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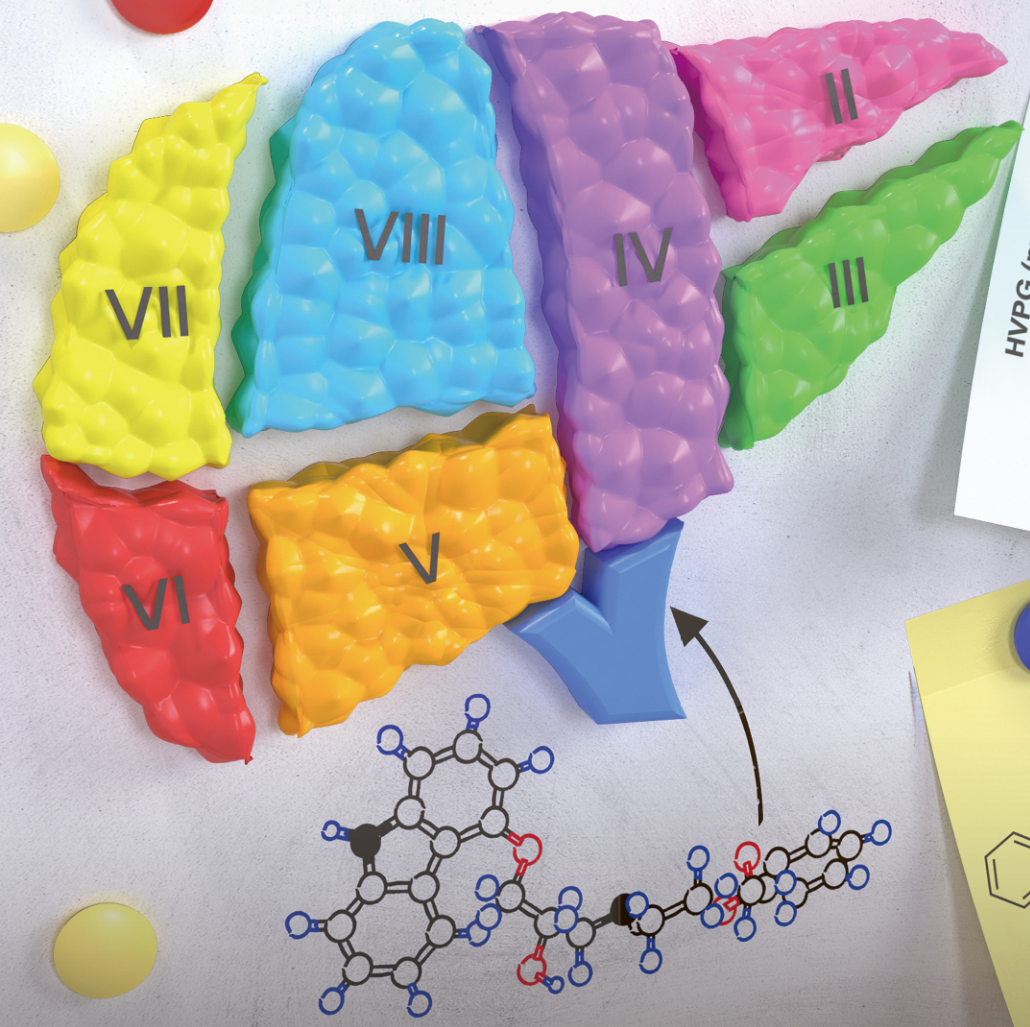

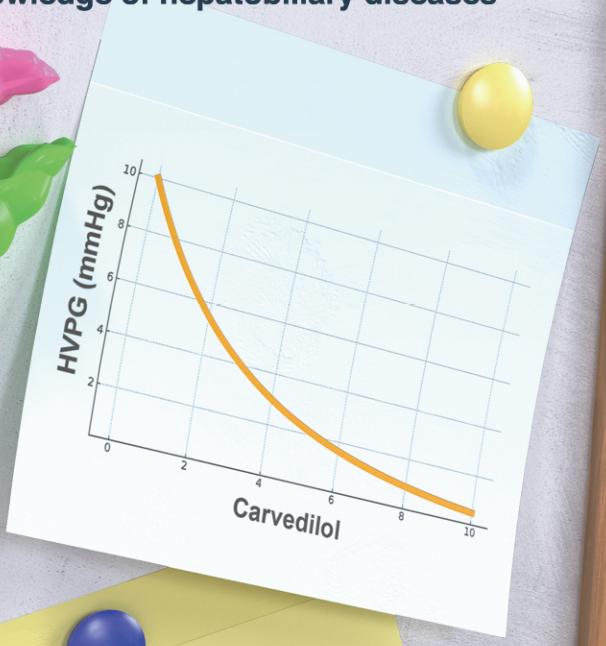

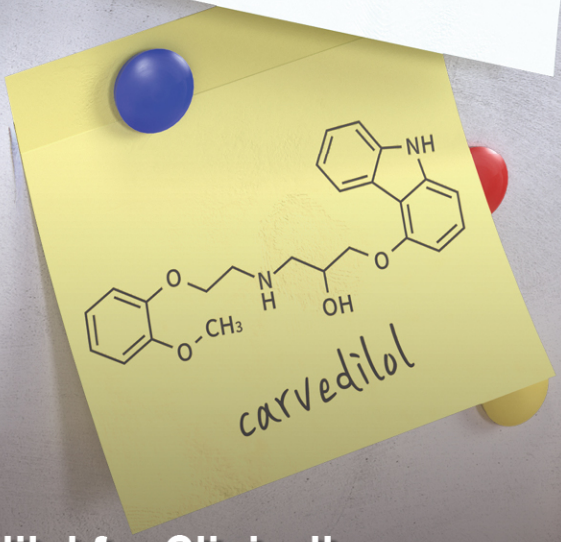

Non-invasive Model guiding Carvedilol for Clinically Significant Portal HTN

Intrapatient variability of tacrolimus on CKD in LT
HCV self-testing and disease burden reduction
MASLD and microbiota
Bariatric surgery for metabolic cirrhosis

