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LIPODYSTROPHY AND METABOLIC DISORDERS IN 646 HIV-1 INFECTED PATIENTS PREVIOUSLY TREATED WITH OR WITHOUT A PROTEASE INHIBITOR.

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OBJECTIVES: To compare the clinical and metabolic features in HIV-infected patients treated with PI to those who received antiretroviral regimen without protease inhibitor (PI).

DESIGN: Cross-sectional analysis of 646 treated HIV-infected patients, routinely followed in 6 Paris hospital centres between January and May 1999. Demographic data, familial and personal vascular risk factors, history of antiretroviral treatment, HIV plasma viral load, CD4 cell count and metabolic data were collected. Clinical examination was based on an assessment of abdominal, dorso-cervical and breast girth, wasting of the limb, face and skin changes noted by the clinician and/or reported by the patients.

RESULTS: The mean age at inclusion in the study was 40 years, 31% were women; 38.5% of patients were heterosexuals, 27.4% IVDUs, 26.8% homo/bisexuals and 7.3% from other exposure groups. At study assessment, 76.9% were protease-inhibitor (PI group recipients) and 23.1% received ART without a PI (PI naïve [PIN] group). Mean delay since HIV diagnosis was similar in the two groups (84.3 in PI vs 89.9 months in PIN; $p=0.20$) as was mean previous duration of ART (45.6 vs 41.6 months; $p=0.13$). Respectively in PI and PIN groups, mean CD4 cell count was 212 vs 309/ μ l ($p<0.001$) and mean plasma HIV-RNA was 4.9 vs 4.3 log copies/ml ($p<0.001$) before initiation of any treatment, while at study assessment, mean CD4 cell count was 397 vs 485/ μ l ($p<0.001$) and mean plasma HIV-RNA was 2.5 vs 2.8 log copies/ml ($p<0.001$). Frequency of clinical lipodystrophy was higher in PI (63.4%) compared to PIN (39.6%) ($p=0.001$). Mean number of symptoms was also higher in PI than in PIN (3.1 vs 2.1 signs; $p<0.001$) as was mean score of severity (5.0 vs 3.5; $p<0.001$).

Among lipodystrophic patients, 3 clinical categories were identified: lipoatrophic (25.7%), hypertrophic (21.4%) and mixed (52.9%). Hypertrophic type was statistically associated with PIN whereas mixed group was associated with PI. No association was found between lipoatrophic type and treatment group. Glucose did not differ by treatment regimen. Mean cholesterol was higher among patients treated with PI than among other patients (5.56 mM/l vs 4.67 mM/l; $p < 0.001$), as was mean triglyceride (2.12 mM/l vs 1.38 mM/l; $p < 0.001$).

CONCLUSIONS: Lipodystrophy is frequently observed in PI treated patients, but also in patients receiving an ART regimen without PI in whom hypertrophic type is more frequently observed. As previously described, duration of infection, previous ART and previous PI treatment was associated with lipodystrophy occurrence. Further analyses are underway to precise risk factors associated with lipodystrophy and dyslipidemia in this large population of HIV-infected patients.

Keywords: AEGIS, Lipodystrophy, HIV-1, Diabetes Mellitus, Lipoatrophic, Protease Inhibitors, CD4 Lymphocyte Count, HIV-Associated Lipodystrophy Syndrome, Viral Load, HIV Protease Inhibitors, Reverse Transcriptase Inhibitors, Hyperlipidemia, Triglycerides, Cross-Sectional Studies, Paris, Human, Female, AIDS

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