

POSTER PRESENTATION

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Relationship between dynamics of Epstein-Barr virus and immune activation in HIV-1 infected subjects in the HAART era

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Purpose of the study

HAART has greatly modified the course of HIV-1 infection; however, its impact seems to be less favourable on lymphoproliferative disorders associated with Epstein-Barr virus (EBV) than on other AIDS-defining illnesses. The aim of this study was to estimate the relationship between EBV levels and other viro-immunological parameters in HIV-1 infected subjects in the HAART era.

Patients and methods

164 HIV-1 infected patients (pts) who attended the Infectious Diseases Unit of Rovigo Hospital, from July 2007 to December 2009 were included in this study. 28% of patients had HBV and/or HCV coinfections. HIV-1 RNA in plasma was quantified by COBAS Taqman HIV-1 test. HIV-1 DNA and EBV-DNA in peripheral blood mononuclear cells (PBMC) were determined by real-time PCR. Lipopolysaccharide (LPS), a marker of microbial translocation, was determined in plasma samples using a chromogenic assay (Limolus Amebocyte Lysate). B-cell activation was analyzed by flow cytometry using monoclonal antibodies CD19PerCP, CD86APC, and CD69PE.

Results

The median (IQR) EBV-DNA load was 41(1-151) copies/105 PBMC. 48% of pts had CD4 >500 cells/ μ l and 27% had undetectable HIV viral load. The EBV-DNA level was significantly higher in pts with CD4

below 500 cells/ μ l than in those with CD4 >500cells/ μ l [72(14-324) vs 18 (1-80) copies/105; $p < 0.0001$] and in pts with detectable HIV-1 RNA than in those with undetectable viremia [49(7-315) vs 17(1-55) copies/105; $p = 0.001$]. Levels of EBV-DNA were higher in the group of pts with CD4 cell counts >500 cells/ μ l and high HIV-1 viremia (>1000 copies/ml) than in pts with low viremia, regardless of the immunological status [48 (5-153) vs 18(1-60); $p = 0.015$]. EBV-DNA was also significantly higher in pts with coinfections than in pts with no coinfections [85(10-527) vs 33(1-114); $p = 0.003$]. Furthermore, pts with high EBV loads (up to 75th percentile) had higher levels of HIV-1 DNA [40 (1-132) vs 10(1-76) HIV-DNA copies/105; $p = 0.050$] and higher levels of LPS [130(88-244) vs 98(81-134) pg/ml; $p = 0.024$] than pts with low EBV loads. B-cell activation in pts with high EBV loads was confirmed by immunophenotyping; two of these pts developed a B-cell lymphoma.

Conclusions

These findings suggest that HIV-1viremia, other coinfections, and immune activation play an important role in the B-cell stimulation and expansion of EBV-infected cells. Persistent HIV-1 viremia, despite immunorestitution, may represent a risk factor for the onset of EBV-related cancers.

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