

Letters to the Editor

Could viral load combined with indirect serum markers be an option for predicting the degree of liver fibrosis in treatment-naïve chronic hepatitis B patients?

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Dear Editor,

In a recent issue of the *Revista Española de Enfermedades Digestivas*, we read with interest the article by Coskun et al. (1) “The diagnostic value of a globulin/platelet model for evaluating liver fibrosis in chronic hepatitis B patients”. However, we wanted to emphasize some points about the article.

Initially, correlations between APGA (aspartate aminotransferase/platelet/gamma glutamyl transferase/alfa fetoprotein), globulin/platelet index or FIB-4 index and fibrosis scores ($r = 0.53$, $r = 0.42$ and $r = 0.41$, respectively, and $p < 0.001$ for all) were described as a strong correlation by the authors. When interpreting the results of correlation analysis, both the r value and p value must be considered. If there is a statistically significant difference, the closer the r value is to 1 or -1, the correlation is considered to be stronger. Thus, we believe that it would have been better if r values between 0.40 and 0.60 had been defined as a moderate correlation.

Secondly, the scoring systems including indirect serum markers have also been reported to not be associated with significant fibrosis and also to have better accuracy in the diagnosis of cirrhosis rather than significant fibrosis in patients with hepatitis C virus (HCV) infection, rather than HBV infection (2-5). Further-

more, although serum HBV deoxyribonucleic acid (DNA) levels were not evaluated and mentioned in the article, this has been considered as more accurate to predict fibrosis degree than the models in light of the complex natural history of chronic HBV infection (3). Moreover, higher serum HBV DNA levels and also the presence of precore/core promoter HBV variants have been defined as predictors or risk factors of significant liver fibrosis in HBV patients (2,5).

Consequently, we think that better results for predicting the severity of liver fibrosis may be achieved by using serum HBV DNA levels combined with indirect serum markers in treatment-naïve HBV patients, but randomized large-scale studies are required.

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