

Role of Serum Hepatitis B Virus Marker Quantitation to Differentiate Natural History

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Rec Date: Oct 19, 2015; Acc Date: Dec 10, 2015; Pub Date: Dec 22, 2015

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Commentary

In the past decade, a growing body of evidence has shown that HBsAg quantification (qHBsAg) not only is a useful marker for monitoring natural history of treatment-naïve Chronic HBV Infection (CHB) but can also predict clinical and treatment outcomes [1]. While, other study indicated that, being used alone, qHBsAg is not a suitable marker for evaluating hepatitis activity and distinguishing between cases of HBeAg-negative CHB and inactive HBV carrier state [2]. In our study, we showed that qHBsAg had high predictive value for discrimination of immune tolerance (IT) and Immune Clearance (IC) phase, and it had moderate predictive value for differentiation of low replication (LR) phase and HBeAg Negative Hepatitis (ENH) [3]. While, role of HBeAg quantification (qHBeAg) in natural phases of HBV infection has not been attracted more attention up to now. We found that in HBeAg positive phase of treatment-naïve CHB, qHBsAg and qHBeAg correlated positively ($P < 0.0001$), and both had strong negative correlation with grade of liver inflammation (G) ($P < 0.0001$) and Fibrosis stage (F) ($P < 0.0001$) (unpublished data). Thus, we might speculate that qHBeAg can either be used as a marker of natural phases of HBV infection in HBeAg positive patients. Indeed, we demonstrated that qHBeAg had moderate predictive value for discriminating IT and IC phase [3]. And qHBsAg and qHBeAg had moderate predictive value for F1, F2 and F3 and for G2 and G3 in treatment-naïve HBeAg positive CHB (unpublished data). In recent years, HBcAb quantification (qHBcAb) has been indicated to be associated with HBV infection induced hepatitis [4]. We also suggested

that qHBcAb are higher in IC and ENH phases than in IT and LR phases. And qHBcAb correlated positively with grade of liver inflammation. Whereas, the qHBcAb has low divisional value for the intermediate grades of inflammation (unpublished data). Next, we will determine whether qHBcAb is different with liver injury of different etiologies in HBV infection, including HBV infection merged with non-alcoholic fatty liver disease or combined with drug induced liver injury. In all, qHBsAg had high and qHBeAg had moderate predictive value for discrimination of IT and IC phase. And both had moderate predictive value for diagnosis of mild to severe liver fibrosis in treatment-naïve HBeAg positive CHB. Both qHBsAg and qHBcAb had moderate predictive value for differentiation of LR and ENH phase.

References

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