# Clinical remission of systemic lupus erythematosus after human immunodeficiency virus infection

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**Human immunodeficiency virus (HIV) infection during the course of systemic lupus erythematosus (SLE) is unusual. The clinical manifestations of SLE can be influenced by the HIV infection. Worsening of HIV has been documented after the use of immunosuppressives. We describe a case of a 37-year-old male patient who underwent complete clinical remission of SLE after serologic conversion.**

*Key words: Systemic Lupus Erythematosus, HIV, AIDS*

The association of systemic lupus erythematosus (SLE) and human immunodeficiency virus( HIV) infection in the same patient is rare (1,2). Both conditions have similar clinical and serologic features making their coexistence a diagnostic and therapeutic challenge (3). Moreover, HIV infection appears to have a favorable impact in the clinical course of SLE (1). On the other hand, the immunosuppressive therapy given for SLE activity has been associated with worsening of HIV infection (4). We hereby present a 37-year-old male patient with SLE that underwent full clinical remission after HIV infection.

## Case Report

A 37-year-old Puerto Rican man was diagnosed with SLE at age 19, in February 1987. Initially, he presented malar rash, arthritis, alopecia, non-painful oral ulcers, headaches, fever, anorexia, weight loss (28 lbs. in three months). Laboratory evaluation showed a white blood cell count of 2.5 x $10^3$/ul, total lymphocyte count of 0.9 x 103/ul, hemoglobin of 8.5g/dl, platelets of 226 x $10^3$/ul and erythrocyte sedimentation rate (ESR) of 50 mm/hr. Coombs and VDRL tests were negative. He had proteinuria of 2.2g/24 hrs. and serum albumin was 3.2g/dl. Serum BUN and creatinine were normal. ANA was positive at 1:1280, with a speckled pattern. Anti-dsDNA antibodies were negative He had C3 hypocomplementemia at 50 mg/dl (64-166 mg/dl). Lupus anticoagulant test and anticardiolipin antibodies were negative. He was initially treated with prednisone (1mg/kg/day) with improvement of clinical symptoms. Renal biopsy was not done.

He had several hospitalizations during the following years in a nearby community hospital: in early 1989 for acute ischemic stroke with right hemiparesis; in September 1989 for osteonecrosis of both femoral heads; in 1992 due to a right middle lobe pneumonia (during this admission HIV test was negative); in 1996 with a right gluteal abscess; and in 2001 with compression fracture of T12, L1, L2, L3 and a central disk herniation at L5-S1 level with resultant paraparesis. The patient was lost to follow up in between the acute events. He adjusted his prednisone dose by himself between 80-100 mg daily. Lower doses resulted in exacerbation of SLE (fever, malar rash, joint pains and oral ulcers). He did not have evaluations for disease activity or organ damage during this period. He did not take antimalarials or steroid sparing immunosuppressives drugs.

In May 30, 2004 he was admitted to the University Hospital with a left hip septic arthritis and a superimposed osteomyelitis of the femur. One year prior to admission he was able to gradually reduce the prednisone dose to 10-20 mg daily without causing any SLE reactivation. During this hospitalization, HIV test was positive by ELISA. He acknowledged homosexual life style and use of different illicit drugs. Immunoblotting showed strongly positive bands for gp160, gp120, p65, p55, p51, gp41, p40, p31, p24, and p18. Absolute CD4 count was 127/uL (359-1519) absolute CD8 count was 618/uL (109-897), CD4/CD8 ratio O.20 (0.92-3.72) and absolute NK (CD56) count was 64/uL (24-406). He had no clinical evidence of SLE activity, serum C3 and C4 levels were normal, anti-ds-DNA was negative and urine analysis was normal. The prednisone dose was tapered down and discontinued without reactivation of SLE.

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## Discussion

The patient fulfilled the ACR diagnostic criteria for SLE namely, malar rash (malar rash, oral ulcers, arthritis, proteinuria ³0.5 g/24 hr, leukopenia, lymphopenia and positive ANA) (5) seventeen years before HIV infection was documented. During his last hospital admission seroconversion was evidenced by a positive HIV test by ELISA and Western blot test. He had been able to decrease his prednisone dose from 80-100 mg prednisone daily to 10-20 mg daily during the last year. Clinical and serologic parameters of the disease activity were normal at the time of admission enabling discontinuation of steroids while in the hospital without disease reactivation. Although the precise time of HIV infection is not known, the low CD4 count and dramatic decrease in prednisone dose suggest that the SLE remission was indeed related to the HIV infection.

The pathogenesis of SLE is multifactorial (6). Although tissue damage is considered mainly a B cell derived process through the production of pathogenic autoantibodies and immune complexes formation, CD4 + T cells also play an important role in the pathogenesis of the disease (7). Therefore, any condition or therapeutic intervention that affects CD4+ T cells could have an impact on SLE.

Most SLE patients reported on the literature experience improvement of the disease after the HIV infection as their CD4+ T cells levels decrease (2). A major clinical concern is the worsening of HIV after the immunosuppression given to control SLE activity (8-11). The treatment of one disease may worsen the other in patients with both diseases (8,12).

Low CD4 + T cells and T cell activation may increase HIV replication (13).

Interestingly, and contrary to others reports, our patient did not show worsening of his HIV infection in spite of his low CD4 + T cell count (8-10). This could be related to the fact that he required less immunosuppression to control the disease.

The possible mechanisms to explain the interaction of HIV and SLE have been reviewed by several authors (10-11,14-17) One one hand, the immunosuppression induced by HIV may reduce the risk of developing SLE. On the other hand, the decreased CD4 + T cell count induced by HIV may improve the clinical manifestations of patients with established SLE. The increased production of pathogenic autoantibodies in SLE may work against HIV infection through the mechanism of molecular mimicry (17). Moreover, reports indicate that HIV treatment with antiretroviral therapy may cause SLE exacerbation due to increase in CD4 + T cell count (13, 4, 12).

In summary, our patient underwent full clinical remission of SLE after HIV infection enabling discontinuation of steroids. The clinical management of patients with coexistent diseases is very complex.

There are no guidelines on the use of immunosuppressive drugs to treat SLE in patients who are also HIV positive. The immunosuppressive therapy for SLE may increase viral replication. On the other hand, antiretroviral therapy, which decreases the morbidity and mortality in HIV (18), may increase SLE activity. Therefore, patients with SLE and HIV infection should be monitored very closely and immunosuppressive treatment frequently reassessed. The use of immunomodulator drugs such as hydroxychloroquine and chloroquine would be a better choice for these patients (19-20).

## Resumen

La coexistencia del virus de inmunodeficiencia humana (VIH) con el lupus (LES) sistémico eritematoso es inusual. Las manifestaciones clínicas del LES pueden ser influenciadas por la infección con el VIH. Además, se ha documentado el empeoramiento del VIH luego del uso de inmunosupresivos. Describimos un caso de un paciente de 37 años con una remisión completa de LES luego de seroconversión con el VIH.

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