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Outcome of chronic hepatitis delta in patients with and without HIV infection in the HAART era

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Background

Around 6–8% of patients with HIV infection are co-infected with hepatitis B virus (HBV) in the Western world. At least 10% of them are superinfected by hepatitis delta virus (HDV). The impact of HIV on liver fibrosis progression in patients with HBV or HDV chronic hepatitis in the HAART era is still uncertain.

Methods

A retrospective case-control study was conducted at our institution comparing the progression of liver fibrosis in patients with HBV or HDV with and without HIV co-infection. Clinical and laboratory outcomes were recorded. Liver fibrosis was measured by transient elastometry (FibroScan®) and by FIB-4. Values of FibroScan above 9.5 kPa and values over 3.25 for FIB-4 were considered as diagnostic of advanced liver fibrosis (F3–F4 Metavir score estimates). Patients with positive serum HCV-RNA were excluded from this analysis.

Summary of results

A total of 26 HDV-infected patients were included in the study, 16 HIV-positive as cases and 10 HIV-negative as controls. Median follow-up was 4 years (3–5). Main demographics: mean age 43/40 years and 68.4%/31.4% males, respectively. Advanced liver fibrosis was recognized in 40% of cases in both groups using FibroScan. It was 43% and 57%, respectively, using FIB-4 ($p = 0.38$). HDV genotype D and A were the most prevalent in both groups, without significant differences in prevalence rates. Although mean time of HBV infection and mean serum HDV-RNA levels were similar in both groups (19 vs. 18 years, $p = 0.7$; 13 vs. 9 log10, $p = 0.6$), hepatic decompensation events occurred more frequently in HIV-negative than HIV-positive patients (6 vs. 2, $p = 0.08$). Two HIV-positive patients were lost to follow-up and one died from lymphoma. All HIV-HDV co-infected patients were under tenofovir-containing HAART regimens, while only 4/10 patients in the control group had received any treatment (interferon) during the study period.

Conclusion

The prognosis of chronic hepatitis delta seems to be worst in HIV-negative individuals than in HIV co-infected patients on antiretroviral therapy. Inhibition of HBV replication by using tenofovir might indirectly ameliorate delta pathogenicity.