Letter to the Editor: A prognostic model in patients with alcohol-associated cirrhosis does exist

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Corresponding Article

Prediction models for liver decompensation in compensated advanced chronic liver disease: A systematic review

Haghnejad, Vincent; Burke, Laura; El Ouahabi, Siham; Parker, Richard; Rowe, Ian A.

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CORRESPONDENCE

Letter to the Editor: A prognostic model in patients with alcohol-associated cirrhosis does exist

To the editor,

We read with great interest the review by Haghnejad et al.[1] on the usefulness of prediction models for liver decompensation in compensated advanced chronic liver disease (cACLD). The authors stated that "no study specifically predicted outcomes in persons with alcohol-associated liver disease." They limited their analysis to studies where modeling the risk of decompensation was the primary objective, although another highly relevant endpoint is liver-related death. A recent study from our research group was focused on precisely this issue. In this study, we built and validated a prognostic model for individualized prediction of liver-related death in outpatients with alcohol-associated cirrhosis.[2]

Haghnejad and colleagues emphasized that "models for predicting decompensation in cACLD are often poorly described and lack external validation." In our study, robust statistical analyses were performed, including competing risk analysis, internal validation by using bootstrap resampling, and external validation (temporal validation). The model accuracy was assessed by measures of calibration and discrimination. These analyses serve to quantify how close predictions are to the actual outcome. A total of 527 and 127 patients were included in the derivation and validation data sets, respectively. The developed model was based on the variables that were independently associated with liver-related mortality in multivariate analyses: age, Child–Pugh score, and abstinence. It was clinically useful and provided the best prognostic accuracy and the best discriminative ability for evaluating 5-year liver-related mortality compared with the Child–Pugh score or the MELD score alone.

Haghnejad and colleagues also stated that parameters considered in prediction models are sometimes "not widely measured in practice." In contrast to this claim, we have developed a model that uses 3 readily available variables that can easily be set. We provided charts showing the individual risk of liver-related mortality according to these 3 variables. The model converts any individual score (age and Child–Pugh score) into a risk of liver mortality at 5 years according to the value of the other variable among abstainers and consumers (Figure 1). We acknowledge that not all patients were compensated. Nevertheless, this model can be applied to compensated patients. We also acknowledge that the model predicts mortality rather than first decompensation, but a detailed analysis of the causes of death allowed us to predict liver-related mortality. Of note, our study emphasized the huge impact of abstinence in these patients, a feature that is poorly

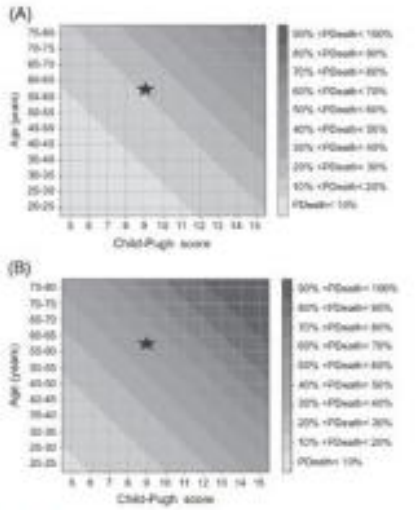

FIGURE 1 Chart predicting the risk of liver-related death at 5 years in patients who abstained and who did not abstain from alcohol. Hypothetical 60-year-old patient with a Child–Pugh score of 9 at baseline (A) who abstained from alcohol (5-year liver-related mortality rate: 23%) and (B) who did not abstain from alcohol (5-year liver-related mortality rate: 51%).