

Poster presentation

Effects of highly active antiretroviral therapy (HAART) on platelet activating factor (PAF) metabolism in HIV-infected patients: in vivo results

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Purpose of the study

PAF, a potent inflammatory mediator, seems to play a role in the pathogenesis of several AIDS manifestations. PAF-antagonists have been studied in this context with promising results. We have previously described the in vitro interaction between HAART and PAF-induced platelet aggregation, as well as between HAART and the specific activities of the basic PAF metabolic enzymes.

Methods

In order to examine the in vivo interactions between PAF and HAART, we studied the effect of antiretroviral treatment on PAF-induced platelet aggregation and on the specific activities of the basic PAF metabolic enzymes from blood samples of HIV-patients.

Summary of results

15 naive, male HIV-infected patients were started on HAART. 8/15 received tenofovir-DF/emtricitabine/efavirenz (group A) and 7/15 abacavir/lamivudine/efavirenz (group B), based on our previous in vitro results showing the former having one of the most potent activities against PAF and the latter one of the weaker ones. The in vivo effects of HAART on the specific activities of the basic PAF metabolic enzymes (PAF-CPT, Lyso-PAF-AT and PAF-AH) of plasma, washed human leukocytes (WHLs), and washed human platelets (WHPs) at baseline and after 6 months on HAART were studied. 8/15 patients (four in

each group), who completed 6 months on HAART were included in our final analysis. In group A, a significant reduction (63%) of the specific activity of PAF-CPT of WHLs was observed after 6 months while the specific activities of plasma-PAF-AH, Lyso-PAF-AT of WHLs, as well as both PAF-CPT and Lyso-PAF-AT of WHPs, remained relatively stable. In contrast, in group B, PAF-CPT of WHLs, plasma-PAF-AH, as well as both PAF-CPT and Lyso-PAF-AT of WHPs, remained relatively stable while the specific activity of Lyso-PAF-AT of WHLs was doubled.

Conclusion

Our preliminary in vivo results confirm the previous in vitro ones: group A's regimen retained its ability to reduce PAF production while group B's regimen seems to enhance it. Further studies are needed to confirm these results and to unveil any possible clinical implications of anti-PAF activity of HAART.

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