Reply. We would like to thank Dr Mohta and colleagues for their interest in our study and for their comments. As we showed in our work, our cohort of patients with advanced nonalcoholic fatty liver disease (NAFLD) developed clinical decompensation at lower hepatic venous pressure gradient (HVPG) values as compared with a cohort of patients with hepatitis C virus–related chronic liver disease. We hypothesized that this may be a consequence of an underestimation of the actual portal pressure as assessed by the HVPG measurement, although we agree with the authors that the causes underlying this observation may be multifactorial. However, we politely disagree with the comment about the rarity of ascites development in patients with presinusoidal portal hypertension.

As stated by the authors, there is growing evidence that indicates the pathogenic role of the microbiome and derangements in the gut-liver axis in the development of NAFLD. Unfortunately, our study was not designed to look at this link and future studies are needed to evaluate the impact on HVPG values of bacterial translocation and systemic inflammation in patients with NAFLD.

Liver stiffness measurement has been extensively studied and compared with HVPG as a noninvasive tool to predict clinical decompensation in patients with chronic liver disease even in the NAFLD population. Data about liver stiffness were not available in our cohort, and therefore we cannot draw any conclusion in this respect. Whether liver elastography correlates better than HVPG with the presence of decompensation, specifically in those patients with an HVPG < 10 mm Hg, needs further investigation.

References

Conflicts of interest
The authors disclose no conflicts.