Highly active antiretroviral therapy for HIV-1 infection: patients' quality of life and treatment adherence

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Chapter

Full adherence to HAART: Is it really necessary? In reply.

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We thank Drs Cainelli and Vento for their interest in our study. They question the necessity of suppressing HIV RNA in plasma to below 5000 to 11,000 copies/mL during antiretroviral therapy to prevent the development of viral resistance and disease progression among asymptomatic HIV-1-infected patients with reasonably maintained CD4 cell counts. Consequently, they also question the necessity of full adherence to HAART in these patients, since this is required for viral suppression.

It is well documented that ongoing viral replication during antiretroviral therapy selects for drug resistant viruses and that persistently detectable viremia is associated with an increased risk of disease progression [1]. Suboptimal adherence to HAART has also shown to be related with HIV-1 disease progression [2].

It is interesting to learn that Drs Cainelli and Vento’s patients who took less than 80% of their prescribed HAART regimen had a mean increase of 121 CD4 cells/μL in 1 year despite a mean HIV RNA level of 11321 copies/mL. Apparently, the level of adherence required for an increase in CD4 cells is lower than that required for viral suppression.

We think it would be interesting to see whether this benefit was maintained over time because there is evidence that immune deterioration is delayed but eventually occurs in most patients who virologically fail on antiretroviral therapy [3].

Drs Cainelli and Vento question the presence of drug resistance as the most important cause for virological treatment failure. We fully agree that non-adherence may also lead to virological treatment failure. The development of resistance is often a consequence rather than a cause of first-line therapy failure. It does, however, compromise future treatment options. Additionally, virological treatment failure may be due to inadequate potency of the antiretroviral treatment and suboptimal plasma concentrations of antiretroviral drugs [4]. Drs Cainelli and Vento recommend to consider non-adherence as the potential cause of virological therapy failure prior to embarking on drug resistance testing. We feel that adherence problems and drug resistance may be considered complementary in cases of virological therapy failure and that absorption problems and drug interactions that adversely affect plasma concentrations of antiretroviral drugs also need to be considered.

Drs Cainelli and Vento propose dual nucleoside analogue reverse transcriptase inhibitor treatment as initial therapy in asymptomatic subjects with a relatively high CD4 cell count to prevent adverse effects and adherence difficulties associated with HAART. We agree that especially in asymptomatic patients with a relatively high CD4 cell count, the potential burden and benefit of antiretroviral therapy need to be carefully weighed. However, we feel that dual nucleoside analogue reverse transcriptase inhibitor treatment may put the patient at risk for developing resistance, which may compromise future treatment options. A recent study showed that disease progression to acquired immunodeficiency syndrome and death clustered among patients starting antiretroviral therapy with CD4 cell counts less than 200/μL [5]. Rather than initiating dual nucleoside analogue reverse transcriptase inhibitor treatment in asymptomatic patients with relatively high CD4 cell counts, one might consider deferring the initiation of antiretroviral therapy in these patients.
References


