less likely to be tested for HBV than for HIV VL in the first six months (21% vs 79%, \( P < 0.0001 \)) and eight times less likely in the last six months of observation (6% vs 49%, \( P < 0.0001 \)). Comparing the first six months to the last six months of observation, the proportion of patients tested for AST (77% and 50% \( P < 0.0001 \)) and platelets (76% and 57% \( P < 0.0001 \)) declined.

**Conclusion.** Adherence to the DHHS management guidelines for monitoring HBV VL among HIV/HBV co-infected patients was low. AST and platelet counts were monitored at a similar frequency to HIV VL, suggesting that these markers may have been checked as part of routine HIV care rather than for HBV monitoring. Focusing on adherence to guidelines may ensure early detection of complications and provide patients with timely and appropriate care.

**Disclosure.** All authors: No reported disclosures.

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**2221. Markers of Cirrhosis and Inflammation in HIV/HBV Co-infection in a Ugandan Cohort**

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**Session:** 244. HIV and HBV

**Background.** Co-infection with HIV and hepatitis B virus (HBV) is common, reaching 23% in Uganda, and can accelerate HBV disease progression. Inflammation contributes to the pathogenesis of both viruses. We compared serological biomarkers of inflammation and FIB-4 scores (to predict liver fibrosis) stratified by HIV and HBV status in a Ugandan cohort.

**Methods.** Subjects with HIV/HBV co-infection were matched up to 2:1 by age and gender with HIV-monoinfected, HBV-monoinfected, and uninfected controls from the Ugandan site of the African Cohort Study (AFRICOS). Demographic and laboratory characteristics, including inflammatory biomarkers, were compared between the groups. FIB-4 scores were stratified as <1.45, 1.45–3.25, and >3.25 for those unlikely to have advanced fibrosis, indeterminate, and likely to have advanced fibrosis, respectively (resp.), in the HIV/HBV and HIV groups, which had these data available.

**Results.** Within the Ugandan subset of AFRICOS, 31 HIV/HBV co-infected patients were available and compared with 62 HIV-monoinfected, 7 (all that were available) HBV-monoinfected, and 62 uninfected subjects. Median age was 37 (range 19–67) years and 78% were male. The HBV group, as compared with the HIV/HBV, HIV, and uninfected groups, had higher prevalence of hepatitis C antibody (29%, 6%, 2%, 3%, resp.; \( P = 0.04 \)), fewer other active infections (0%, 48%, 52%, 26%, resp.; \( P = 0.002 \)), fewer non-antiretroviral medications (median 0, 1, 2, 0, resp.; \( P < 0.001 \)), and a smaller proportion on an antiretroviral (0%, 32%, 37%, 18%, resp.; \( P = 0.03 \)). The HIV/HBV group had generally higher levels of inflammation overall and statistically had significantly higher levels of MMP-12 and lower levels of FGF-23 compared with the other groups (Figure 1). The HIV/HBV group had a lower proportion of subjects unlikely to have advanced fibrosis by FIB-4 (Figure 2; \( P = 0.046 \)).

**Conclusion.** Elevated MMP-12 in the HIV/HBV group suggests that elastin degradation may be a mechanism contributing to the accelerated progression of liver disease seen in co-infection. Prior literature demonstrated that FGF-23 is elevated in end-stage liver disease and predicts mortality, but we did not observe higher levels in HIV/HBV co-infection in this cohort with little advanced liver disease. Further studies are needed to characterize the inflammatory milieu and evaluate the impact of time and treatment of HIV/HBV.

**Disclosure.** T. A. Crowell, Gilead Sciences: Speaker, Speaker honorarium

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**2222. Calculated Globulin Adds Predictive Value to Hepatitis B Vaccine Response in HIV-infected Persons Independently of HIV Viral load and CD4 Cell Count**

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**Session:** 244. HIV and HBV

**Background.** Response rates to hepatitis B (HBV) virus vaccine are low compared with the general population. Recent data suggest baseline total IgG levels add...
predictive value for vaccine failure. We retrospectively analyzed the relationship of calculated globulin (CG) levels with HBV vaccine response in the U.S. Military HIV Natural History Study (NHS).

Methods. NHS is a longitudinal observational cohort of DoD active duty and beneficiaries with HIV infection, enrolling since 1986. Inclusion criteria was: (1) no current or past HBV or hepatitis C infection; (2) HBV vaccination after past positive HIV date; (3) available post-vaccination follow-up serum HBV surface antibody (HBsAb) test; (4) CD4 cell count, HIV RNA viral load (VL), and protein levels within 90 days prior to the last vaccine dose. Using a standard approach, CG levels were derived by subtracting the albumin level from total protein. Variables were analyzed using uni- and multivariate logistic regression model.

Results. Data from 674 eligible participants were analyzed. Subjects were 87% male, 44% Caucasian, 41% African-American. At time of last vaccine dose, median values were age, 34 yrs; CD4 cells/μl, 515; nadir CD4 cells/μl, 318. 51% were receiving ART and VL was <400 copies/ml in 51%. Overall, HBV vaccine response rate was 54%. Among CG quartiles, HBV vaccine response was 70%, 60%, 40% and 44% from lowest to highest quartile respectively (P < 0.001). In the multivariate analysis, CD4 cell count and CG levels at time of last dose independently predicted successful HBV vaccine response in HIV-infected persons. These data suggest B-cell dysfunction, characterized by higher CG levels, may be clinically significant regardless of VL and CD4 cell count.

