

Pulmonary HIV/AIDS-associated sarcoidosis

Editorial

The majority of cases with human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) associated pulmonary sarcoidosis have been related to immune reconstitution secondary to highly active antiretroviral therapy (HAART). Some previous studies demonstrated association between worsening of sarcoidosis course and CD4+ T lymphocyte count higher than 200cells/mm³, as well as association between the improvement of clinical symptoms and CD4+ T-cell count less than 200cells/mm³. Nevertheless, the onset of pulmonary sarcoidosis in HIV-infected/AIDS patients was demonstrated before the HAART era. The majority of the patients reveal restrictive lung pattern, but obstructive lung pattern can be identified. Bronchoalveolar lavage (BAL) demonstrates significantly high eosinophil, neutrophil, and lymphocyte counts, including significantly high CD4+: CD8+ T-cell ratio. BAL CD4+: CD8+ T-cell ratio is also significantly higher than the ratio in the blood at the time of diagnosis. Interleukin-2 or interferon- α 2a may be associated with pulmonary sarcoidosis development. The course of pulmonary sarcoidosis is similar in both HIV-infected/AIDS and HIV-seronegative patients. There is no significantly different change in virologic and immunologic parameters between patients with HAART in whom pulmonary sarcoidosis is clinically and radiologically cured or improved and pulmonary sarcoidosis patients with HAART that deteriorated or remained stable.

In conclusions, HIV-infected/AIDS patients with long-term immunologic reconstitution during HAART can develop pulmonary sarcoidosis. BAL-fluid analysis findings, clinical and radiological findings, and outcome are similar in both HIV-infected/AIDS and HIV-seronegative patients.

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Conflict of interest

The author declares no conflict of interest.

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