JHEP|Reports

Cirrhotic cardiomyopathy and beyond: Underscoring the interaction between the liver and the heart

To the Editor:

The review by Liu *et al.* elegantly summarizes the pathophysiology and clinical burden of cirrhotic cardiomyopathy (CCM).[1] We aim to highlight the complex and dynamic interactions between the liver and the heart.

First, CCM can be considered end-stage cardiomyopathy with multiple causes, including coronary artery disease (CAD), and various liver diseases are associated with CAD. Notably, metabolic dysfunction-associated steatotic liver disease (MASLD) and primary biliary cholangitis (PBC) are associated with obstructive and non-obstructive CAD, respectively, requiring different clinical management and prevention strategies.

Patients with MASLD have different degrees of dysmetabolism and insulin resistance, which translate into an increased risk of obstructive coronary artery disease and atherosclerosis.[2] The progression of MASLD towards compensated advanced chronic liver disease is also strictly associated with cardiovascular events.[3]

PBC is characterized by an increased microvascular resistance and reduced coronary artery flow reserve.[4] Additionally, these patients are more likely to suffer from autonomic dysfunction, a condition associated with an increased risk of cardiovascular mortality and reduced tolerance of beta-blockers.[5]

Efforts should be directed towards identifying CAD in the early stages, activating tailored strategies against obstructive or non-obstructive CAD, and possibly mitigating the risk of developing CCM.

Second, we aim to underscore the intricate interplay between the liver and the heart along the trajectories of the natural history of cirrhosis. The revised Baveno VII classification of cirrhosis, including compensated advanced chronic liver disease, non-acute decompensation, stable decompensated cirrhosis, unstable decompensated cirrhosis, pre-acute-on-chronic liver failure (pre-ACLF), and ACLF, better aligns with the dynamics of hepatic insufficiency and portal hypertension.[6] A comparison of the natural history of cirrhosis and decompensated heart failure has become more straightforward with this classification, as both conditions are characterized by phases of decompensation and shared triggers, such as ischemia, infection, or alcohol consumption. Given the potential pathohistological association and epidemiological burden of cardiovascular diseases, dedicated studies are needed to evaluate the role of the heart as a potential trigger for ACLF.

Finally, considering the high mortality risk associated with both conditions, a multidisciplinary and holistic approach are imperative in managing patients with CCM.[7,8]

In conclusion, while the review by Liu *et al.* serves as a comprehensive exploration of CCM, we underline the importance of a tailored cardiovascular prevention, and propose further studies to better understand the complex interplay between decompensated heart failure and liver failure.

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Received 22 March 2024; Received in revised form 8 April 2024; Accepted 11 April 2024; Available online 23 May 2024
https://doi.org/10.1016/j.jhepr.2024.101114

Financial support

No funding was obtained for this research.

Conflict of interest

All authors have no relevant conflicts to disclose.
Please refer to the accompanying ICMJE disclosure forms for further details.

Authors' contributions

Conceptualization and draft of the letter: GL and VC. Critical revision for important intellectual content: SC and LR.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhepr.2024.101114.

References

Author names in bold designate shared co-first authorship

[1] **Liu H**, **Naser JA**, Lin G, et al. Cardiomyopathy in cirrhosis: from pathophysiology to clinical care. JHEP Rep 2024;6(1):100911.

[2] Zhang Z, Zheng M, Lei H, et al. A clinical study of the correlation between metabolic-associated fatty liver disease and coronary plaque pattern. Sci Rep 2023;13(1):7224.
[3] Bischoff SC, Barazzoni R, Busetto L, et al. European guideline on obesity care in patients with gastrointestinal and liver diseases - joint ESPEN/UEG guideline. Clin Nutr. 022;41(10):2364-2405.
[4] Cazzagon N, Dal Lin C, Famoso G, et al. Coronary flow reserve in patients with primary biliary cholangitis. Dig Liver Dis 2019;51(4):542–548.
[5] Jopson L, Dyson JK, Jones DEJ. Understanding and treating fatigue in primary biliary cirrhosis and primary sclerosing cholangitis. Clin Liver Dis 2016;20(1):131–142.
[6] Mendizabal M, Cançado GGL, Albillos A. Evolving portal hypertension through Baveno VII recommendations. Ann Hepatol 2024;29(1):101180.
[7] Xu M, Chen Y, Artru F. Acute decompensation ofcirrhosis versus acute-on-chronic liver failure: what are the clinical implications? United Eur Gastroenterol J 2024;12(2):194–202.
[8] McDonagh TA, Metra M, Adamo M, et al. 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: developed by the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur J Heart Fail 2024;26(1):5–17.