Poster presentation

Is HAART based on newest active antiretroviral drugs influenced by GSS?

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Purpose of the study
Treatment-experienced HIV-infected patients with limited therapeutic options continue to receive a partially suppressive treatment regimen pending the availability of two new antiretroviral drugs. We analyzed the viro-immunological and resistance data of 38 outpatients.

Methods
Major enrollment criterion was the administration of maraviroc, enfuvirtide, raltegravir, etravirine, darunavir/r or tipranavir/r, alone or in combination, in the latest antiretroviral regimen, decided upon the last genotypic RNA resistance test. This allowed us to assess the genotypic sensitivity score (GSS) at the same time. We also recorded previous presence of specific mutations in all available genotypic resistance tests, their persistence in time and their correlation to the last GSS.

Summary of results
Undetectable viral load was obtained in 64% of patients without difference between GSS classes (HIV-RNA median value <50 cp/ml in any class); we also reported a mild increase in CD4 count (6 months median of 290/μl for GSS1, 299/μl for GSS2 and 367/μl for GSS3). This was not significantly related to GSS considering either GSS1 vs. 2 vs. 3 or GSS1 vs. 2 plus 3. Our data showed persistence of specific mutations for NRTI: 184IV, 210W, 215FY, 219EQ (over 50% in every test) and 67N in almost 70% of all tests; for NNRTI: 103N and 181CIV in over 25% of cases with higher tendency to persist for repeated tests; for PI: 10FIRVC, 36ILV, 41L, 46IL, 54VLAMTS, 71OVT, 82AFTSLI, 90M (about 50% in every test) and 63P in almost 70–80% of all cases. This frequency (weighted for the number of tests) was correlated to GSS and revealed a particular pattern for the majority of these mutations: a higher prevalence when related to GSS2 vs. GSS1 and 3. Conversely, NNRTI 181CIV and NRTI 210W showed larger numbers in GSS1 and 3.

Conclusion
Starting an antiretroviral regimen with drugs from the newest active classes can offer viro-immunologic success either with GSS1, 2 or 3. Lack of statistical difference can be explained with a low number of enrolled patients and different viro-immunologic conditions at baseline (higher viral loads and lower CD4 for GSS2 and 3). High frequency for specific mutations confirms the key role of some mutations acquired following the exposure to certain drugs and their persistence through multiple changes of drug regimen. The association between GSS2 and mutation frequency (time weighted) could be linked to patients with better compliance and long persistence of key mutations, which appeared after a long drug exposure.