We report a case of multifocal tuberculosis that occurred about three months following ARV, with the objective of contributing to a better management of tuberculosis-HIV co-infection.

**Observation**

A 42-year-old patient tested HIV positive in May 2015 and placed under Atripla with a rate of TCD4 lymphocyte with 302 cell / mm³. She was admitted on 07 July 2015 for vomiting and alteration of the general condition of progressive installation. His condition got worse over the last month during a malaria episode requiring her hospitalization in a health center where antiretroviral treatment was initiated. As her condition was not improving, she was taken to the CHU YO for treatment. When she was admitted, her general condition was altered with cachexia, the conjunctiva was pale and jaundiced; there was a fold of undernutrition, the blood pressure at 100/60mmHg, the heart rate at 96 beats / min, the temperature at 38°C, respiratory rate at 34 cycles/minute; pulmonary auscultation showed crackling lung sounds in both lung fields, opacity at the percussion of the lung bases, more stressed on the right, a painful hepatomegaly. Abdominal ultrasound revealed homogeneous hepatomegaly with mid-abundance ascites. Pulmonary radiography showed right base pleurisy and...
bilateral pneumopathy. BAAR research turned negative. Complete blood counts indicated a leukocytosis at 12,700/mm³, a moderate anemia at 8.5 g/dl. Thick drop was negative, the TCD4 lymphocyte count was 147 cells/mm³, anti-HBc antibodies were positive, HBS antigen was negative. She was placed on anti-tuberculosis treatment on 10/07/2015 (3cp/d) and the X-Ray examination on 4 August 2015 noted a radiographic improvement with the disappearance of pleurisy (see Figures 1 and 2). She left the hospital on August 10, 2015.

**DISCUSSION**

In sub-Saharan Africa, the HIV pandemic has a strong influence on the epidemiology of tuberculosis. The incidence rate for tuberculosis is the highest in the world (Range et al., 1998; Harries, 1990; Cantwell and Binkin, 1996). The level of TCD4 lymphocyte is a determining factor in the progression of HIV infection and in the development of opportunistic infections. In HIV patients, the development of common tuberculosis causes transitional lymphopenia of CD4+. This lymphopenia is due to a reaction to mycobacterial infection (Badri et al., 2001). HIV-positive patients with tuberculosis are all eligible for antiretroviral treatment because they are classified either in clinical stage III or IV of the WHO (Harries et al., 2006; World Health Organization, 2003). Tuberculosis and antiretroviral treatment improve the general condition and level of performance, increase weight, which are cost effective basic indicators of successful treatment (Kwanjana et al., 2001). The determination of the HIV viral load is used to monitor the effectiveness of antiretrovirals, at the beginning of treatment and every six months thereafter. Three months following the beginning of an antiretroviral therapy, the realization of the viral load and the determination of the level of TCD4 lymphocytes should have allowed us to make the differential diagnosis between a common tuberculosis and the manifestation of an inflammatory syndrome of immune restoration, as a decline in the viral load accompanied by an increase in the level of TCD4 lymphocyte is in favor of the inflammatory syndrome of immune reconstitution (Poda et al., 2009). But the use of viral load is very limited in our context because of its low accessibility (Fujiiwara et al., 2005).

**Conclusion**

Tuberculosis developing within three months following initiation of antiretroviral therapy is not always a manifestation of immune restoration inflammatory syndrome. The accompanying TCD4 lymphopenia is often transitional and does not require a change in antiretroviral therapy. For a better follow-up of patients co-infected with TB-HIV it is important to make HIV viral load accessible. At the stage of severe immunosuppression, tuberculosis is often multifocal. Its early detection and treatment, as well as the availability of prophylaxis, are likely to have a favorable impact on the prognosis of TB-HIV co-infected patients.

**REFERENCES**


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