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UTILITY OF HBSAG QUANTIFICATION IN DEVELOPING AN INDIVIDUALISED APPROACH TO THE MANAGEMENT OF CHRONIC HEPATITIS B VIRUS IN CLINICAL PRACTICE

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Introduction HBsAg is a surrogate marker of cccDNA, its loss is associated with sustained immune control and reduction

in the development of HCC. HBsAg quantification (qHBsAg) has facilitated the on-treatment monitoring of response to pegylated interferon α (IFNα) with the potential to individualise therapy according to response. HBsAg decline with oral antivirals (OAV) is reported to be a slower process; however any reduction in HBsAg could potentially be used as a tool to determine duration of therapy, in lieu of recommended lifelong treatment. This study examined the utility of HBsAg quantification in developing individualised treatment strategies for both IFNα and OAV treated patients.

Methods 86 CHB patients (male=72) were included for analysis. All patients underwent qHBsAg (ABBOTT Architect) in addition to longitudinal measurement of ALT and HBV DNA. 60 patients (male=54), median age 45 (range 21–70), were treated with combination Tenofovir and Lamivudine; qHBsAg was recorded at baseline, 12 and 24 months. 26 patients (male=18), median age 31 (range 18–55), were treated with IFNα. HBsAg was quantified at baseline, 12 and 24 weeks. HBsAg decline was compared in the OAV and IFNα groups at 12 and 24 months versus 12 and 24 weeks respectively.

Results In the OAV group, baseline median ALT 42.5 (range 15–181) and median HBV DNA 3.43 log (range 1.5–7.9). At 12 months, ALT normalisation was achieved in 60% and undetectable DNA in 90%. Mean HBsAg decline in this group was 0.06 log at 12 months. ALT normalisation and viral suppression was sustained at 24 months. HBsAg decline, however, was not sustained and there was an overall mean increase of 0.1 log from baseline. In the IFNα group, baseline median ALT 113.6 (range 29–448) and median HBV DNA 7.21 log (range 3.58–9.25). HBsAg decline was measured 12 weekly. 8/26 (31%) patients had a >0.5 log decline at 12 weeks and this increased to 45% at 24 weeks. HBsAg decline was significantly greater in the IFNα group when compared with the OAV group at 12 weeks versus 12 months and more marked at 24 weeks versus 24 months (p=0.03 and p=0.006 respectively).

Conclusion These data highlight the utility of qHBsAg in IFNα therapy and raises the possibility of an individualised, response guided approach allowing early changes in treatment regimen if indicated. Consistent with published data, the 24-week HBsAg is a better predictor of sustained immune control. Conversely, there was a limited HBsAg decline in OAV treated patients over prolonged follow-up and this reduction was not sustained. This suggests that HBsAg quantification alone cannot be used to individualise therapy and determine treatment duration with OAV's.

Competing interests None.

Keywords chronic hepatitis B (CHB), oral antiviral (OAV), pegylated interferon α (PEG-IFNα), surface antigen quantification (qHBsAg).