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Noninvasive liver fibrosis assessment: Why does the APRI not work for hepatitis B?

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Dear Editor,

We read the article by Yilmaz *et al.* with interest (1). Chronic liver disease requires the regular assessment of fibrosis to risk-stratify patients to treat and monitor the complications of liver cirrhosis (2). Due to the overwhelming burden of liver disease worldwide, there is a patent need for noninvasive methods of assessing fibrosis to enable us to test large numbers of patients regularly. The aspartate transaminase-to-platelet ratio index (APRI) is valuable, primarily due its universal availability, simplicity, and, hence, ease of use (1). The reliability of the APRI in hepatitis C and nonalcoholic fatty liver disease (NAFLD), as reported by Yilmaz *et al.* should broaden its application in these conditions. As in many studies, APRI was less reliable for hepatitis B (3-5). Yilmaz *et al.* attribute the failure of APRI to adequately predict fibrosis to platelet count, claiming that both hepatitis C and NAFLD are associated with lower platelet counts than hepatitis B. In their own cohort, however, this was not the case, as the mean platelet counts were 224,714, 221,851, and 249,871 for hepatitis B, hepatitis C and NAFLD, respectively.

We wonder whether the true difference is due to the natural history of the three diseases. Whereas hepatitis C and NAFLD tend to have a steadier course of chronic inflammation and relatively stable aspartate transaminase (AST) levels, AST levels in hepatitis B fluctuate due to flares. During a flare (high AST level), AST levels can normalize during times of low viral replication. Fibrosis, however, could already have occurred during the earlier flare. Yet, APRI levels would have decreased from high to normal as AST levels normalized. APRI would therefore modulate while the underlying fibrosis remained unchanged. APRI might be more suited to predict fibrosis in slowly and continuously progressing diseases of the liver rather than those that are associated with flares.

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Authors' Reply: Noninvasive liver fibrosis assessment; Why does the APRI not work for Hepatitis B?

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Dear Editor,

We thank Dr. Selinger and Leong for their cogent comments on our manuscript (1). The authors reasoned that one mechanism whereby the APRI appears to be of limited clinical utility in hepatitis B could be episodes of increased viral replication, or "flares," associated with increased immune activity and subsequent fluctuations in liver transaminase levels. This potential mechanism is attractive, because typical HBsAg-positive chronic HBV infections are often characterized by unpredictable flares in viral replication, immune activity, and liver damage (6). Starting from these premises, it is clear that the APRI may be influenced by falsely high values in patients with AST flares and falsely low values in patients with viral suppression and normalization of transaminases. Given the fluctuating nature of chronic HBV infections (7), the confounding effect of AST flares on APRI measurements is likely to be more pronounced in patients with hepatitis B than in those with hepatitis C. To address this vexing clinical issue, additional research is warranted to determine the clinical settings in which liver stiffness measurements can accurately predict liver fibrosis and to establish cutoff values for differentiating the stages of fibrosis and cirrhosis. These studies should also compare the performance of the APRI in patients with varying degrees of necroinflammation, and untreated patients and patients who are receiving antiviral therapy.

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