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Original article

Atherogen lipid profile in HIV-1-infected patients with lipodystrophy syndrome

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Abstract

Background: Cases of lipodystrophy syndrome and metabolic disorders have been described since the onset of highly active antiretroviral therapy in HIV-infected patients. The aim of our study was to estimate the prevalence of lipodystrophy (LD) and to define the associated lipid profile of these patients. *Methods:* The following were determined for each patient: lipid profile (cholesterol and its subfractions, atherogenicity ratios, and triglycerides), blood glucose, and immunovirological markers ($CD4^+$ cell count and plasma viral load). Patients were classified into two groups on the basis of whether or not they presented with clinical signs of LD. *Results:* Among 233 HIV-infected patients included in the study, 61 cases (26.1%) of lipodystrophy (LD) were noted. Compared with non-LD patients (NLD), LD patients were older men ($P<10^{-4}$) with a lower $CD4^+$ lymphocyte cell count ($P<0.007$) and more often at the AIDS stage ($P<10^{-3}$) (OR=3.2 (95% CI: 1.47–6.2)). Multivariate analysis showed a correlation between LD cases and age (10 years older) (OR=1.78 (95% CI: 1.23–2.57), $P<0.002$) and the decrease in $CD4^+$ cell count (100 $CD4^+/mm^3$ lower) (OR=1.31 (95% CI: 1.09–1.58), $P<0.004$). An analysis of lipid subfractions and atherogenicity ratios clearly indicated a proatherogenic lipid profile for the LD patients. *Conclusions:* The underlying physiopathological mechanism of LD is still unknown. However, the lipid profile of HIV-1-infected patients with a LD syndrome appears to place these patients at an increased risk of progression of atherosclerosis.

Keywords: Atherosclerosis; Cholesterol; Dyslipidemia; HIV-1; Lipodystrophy; Protease inhibitor

1. Introduction

Abnormalities in fat distribution have been amply described since 1997 among patients on highly active antiretroviral therapy (HAART) with protease inhibitors (PI) [1–7]. Lipodystrophy syndrome (LD), which associates various degrees of fat wasting and central adiposity, was initially described as a pseudo-Cushing's syndrome [1]. Fat wasting is accompanied by a reduction in subcutaneous fat, causing facial emaciation (atrophy of buccal fat pads), accentuation of facial bone structure, hollowing of the buttocks, and a pitted appearance of the lower and/or upper limbs [3–6]. Fat accumulation usually occurs in various regions (mesenteric and/or retroperitoneal, abdominal subcutaneous, cervical, supraclavicular, and mammary) [1–9]. Cases of LD have been described more frequently since the introduction of HAART that includes protease inhibitors (PI) and less often without PI [10–12]. In addition, PI give rise to disturbances in lipid metabolism [8] and glucose tolerance [9]. These metabolic distur-

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