? Shall this be the log 10 of the HBsAg concentration in IU/mL? Lg should be replaced throughout by log. LgHBsAg could be misunderstood as large HBsAg. 4. L93. Introduction. What is meant here by "international standard value" as a substitute for prothrombin? Should it be INR? 5. L101-104. It should be briefly mentioned what the biological function of GP73 is. 6. L127. M&M. Exclusion criteria. "Other hepadnavirus (hepatitis A,C,D, V); " Replace "hepadnavirus" by "hepatitis viruses". HAV, HCV and HDV are not hepadnaviruses. What is meant by hepatitis V? 7. L129. Were HBV-related HCC cases excluded? 8. L130 or elsewhere. Mention whether the patients received antiviral therapy, for how long and with which drugs. (See point 12). 9. L 139-144. Staging. The text is confusing. Shall "30% <

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prothrombin time activity (PTA) ≤ 40%" mean "prothrombin time activity (PTA) 30 - 40%"? Correct the other data accordingly. 10. L153, 154. The methods for quantitation of AFP, GP73, HBV DNA, and HBsAg and the producers of the test kits should be reported. 11. The legend to table 1 is too brief. It should be mentioned here also that the numbers refer to means or medians and s.d. or 25%/75% Quartiles. a. The units for logHBsAg and logHBV-DNA are missing. IU/mL? b. The prevalence of HBeAg and anti-HBe in the two groups is missing. The brief sentence in L253 should be supported with exact numbers. 12. L220, 235. Discussion. A severe shortcoming of the paper is that it is not discussed why the patients experienced ACLF although their status as CHB patients with more or less progressed cirrhosis was known for at least 6 months. According to table 1, the median of HBV DNA is in most patients ca. 10E5 – 10E8 (probably IU/mL, see 11a). That means, the majority of the 207 patients did not receive an efficient antiviral therapy with tenofovir or entecavir, or at least too late. The perspectives for a liver transplantation are dim if the patient still has so high levels of HBV. This must lead to immediate infection of the new liver. Antiviral therapy cannot prevent entry of the HBV genomes into the nucleus and establishment of cccDNA. 13.

L232-241. Concerning the role of NLR in ACLF more recent articles should be cited, e.g.: Clin Lab 2021 Dec 1;67(12). doi: 10.7754/Clin.Lab.2021.210238. Peripheral Blood Cell Ratios as Prognostic Predictors of Mortality in Patients with Hepatitis B Virus-Related Decompensated Cirrhosis. XiaoTing Qi, ChangMin Wang, XinJie Shan. PMID: 34910423 --- The title does not explicitly mention ACLF, but mortality within 30 days with decompensated cirrhosis is virtually identical with ACLF. There are more similar papers to be cited. 14. L245. Correct this line: GP73 is also increased in HCC but it is not a liver cancer marker. It is a marker for hepatic inflammation. 15. In table 1, the laboratory markers NLR, INR, HBsAg and AFP appear at p-levels as discriminatory or even better than GP73. It should be discussed in more detail why the authors favor so

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much GP73. a. A scatter plot for the individualGP73 levels in the two groups would be informative. b. Ref. 8 writes: "The sensitivity and specificity of GP73 for the diagnosis of G2 hepatic necroinflammation was 42.35% and 95.0%, respectively, at a cut-off value of 88.38 ng/mL." c. Ref. 9 writes: When the cut-off value was set at 182.1 ng/mL, the sensitivity and specificity of HBV-ACLF diagnosis were 77.62% (95% confidence interval [CI]: 71.37%-83.07%) and 95.50% (95% CI: 92.27%-98.26%), respectively. d. Ref. 14 reports GP73 levels for various groups of CHB patients with increasing severity. How do their results compare to this study? How many patients would exceed the cutoffs suggested by other authors? e. Could the authors of this study also calculate a cutoff? At least they should discuss these three papers in more detail. f. The evaluation of these markers should have included combinations of markers, at least the combination of GP73 and NLR. 16. Table 1 suggests that HBV DNA was in some deceased patients even higher than in the survivors. a. This suggests furthermore that HBV replication was the major pathogenic factor leading to the seemingly "acute" exacerbation both in the survivors and the deceased. b. The lower HBsAg levels in the deceased patients reflects partially the loss of functional liver tissue. 17. References a. Ref. 1 is in Chinese. WJG is a Chinese journal but claims to publish for the world. There must be suitable survey articles on the problem in English as number 1. The authors should cite it later and discuss how much their data contribute to the Chinese (and possibly international) guidelines.

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Professional title: Associate Professor, Chief Physician
Reviewer's Country/Territory: Thailand
Author's Country/Territory: China

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SPECIFIC COMMENTS TO AUTHORS

1. The units should be the standard unit eg. BUN mg/L, CHE U/L etc. 2. The abbreviation should be arrange as A to Z. 3. The references should be modified as correct format.