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**Letter to the Editor:**

Should We Really Take Anti-Viral Therapy into Account in Chronic Hepatitis B Patients with Normal Liver Function?

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Dear Editor-in-Chief

We read with a great interest the article written by Lee and colleagues entitled "Hepatocellular Carcinoma Risk of Compensated Cirrhosis Patients with Elevated HBV DNA Levels according to Serum Aminotransferase Levels" which was recently published in your Journal of Korean Medical Science [1]. The authors concluded the beneficial effects of prescribing appropriate anti-viral therapy (AVT) for reducing HCC risk in cirrhotic patients with elevated HBV DNA and normal aminotransferase levels. They have done a valuable work with an appropriate sample size which has considered a really challenging and important subject in chronic hepatitis B (CHB) patients. Despite our interest to the results of the Lee et al. study there are some challenging points about their work; so some comments may be of benefit. The first, the authors have claimed that high levels of ALT were associated with higher risk of HCC in patients with high HBV DNA level. Regarding available data, there is an overlap between high ALT and high HBV DNA levels in their study where they both increase the chance of HCC independently [2]. Also high ALT level may be related to high HBV DNA level or possible non-alcoholic fatty liver (NAFLD) in the study individuals that will enhance the risk of HCC [3]. Due to limitations of retrospective studies, no information was provided regarding patients' BMI by Lee et al. for considering this subject in their study. Another important issue is that the authors have concluded that AVT duration is associated with lower HCC risk in patients with normal aminotransferase levels and suggested prompt AVT to be considered in these patients. This conclusion involves providing more evidence since there are efficient data on higher risk of HCC in chronic hepatitis B patients with high ALT and HBV DNA levels. However, it is difficult to conclude

that AVT can have protective effect on HCC in patients with normal ALT level, high HBV DNA level and without liver damage; as it has been shown that these patients could be considered as HBV inactive carriers (IC) regarding treatment [4]. On the other hand, it was concluded that treatment naïve patients who underwent AVT may have even higher risk of HCC development than patients with inactive stage CHB [5]. Previous data have clarified the role of HBs Ag in differentiation of CHB from IC patients; however it was mentioned not to be cost-effective [6]. It was concluded that CHB patients had higher levels of HBV DNA, HBs Ag and ALT levels in comparison with IC patients [6]. In conclusion we appreciate the valuable effort of the authors, however we are wondering if we could kindly ask them to interpret better our concerns.

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