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CASE STUDY

MULTIFOCAL TUBERCULOSIS SIMULATING IMMUNE RESTORATION SYNDROME

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ABSTRACT

The development of common tuberculosis in a human immunodeficiency virus (HIV) infected patient causes an increase in viral load, resulting in TCD4 lymphopenia. When it develops three months following the initiation of an antiretroviral therapy (ART), its manifestations may simulate an inflammatory syndrome of immune restoration. The authors report a case of multifocal tuberculosis developing approximately three months following the ARV treatment, with the aim of contributing to a better management of tuberculosis-HIV coinfection. It is a 45-year-old patient tested positive for HIV1 and placed on Atripla since May 07, 2015 with a CD4 T cell rate of 302 cells/mm3. She was admitted on July 7, 2015 in the Department of Infectious Diseases for chronic cough and alteration of general condition in whom multifocal tuberculosis (pulmonary, pleural and peritoneal) was diagnosed with a TCD4 lymphocyte fallen down to 147 cells/mm3. The interview indicated a good adherence to antiretroviral therapy. Placed under a tuberculosis quadritherapy, the evolution was favorable and she left the hospital on August 10, 2015. Tuberculosis developing three months after initiation of antiretroviral therapy is not always a manifestation of immune restoration syndrome. The accompanying TCD4 lymphopenia is often transitional and does not require a change in antiretroviral therapy.

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INTRODUCTION

The development of HIV infection has resulted in an increase in tuberculosis in the world, particularly in developing countries (Ouédraogo *et al.,* 2001). It is one of the most communicable diseases, a major cause of deaths in the world. The WHO estimates that out of 9 million people who contracted tuberculosis in 2013, 1.1 million people were living with HIV (WHO, 2014). The African region is home to 4/5th of the cases and deaths due to tuberculosis HIV co-infection in the world (WHO 2014). The number of deaths due to HIV associated to tuberculosis has been declining over the last decade (WHO, 2014), thanks to the availability of anti-tuberculosis drugs and antiretrovirals. The development of common tuberculosis in HIV-infected patients causes an increase in HIV viral load and a decrease in CD4 lymphocytes (Badri *et al.,* 2001). If it develops within three months following the initiation of an antiretroviral therapy, it may pose a differential diagnosis problem with an inflammatory syndrome of immune restoration.

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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 HIV1 positive in May 2015 and placed under Atripla with a rate of TCD4 lymphocyte with 302 cell / $mm^{3.}$ 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/mm^3, 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.

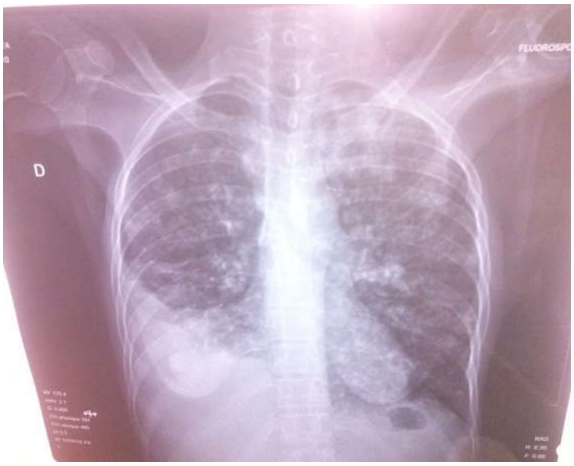

Figure 1. Picture before antituberculosis

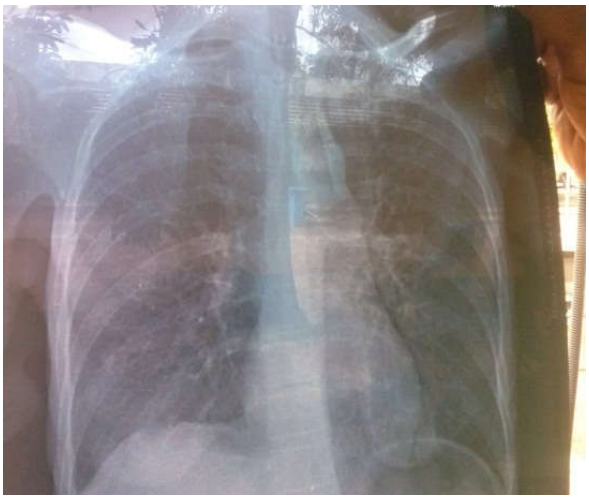

Figure 2. Picture one month later

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 (Fujiwara *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.

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