HIV-associated asymmetric lipodystrophy syndrome

Mr. Editor:

Since the introduction of HIV-1 protease inhibitors as components of antiretroviral drug combination regimens, the clinical course of HIV disease and opportunistic infections have changed dramatically. However, parallelly to the virological, immunological and clinical benefit of highly active antiretroviral therapy (HAART) several adverse drug reactions have been observed and actually they are attributed to almost all components of it.

Particularly, peripheral lipodystrophy, central adiposity, dyslipidaemia and insulin resistance have been described with a prevalence of up to 80% in patients infected with HIV receiving HAART. Some years after the first description of HIV therapy-associated abnormal fat redistribution, the case definition, diagnostic procedure and treatment options remain still to be well defined.

An Etiopian 42-year-old woman married with and HIV-positive Italian man was referred to our outpatient department in November, 1998 because of chronic HIV infection. At that moment, her CD4 lymphocyte count was 338 cells/mm$^3$ and plasmatic HIVRNA was 34,000 copies/ml. No previous history of opportunistic infections was recorded. The patient was then started an antiretroviral treatment (ART) including zidovudine, lamivudine and saquinavir (h.g. capsules) then, she followed laboratory and clinical controls first after 6 weeks and then every 12 weeks.

After 6 weeks of therapy the patient achieved a plasma HIVRNA level of < 50 copies/ml which was always been maintained, with a slow increase of CD4 cells count (475 at 6 weeks). After 16 months of ART without clinic or biochemical evidence of side effects, the patient referred slight body shape modifications characterised by an increase on volume of the right upper thigh. The sonography confirmed an abnormal fat deposit on the right limb (16-17 mm) bigger than on the left one (9 mm). HIV viral load remained < 50 copies/ml and CD4 cell count increased progressively to 738 cells/mm$^3$. No metabolic abnormalities were found. Patient follow-up at 22 months still showed and optimal viroimmunological response to the therapy (HIVRNA < 50 copies/ml and CD4 cell count 798 cells/mm$^3$) without metabolic alterations. At that moment the patient had also noticed symmetrical morphological alterations on abdomen (increase) and arms (decrease) associated with enlargement of the fat-pat of both hands. At the physical examination, an objective volumetric difference between both thighs could be observed (measurement showed a greatest diameter of 5 cm in the right thigh respect to the left one). At this time, the measurement of thigh fat by sonography did not show significant differences with the first one.

Discussion

HIV-associated lipodystrophy syndrome is characterised by changes in body fat distribution and metabolic disturbances and it has been first related to some antiretroviral drugs. The clinical presentation can be very heterogeneous with different effects on adipose tissue (fat loss or fat accumulation) and at different sites but usually symmetrical. Three different forms of lipodystrophy: have been described pure fat atrophy with a bilateral loss of cutaneous fat in the extremities, buttock or face; pure fat hypertrophy with fat accumulation in neck and abdomen, and/or breast enlargement; and mixed type; all of them accompanied or not with metabolic alterations. Adipose redistribution in HIV positive patients during antiretroviral therapy has previously been associated to the nadir of CD4 lymphocytes and the immunoreconstitution.

Here we have described a patient with asymmetric and peripheral fat accumulation as an unusually form of lipodystrophy presentation in an HIV-positive patient as objectively measurement by sonography. To our knowledge, only breast enlargement has been described occurring unilaterally during antiretroviral therapy, but it was attributed to breast hypertrophy rather than hypertrophy of adipose tissue.

REFERENCES


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