

## Reply to the letter by H. Mawatari et al. regarding “Thrombocytopenia is more severe in patients with advanced chronic hepatitis C than B with the same grade of liver stiffness and splenomegaly”

Hitoshi Ikeda · Kazuaki Tejima · Ryota Masuzaki ·  
Yutaka Yatomi · Kazuhiko Koike

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We thank Mawatari et al. for their interest in our article [1] and their presenting data regarding the platelet count in patients with hepatitis C virus-related chronic liver disease (CLD-C) and in those with nonalcoholic fatty liver disease (NAFLD) (please see Letter to the editor). The usefulness of transient elastography for the detection of liver fibrosis in NAFLD has been well established [2, 3], although the controversy still exists [4]. We had an impression in the clinical setting that thrombocytopenia might be less severe in patients with NAFLD than in those with CLD-C, and this point has been clearly determined in their study with adjustment for the degree of liver stiffness. Considering the mechanism of the decrease in the platelet count, we are interested in the splenomegaly in their patients with CLD-C and NAFLD. If the spleen size is not different in patients with the same degree of liver stiffness, the impaired platelet production rather than the enhanced platelet destruction may explain the distinct thrombocytopenia between CLD-C and NAFLD as well as hepatitis B virus-related chronic liver disease and CLD-C [1]. Nonetheless, it has become more likely that thrombocytopenia in chronic liver disease may vary according to its etiology.

Thrombocytopenia is still a big problem in chronic liver disease, considering the interferon therapy for CLD-C or the treatment for hepatocellular carcinoma frequently arisen from chronic liver disease. Its mechanism should be further investigated.

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H. Ikeda (✉) · K. Tejima · R. Masuzaki · Y. Yatomi ·  
K. Koike  
Tokyo, Japan  
e-mail: ikeda-1im@h.u-tokyo.ac.jp