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LIPODYSTROPHIC SYNDROMES IN A COHORT OF HIV-1-INFECTED PATIENTS RECEIVING HAART WITH A PROTEASE INHIBITOR

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A retrospective-prospective observational study investigated lipodystrophic syndromes in a cohort of 196 HIV-1-infected patients receiving HAART with a protease inhibitor (PI). As of January 1999, 49 patients were clinically identified as having either pure lipoatrophy ($n=14$) or pure obesity ($n=5$), or obesity associated with lipoatrophy ($n=30$), after 20.1 ± 6.1 months on HAART. Patients with and without lipodystrophy were not different for age, sex, CD4 count and plasma HIV RNA at month 0, month 12 and month 24 of HAART. Compared to patients without lipodystrophy, patients with lipodystrophic syndromes had longer durations of known HIV infection (103.6 ± 51.3 versus 83.5 ± 49.4 months, $P=0.015$), HAART (28.6 ± 6.1 versus 22.3 ± 8.6 months, $P<0.0001$) and nucleoside treatment before HAART (20.2 ± 17.5 versus 10.9 ± 15.8 months, $P=0.0005$). Lipoatrophic patients were younger than patients with obesity or obesity plus lipoatrophy (37.1 versus 42.9 years, $P=0.03$); and had longer exposure to all nucleosides prior to HAART (38.5 ± 21.1 versus 20.2 ± 20.8 months, $P=0.006$), whereas durations of known HIV infection and HAART were not statistically different (respectively: 124.6 ± 46.4 versus 90.3 ± 49.3 months, $P=0.054$; and 28.9 ± 4.5 versus 26.3 ± 5.8 months, $P=0.073$). Longitudinal analysis of lipodystrophic patients showed increased fasting blood triglycerides and cholesterol at time of lipodystrophy as compared to values at HAART initiation (respectively: 3.58 ± 2.62 versus 1.58 ± 0.89 mmol/l, $P<0.0001$; and 5.85 ± 1.98 versus 4.55 ± 1.25 mmol/l, $P<0.0001$); weight and fasting glycaemia did not vary significantly ($P=0.98$ and $P=0.23$, respectively). A significant elevation of blood triglycerides and cholesterol was also noted between month 0 and month 12 of HAART in non-lipodystrophic patients, respectively: 1.96 ± 1.71 versus 1.47 ± 0.81 mmol/l, $P=0.027$; and 5.32 ± 1.42 versus 4.26 ± 0.90 mmol/l, $P<0.0001$). None of these parameters were modified during nucleoside analogue therapy. In conclusion, length of nucleoside

exposure before HAART may be a risk factor for lipoatrophy, whereas elevation of blood lipids is seen independently of lipodystrophy and may be dependent on PI exposure.

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