Check for updates

pISSN 2287-2728
eISSN 2287-285X

CMH

https://doi.org/10.3350/cmh.2018.0098
Clinical and Molecular Hepatology 2018;24:372-373

Editorial

Cirrhotic cardiomyopathy: An independent prognostic factor for cirrhotic patients

Yun Bin Lee and Jeong-Hoon Lee

Department of Internal Medicine and Liver Research Institute, Seoul National University College of Medicine, Seoul, Korea

Keywords: Cardiomyopathy; Liver cirrhosis; Diastolic dysfunction; Echocardiography

See Article on Page 409

Cirrhotic cardiomyopathy (CCMP), which indicates cardiac dysfunction in cirrhotic patients, has received attention as a complication of liver cirrhosis. Cardiac dysfunction observed in cirrhotic patients is characterized by electrophysiological abnormalities, diastolic dysfunction, and impaired and blunted responsiveness to physiologic or pathologic stress in the absence of underlying heart disease.[1-3] Sinusoidal portal hypertension in advanced liver cirrhosis causes increased production of a number of vasoactive mediators, such as nitric oxide, endothelin, and prostaglandin production, which drives splanchnic vasodilation resulting in hyperdynamic circulation.[2,4,5] Electrophysiological abnormalities including QT–interval prolongation and echocardiographic findings reflecting diastolic and/or systolic dysfunction in cirrhotic patients have been demonstrated as typical findings indicating CCMP.[2,6] However, diagnostic criteria of CCMP have not yet been established, and the association between CCMP and the clinical outcome of cirrhotic patients has not been well clarified.

In this issue of *Clinical and Molecular Hepatology*, a Korean prospective study by Lee et al. implicates that the presence of left ventricular diastolic dysfunction (LVDD), which is an early manifestation of CCMP, is associated with poor survival in patients with decompensated cirrhosis.[7] Seventy patients with decompensated cirrhosis were enrolled prospectively and cardiac function of the study patients was assessed using 2D echocardiography with tissue Doppler imaging. LVDD was diagnosed in 44 of 70 patients (62.9%), and 10 patients (22.7%) among 44 patients with LVDD at baseline were Child-Pugh class A. Overall survival was significantly worse in these patients with LVDD than those without LVDD (31.1 months vs. 42.6 months, P=0.01). The presence of LVDD was an independent predictor of overall survival (vs. no LVDD; adjusted hazard ratio [HR], 4.69; 95% confidence interval [CI], 1.06–20.8; P=0.042). These findings implicate that a considerable number of patients among cirrhotics with relatively preserved liver function may have CCMP. Moreover, CCMP was associated with poor survival outcome independently of severity of liver disease, suggesting the need for screening and monitoring for cardiac function in cirrhotic patients even if the patient is asymptomatic.

In a study by Lee et al., there was no difference in liver function index (Child-Pugh class and MELD score) according to the presence or absence of LVDD.[7] Recently, an European prospective,

Abbreviations:

CCMP, cirrhotic cardiomyopathy; CI, confidence interval; CMR, cardiac magnetic resonance; ECV, extracellular volume; HR, hazard ratio; LVDD, left ventricular diastolic dysfunction; LV-GLS, left ventricular global longitudinal strain

Corresponding author : Jeong-Hoon Lee
Department of Internal Medicine, Seoul National University Hospital, 101 Daehak-ro, Jongno-gu, Seoul 03080, Korea
Tel: +82-2-2072-2228, Fax: +82-2-743-6701
E-mail: pindra@empal.com
https://orcid.org/0000-0002-0315-2080