Conclusion. CG levels at time of last dose independently predicted successful HBV vaccine response in HIV-infected persons. These data suggest B-cell dysfunction, characterized by higher CG levels, may be clinically significant regardless of VL and CD4 cell count.

Table 1. Comparison of laboratory characteristics between coinfected and mono-infected patients

| Characteristics | HIV+ (n = 471) | HIV+ / HBsAg* (n = 47) | p |
|-----------------|----------------|------------------------|---|
| WHO Clinical Stage |                |                        |   |
| Stage I (%)     | 210 (45.5)     | 22 (46.8)              |   |
| Stage II (%)    | 138 (29.9)     | 14 (29.8)              |   |
| Stage III (%)   | 109 (23.6)     | 10 (21.3)              |   |
| Stage IV (%)    | 4 (0.9)        | 1 (2.1)                | 0.848 |
| Median platelets | 226 (177 – 269) | 212 (176 – 268) | 0.278 |
| Median CD4+T cell count, cells/mm3 (iQR) | 363 (204 – 508) | 327 (131 – 462) | 0.203 |
| Median ALT (iQR) | 21.4 (16.0 – 30.1) | 26.2 (18.0 – 35.0) | 0.054 |
| Median AST (iQR) | 28.8 (22.1 – 38.1) | 29.4 (26.0 – 39.8) | 0.244 |
| HBV DNA, IU/mL | 1484 (224 – 727292) | 10 (2137) | 0.0007 |
| Median (iQR) | 1484 (224 – 727292) | 10 (2137) | 0.0007 |
| < 20 (%) | 45 (95.7) | 10 (21.7) | 0.0007 |
| ≥ 20 – < 20 000 (%) | 44 (95.6) | 2 (4.3) | 0.0007 |
| ≥ 20 000 (%) | 44 (95.6) | 2 (4.3) | 0.0007 |
| APRI > 2.0 | 1.22 (1.14 – 1.31) | 1.13 (1.04 – 1.22) | 0.0007 |
| CD4 cell count (per 100 cells) | 0.68 (0.60 – 0.77) | 0.77 (0.65 – 0.97) | 0.0007 |
| Gender | 1.21 (1.04 – 1.43) | 0.94 (0.77 – 1.13) | 0.0007 |
| Non-white race | 1.11 (0.81 – 1.51) | 1.21 (1.03 – 1.76) | 0.0007 |
| >3 vaccine doses | 1.71 (1.26 – 2.37) | 1.09 (0.79 – 1.59) | 0.0007 |
| CD4 cell at last vaccine | 1.22 (1.14 – 1.31) | 1.13 (1.04 – 1.22) | 0.0007 |
| Stage IV (%) | 4 (0.9) | 1 (2.1) | 0.848 |
| Median platelets | 226 (177 – 269) | 212 (176 – 268) | 0.278 |
| Median CD4+T cell count, cells/mm3 (iQR) | 363 (204 – 508) | 327 (131 – 462) | 0.203 |
| Median ALT (iQR) | 21.4 (16.0 – 30.1) | 26.2 (18.0 – 35.0) | 0.054 |
| Median AST (iQR) | 28.8 (22.1 – 38.1) | 29.4 (26.0 – 39.8) | 0.244 |
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| Non-white race | 1.11 (0.81 – 1.51) | 1.21 (1.03 – 1.76) | 0.0007 |
| >3 vaccine doses | 1.71 (1.26 – 2.37) | 1.09 (0.79 – 1.59) | 0.0007 |
| CD4 cell at last vaccine | 1.22 (1.14 – 1.31) | 1.13 (1.04 – 1.22) | 0.0007 |

Disclosures. All authors: No reported disclosures.

2224. Longterm Evaluation of Hemoglobin A1c Following Hepatitis C Therapy in Patients with and without HIV Co-infection

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Background. Historically, eradication of HCV with interferon-based treatments was linked with decreased incidence of diabetes. While viral clearance with direct acting agents (DAAs) may be associated with acute decreases in fasting glucose levels and hemoglobin A1c (HbA1c), there remains a need for larger prospective studies to address the longterm impact of sustained viral response (SVR) achieved with DAAs on glucose metabolism.

Methods. Prospective longitudinal cohort study of 251 subjects with chronic HCV (100% genotype 1a/b, 31% HIV+, 17% diabetes) evaluated pre- and post-treatment with median follow-up of 28 mos. Change in HbA1c, glucose, lipid and transaminase levels were compared based on SVR, HIV, diabetes status and fibrosis stage.

Results. There was no difference in change in HbA1c between subjects who achieved SVR (n = 241) compared with those who did not. Mean change in HbA1c did not differ from zero (−0.022 ± 0.53%) for those with SVR. Further, when subjects were grouped based on HIV, diabetes or fibrosis stage, there were no significant differences in changes in HbA1c or glucose following SVR. Subjects with HIV had smaller reductions in transaminase values (change ALT −33.3 ± 51 IU/L HbA1c vs. −47.8 ± 45 IU/L HbA1c, P = 0.0007). Following SVR, total and LDL cholesterol increased (P = 0.0002 and P = 0.0003, respectively) whereas triglyceride levels decreased (P = 0.0086).

A greater proportion of subjects (7%) started or increased medication therapy for diabetes following SVR compared with the percentage who decreased diabetes therapy (3%). There was a statistically significant positive correlation between change in BMI and change in HbA1c (r = 0.17, P = 0.006).

Conclusion. The current study failed to identify sustained benefits in glucose or HbA1c in HCV treated patients, irrespective of HIV, diabetes or fibrosis stage. HIV infection blunted improvements in transaminase levels related to SVR. While HbA1c...