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SERUM CORTISOL: DHEA RATIO IS THE BEST PREDICTOR OF CLINICAL EVOLUTION AND LIPID VARIATIONS ASSOCIATED TO LIPODYSTROPHY

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OBJECTIVES: We have previously shown that lipid alterations associated to the Lipodystrophy syndrome (LD) in HIV-1-infected men on combined antiretroviral therapy (ART) are correlated with decreased DHEA, increased cortisol-DHEA ratio and increased serum IFN- α levels. In this study, we asked whether clinical evolution and changes in circulating lipids in LD-positive men is associated with changes in the parameters.

METHODS: Thirty-five HIV-1-positive men on ART, 20 of them presenting with LD, defined by clinical examination and computed tomography scan, were followed during 25.9 ± 4.0 months and tested at two time points. LD patients were subdivided into three groups, unchanged ($n=5$), aggravated ($n=9$) and improved ($n=6$), according to their clinical evolution and anthropometric measurements, including waist-hip ratio and skinfold thickness in the four usual sites. Serum lipids (cholesterol, triglycerides and their subclasses and apolipoproteins), cortisol, DHEA and cytokines (IFN- α , TNF- α , IL-6) were measured and compared to baseline values of LD-negative patients.

RESULTS: We show that: (1) LD aggravation is associated to persistent elevated lipids a decrease in serum DHEA (-60%, $P < 0.02$ and -66%, $P < 0.008$), an increase in cortisol: DHEA ratio (+170%, $P < 0.009$ and +200%, $P < 0.005$) and persistent high levels of IFN- α . (2) LD improvement is associated to normalization of serum lipids, an increase in serum DHEA (+190%, $P < 0.001$) leading to normalization of the cortisol: DHEA ratio and normalization of IFN- α levels. (3) The absence of LD evolution is associated with

persistent elevated lipids, a slight increase in serum DHEA, a slight decrease in both cortisol-DHEA ratio and IFN- α . (4) Importantly, we found that, in aggravated and improved patients, there is a negative correlation between atherogenic lipids, such as VLDL cholesterol and VLDL triglycerides and serum DHEA ($r=0.57$, $P<0.03$ and $r=-0.60$, $P<0.02$, respectively) and a positive correlation between these lipids and cortisol-DHEA ratio ($r=0.63$, $P<0.016$ and $P<0.54$, $P<0.046$, respectively). (5) The follow-up of LD-negative men ($n=15$) revealed that those who began to develop LD ($n=6$) showed an increase in lipid levels, a decrease in serum DHEA, an increase in cortisol-DHEA ratio and an increase in IFN- α levels. In contrast, patients who remained LD-negative ($n=9$), maintained normal lipids, elevated cortisol, elevated DHEA leading to a normal cortisol-DHEA ratio, and normal levels of IFN- α .

CONCLUSIONS: This study suggests that cortisol-DHEA ratio is the best predictor of clinical evolution and atherogenic lipid alterations in LD patients.

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