Received : Nov. 26, 2018 / **Accepted :** Dec. 3, 2018

multicenter study investigating the presence of diffuse myocardial fibrosis in cirrhotic patients using cardiac magnetic resonance (CMR) was reported.[8] Structural myocardial changes including diffuse myocardial fibrosis were assessed by quantification of the extracellular volume (ECV) on CMR. Myocardial ECV was significantly higher in cirrhotic patients compared to the healthy controls (31.2% vs. 27.4%, P=0.04), and furthermore, increased across the Child-Pugh class (26.9% in Child-Pugh class A, 31.5% in Child-Pugh class B, and 34.4% in Child-Pugh class C; P=0.02). In addition, ECV was associated with worse transplant-free survival (HR, 3.6; 95% CI, 1.1–11.6; P=0.03). The authors proposed a novel method for the diagnosis of subclinical CCMP by applying ECV measurement technique using CMR in patients with cirrhosis and showed possibility of ECV measurement as an index reflecting cardiac structural change of CCMP. Our group also reported results from a prospective study including 52 patients (32 cirrhotics and 20 healthy controls) to investigate the association between diffuse myocardial fibrosis and the severity of liver disease and to explore cardiac functional/structural changes after liver transplantation.[9] In our study, LV hypercontractility in patients with cirrhosis was reaffirmed via CMR-based LV ejection fraction and LV-global longitudinal strain (LV-GLS) measurements. We found that both increased LV-GLS and ECV in cirrhotic patients decreased at 1 year after transplantation, suggesting normalization of the LV systolic function and a decrease in diffuse myocardial fibrosis. In addition, our case-control study showed that nonalcoholic fatty liver disease was associated with increased risk for LVDD and the risk was incrementally increased according to fibrosis grade among subjects without cirrhosis.[10] These findings suggest that LVDD may develop in earlier stage of chronic liver disease.

CCMP is not just a phenomenon secondary to hyperdynamic circulation, but it has important prognostic value in cirrhotic patients. Moreover, CCMP is a concept that includes not only a functional change due to hemodynamic change but also a structural change in myocardium itself. It is necessary to establish diagnostic criteria for early diagnosis of CCMP in patients with cirrhosis. LVDD might be an early indicator of CCMP. Additionally, further studies to investigate whether thorough monitoring and management can improve long-term outcomes of cirrhotic patients are warranted.

Authors' contribution

Manuscript writing: Yun Bin Lee and Jeong-Hoon Lee
Final approval of manuscript: Yun Bin Lee and Jeong-Hoon Lee

Conflicts of Interest

The authors confirm that this article content has no conflict of interest.

REFERENCES

1. Møller S, Henriksen JH. Cirrhotic cardiomyopathy. J Hepatol 2010;53:179-190.
2. Zardi EM, Abbate A, Zardi DM, Dobrina A, Margiotta D, Van Tassell BW, et al. Cirrhotic cardiomyopathy. J Am Coll Cardiol 2010;56:539-549.
3. Møller S, Hove JD, Dixen U, Bendtsen F. New insights into cirrhotic cardiomyopathy. Int J Cardiol 2013;167:1101-1108.
4. Woitas RP, Heller J, Stoffel-Wagner B, Spengler U, Sauerbruch T. Renal functional reserve and nitric oxide in patients with compensated liver cirrhosis. Hepatology 1997;26:858-864.
5. Bernardi M, Fornalè L, Di Marco C, Trevisani F, Baraldini M, Gasbarrini A, et al. Hyperdynamic circulation of advanced cirrhosis: a re-appraisal based on posture-induced changes in hemodynamics. J Hepatol 1995;22:309-318.
6. Pozzi M, Carugo S, Boari G, Pecci V, de Ceglia S, Maggiolini S, et al. Evidence of functional and structural cardiac abnormalities in cirrhotic patients with and without ascites. Hepatology 1997;26:1131-1137.
7. Lee SK, Song MJ, Kim SH, Ahn HJ. Cardiac diastolic dysfunction predicts poor prognosis in patients with decompensated liver cirrhosis. Clin Mol Hepatol 2018;24:409-416.
8. Wiese S, Hove J, Mo S, Mookerjee RP, Petersen CL, Vester-Andersen MK, et al. Myocardial extracellular volume quantified by magnetic resonance is increased in cirrhosis and related to poor outcome. Liver Int 2018;38:1614-1623.
9. Lee YB, Kim HM, Chung SW, Kim M, Kim SW, Yoon JS, et al. Characterization of cirrhotic cardiomyopathy using cardiac magnetic resonance imaging: a prospective study [Abstract]. Clin Mol Hepatol 2018;24(Suppl 3):68-69.
10. Chung GE, Lee JH, Lee H, Kim MK, Yim JY, Choi SY, et al. Nonalcoholic fatty liver disease and advanced fibrosis are associated with left ventricular diastolic dysfunction. Atherosclerosis 2018;272:137-144.