Regimens and outcomes for people with HIV and known hepatitis B core antibody positivity

Alif Aziz[1], Sanjay Bhagani[2], Alan Hunter[2], Tabitha Mahungu[2], Tristan J Barber[2,3]

UCL Medical School[1], Royal Free Hospital Ian Charlson Day Centre[2], UCL Institute for Global Health[3]

Background

In people with HIV who have markers indicative of previous hepatitis B virus (HBV) infection reactivation is a concern, particularly in those without significant surface antibody response (sAb). With newer HIV regimens containing fewer agents becoming more widely used, we assessed management and outcomes of our anti-HBc+ cohort. Annual blood tests at our centre include HBsAg and anti-HBs status. HBV reactivation was defined as HbsAg+ and/or HBV DNA+ at any stage.

Methods

From our HIV clinic cohort, all patients who were anti-HBc+ and sAg- from 2015 onward till 1st December 2021 were identified and demographic and HIV related data was collected: (diagnosis date, recent CD4, HIV VL), hepatitis markers and current antiretroviral regimen (ART). For those who had evidence of HBsAg or HBV DNA positivity, a notes review was conducted. (i.e. those without HBV sAb>10 and not receiving TXF+XTC). We collected data on most recent HBsAg and HBV DNA sampling, noting indicators of HBV reactivation.

Results

530 individuals identified (17%; total HIV cohort 3133). 367 male, 163 female. Mean age 54yrs (IQR:48-61). 328/530 (61.9%) on TXF+XTC, 156 (29.4%) receiving one agent active against HBV (TXF or XTC), 46/530 (8.7%) receiving ART that did not contain either. Median time from data censoring to last follow-up blood tests 8 months (IQR: 4-9).

Group A2 consists mostly of patients with previously identified chronic HBV (i.e. previously HBsAg+) who have appropriately maintained TXF/XTC.

HBV reactivations were not seen in any patients in groups B or C. A planned reanalysis will determine the PYFU for each group.

Data at baseline are inconsistently recorded due to the limitations of our electronic database prior to 2015. Group A contains clearly contains some participants with chronic hep B being appropriately managed on two active agents.

However testing for HBV DNA was only available in 24/530 (4.5%) overall and in only one person from the 1 HBV agent and 0 HBV agent subgroups.

Figure 1: Flow Diagram

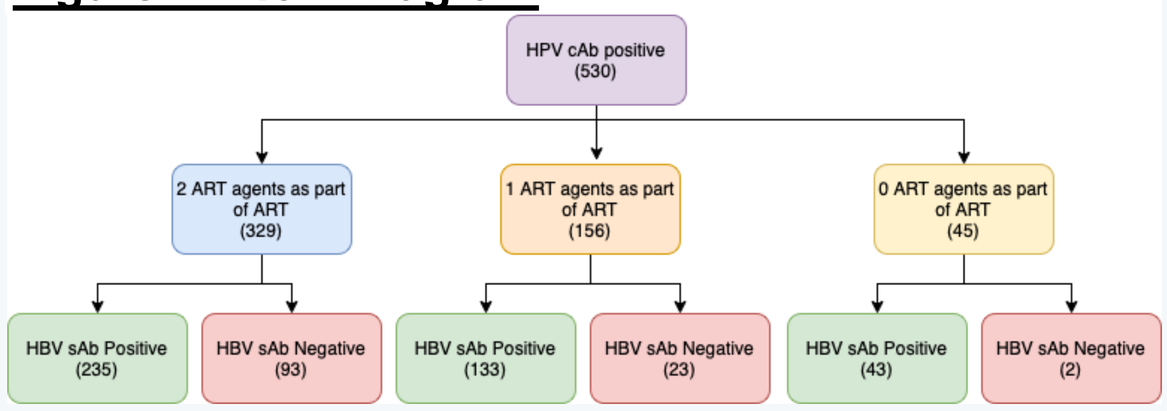

Table 1

	2 HBV agent as part of ART (A)	1 HBV agent as part of ART (B)	0 HBV agents as part of ART (C)
Group 1: HBV sAb positive (>10mIU)	235	133	43
HBsAg+	0 (0%)	0 (0%)	0 (0%)
HBV DNA+	3/236 (1%)	0 (0%)	0 (0%)
Group 2: HBV sAb negative	93	23	2
Number (%) HBsAg+	49 (53%)	0 (0%)	0/2
HBV DNA Tested	23	1	0
HBV DNA+	20/23 (87%)	0/1 (0%)	0 (0%)

Conclusion

It is reassuring that in the people living with HIV who are anti-HBc+, there were no documented incidences of HBV reactivation in those receiving single-HBV active agent ART or no HBV active agent ART. Ongoing prospective follow-up with regular HBsAg and HBV DNA testing are required, especially on those who are receiving less than 2 active agents for HBV as part of their HIV regimen, before recommendations in guidelines can be changed. We note HBV DNA testing was performed in only 24 (5%) patients in the cohort presented here.

Email: alif.aziz.19@ucl.ac.uk

Royal Free London NHS Foundation Trust