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Disturbance of HDL apolipoprotein AI metabolism in severe hyperlipidemic and lipodystrophic HIV patients on a protease inhibitor treatment

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Background

The aim of this study was to characterize the metabolic abnormalities resulting in low HDL apolipoprotein AI (HDL-AI) levels in lipodystrophy HIV infected patients during protease inhibitor therapy.

Methods

Seven HIV infected patients, normolipidemic with no lipodystrophy (group A) and seven hyperlipidemic with lipodystrophy (group B) were studied. Patients are on protease inhibitors since at least six months. Patients were underwent *in vivo* kinetics of HDL-AI using a 14 h primed constant infusion of [5,5,5,$^2H_3$] leucine. Kinetic data were analyzed by monocompartmental model using SAMII program to drive metabolic parameters (FCR, Fractional Catabolic Rate, and APR, Absolute Catabolic Rate).

Results

Subjects in group B showed significantly higher plasma triglycerides ($p < 0.05$). HDL cholesterol and apolipoprotein AI (apoAI) were significantly ($P < 0.05$) lower in group B compared to group A. HDL are more enriched in triglycerides in group B compared to group A ($P < 0.005$). Kinetic study showed no change of fractional catabolic rate between two groups but significantly ($P < 0.05$) lower APR in group B compared to group A.

Discussion

These results showed that the hypertriglyceridemia and low HDL level associated with lipodystrophy in HIV infected patients during treatment is related essentially to low absolute catabolic rate. As was almost reported, the HDL enriched in TG are quickly catabolised. In our patient from group B, although HDL were enriched in TG their FCR was normal suggesting a primary abnormality in apoAI synthesis and/or secretion.

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