KASL

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Correspondence

Steatotic liver disease in chronic hepatitis C related hepatocellular carcinoma: Inflictor or bystander?: Correspondence to editorial on "Dynamic change of metabolic dysfunction-associated steatotic liver disease in chronic hepatitis C patients after viral eradication: A nationwide registry study in Taiwan"

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Keywords: HCV; MASLD; SVR

Dear Editor,

We acknowledge and concur with the Editorial regarding the importance of the surveillance of both cardiometabolic risk factors (CMRFs) and concurrent steatotic liver disease (SLD) in chronic hepatitis C (CHC) before and after viral eradication.[1] The authors proposed an algorithm that enables holistic care after hepatitis C virus (HCV) eradication from both metabolic and hepatic aspects. While the European Association for the Study of the Liver (EASL) has advocated the last version regarding the treatment of HCV infection in 2020,[2] the authors denoted an updated EASL position statement with respect to the post-HCV cure follow-up.[3] It highlighted the importance of identifying metabolic dysfunction-associated steatotic liver disease (MASLD) in patients with borderline advanced liver fibrosis (liver stiffness of 8–10 kPa or fibrosis-4 index of 1.45–3.25), of whom diet and lifestyle modification and continuous fi-

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brosis assessment are warranted. Notably, the statement was based on the status before antiviral therapy. As the two components of MASLD, SLD and CMRF, would evolve after HCV eradication, the dynamic change of MASLD status should be taken into consideration.[4]

Critically, accompanied with the new definition of MASLD, it remains unclear whether SLD or CMRF contributes more to the development of hepatocellular carcinoma (HCC) in the post-sustained virological response (SVR) era. We collected 1,120 biopsy-proven CHC patients and observed a trajectory relationship between hepatic steatosis and liver fibrosis (Fig. 1). The proportion of hepatic steatosis increased with the progression of fibrotic stage from F0 (20%), F1 (40%) to F2 (63%), indicating a potential lipotoxicity effect in fibrogenesis. Nevertheless, the proportion of hepatic steatosis started to decrease from F3 (55%) to F4 (41%), which might be attribute to burnout phenomenon (Fig. 1). While we adopt cross-sectional hepatic steatosis as the baseline variable to judge the longitudinal long-term outcome, it is hard to avoid and adjust the most critical confounder of HCC, the dynamic progression of liver fibrosis. On the other hand, the other component of MASLD, CMRF, possesses a mutual link with SLD.[5] We have recently demonstrated that liver disease severity increased with the increasing number of CMRFs carriage in CHC patients with MASLD.[6] Amongst the CMRFs, insulin resistance and diabetes are the major determinants for HCC in CHC and other etiologies,[7,8] which are also the driving force for hepatic steatosis. Under the circumstances, SLD may be a bystander rather than a direct insult for hepatocarcinogesis. With the emergent definition of MASLD and its evolution after HCV eradication, its relationship with HCC in the post-SVR era adds complexity and awaits clarification. In addition, subjects with MASLD are prone to have metabolic disarrangements. The frequent use of aspirin, metformin and statin as the chemoprevention of HCC also needs to be judged.[9-11] Taken collectively, further studies are warranted to address the impact of pre- and post-SVR MASLD on HCC by weighting coexistent CMRFs and liver fibrosis.

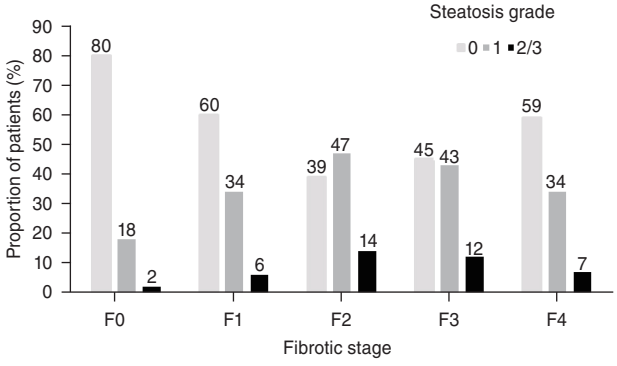

Figure 1. Association of hepatic steatosis and liver fibrosis in 1,120 biopsy-proven chronic hepatitis C patients. S0-3, hepatic steatosis grade 0-3; F0-4, fibrotic stage 0-4.

Authors' contribution

Conception and design: Ming-Lung Yu. Acquisition of data: Chung-Feng Huang, Chia-Yen Dai, Ming-Lun Yeh, Jee-Fu Huang, Wan-Long Chuang and Ming-Lung Yu. Data analysis and interpretation: Chung-Feng Huang and Ming-Lung Yu. Manuscript drafting and critical revision: Chung-Feng Huang, Jee-Fu Huang and Ming-Lung Yu. Approval of the final version of the manuscript: Jee-Fu Huang and Ming-Lung Yu.

Conflicts of Interest

The authors have no conflicts to disclose.

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Abbreviations:

CHC, chronic hepatitis C; CMRF, cardiometabolic risk factor; EASL, European Association for the Study of the Liver; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; MASLD, metabolic dysfunction-associated steatotic liver disease; SLD, steatotic liver disease; SVR, sustained virological response

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