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HIV lamivudine resistance mutations in HBV co-infected Romanian patients

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Purpose of the study

Previous studies of our team have shown that Romanian teenagers horizontally infected with HIV in early childhood, have a high prevalence of HBV co-infection (78.3% being anti-HBc positive vs. 21.8% of age-matched HIV-negative controls). Our aim was to investigate the frequency of mutations and variables potentially associated with an increased risk of liver disease evolution in HIV/HBV co-infected individuals receiving 2NRTI + 1PI boosted with ritonavir.

Methods

We investigated 38 HIV+ adolescents (mean age 13.8 ± 1.3 years) with constant detectable HBV viral load, of whom 92.3% were chronic HBsAg carriers, 61.53% were HBeAg+, and 68.2% had lamivudine (LAM) in their regimen. HBV genotype was determined using commercial Line Probe Assay (Innogenetics) and sequencing of the HIV pol gene was carried out using Trugene genotyping kits (Bayer Diagnostics Inc.); for HIV subtyping the nucleotide sequences were submitted to the Stanford database.

Summary of results

All patients were infected with the HIV F1 subtype, while HBV genotype A was the most prevalent (84.6% of cases). A small percentage of patients presented other HBV genotypes (7.7% D and 7.7% both A and D). In spite of the long evolution of HIV infection only 23.07% of patients had worsened HIV-related parameters (CD4 count less than 100 cell/μL, average HIV titer above 750,000 copies RNA HIV/ml), associated with increased transaminases levels (average values 72 mg/dl). No correlation between HIV viral loads or the degree of immunosupression and HBV viral load was found. 75% of the patients with LAM exposure vs. 55.5% patients without LAM were HBeAg+. HIV pol gene sequencing demonstrated that only 10 patients had a high degree of resistance to LAM, including those who had never received this drug. The most common mutation in the RT gene was M184V/I (that causes high-level resistance to LAM, but also confers a diminished HIV replicative fitness); associated with type 2 TAMs: D67N, K70R, T215F, and K219Q/E that confer NRTI resistance.

Conclusion

We suggest that association of HIV F subtype and genotype A of HBV may generate a less severe evolution of HBV infection irrespective of the LAM treatment with less frequent mutations in pol HIV gene. The absence of type 1 TAMs opens the possibility to treat these patients with tenofovir-containing regimens with a good virological response. Further studies are needed to clarify the kinetics and significance of different mutation patterns observed in naïve vs. LAM treated HIV/HBV co-infected patients.