中文題目：一位病患同時被診斷全身性紅斑性狼瘡和人類免疫缺乏病毒感染:病例報告

英文題目：Concomitant with Human Immunodeficiency Virus Infection in a Patient of Systemic Lupus Erythematosus: a Case Report

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Background

The coexistence of systemic lupus erythematosus (SLE) and human immunodeficiency virus (HIV) infection is uncommon. Several clinical and laboratory manifestations of both HIV infection and SLE could be similar that it makes great challenges to the diagnosis of concurrent diseases. Here, we describe a male patient with syphilis history, who presented with fever, leukopenia and thrombocytopenia, was diagnosed with SLE and HIV infection simultaneously.

Case Report

A 27-year-old male homosexual patient with a history of treated syphilis was hospitalized because of intermittent high fever for one week with nausea, vomiting, abdominal pain, poor appetite, and general malaise. Initially, he visited the local hospital where leukopenia and thrombocytopenia (WBC:2400/μL, Platelet:72000/μL) were found that he was transferred to our hospital. In our emergent department, high fever with the temperature 38.4°C was still noted. Leukopenia (WBC:2200/μL), thrombocytopenia (platelet:62000/μL), abnormal liver function tests(AST:369 IU/L, ALT:391 IU/L) and proteinuria (protein-creatinine ratio of urine: 493.13mg/g) were also found. Intravenous fluid supplement with the antibiotic of ceftriaxone were prescribed for suspected infectious gastroenteritis and the patient was hospitalized.

However, after treatment for 3 days, the above symptoms persisted without improvement. Cultures of stool and blood all showed negative results. There was also no viral hepatitis. Because the patient's mother was a patient of SLE, we arranged autoimmune disease laboratory examinations. Elevated antinuclear antibody (ANA)　(1:2560, speckle pattern), anti-double-stranded DNA (114.082 U/mL), anti-SSA (>240 U/mL) and anti-SSB (>320 U/mL) antibodies were confirmed with mild low C3 level (75.4mg/dL). Thus the diagnosis of SLE was made according to the SLE classification criteria by the Systemic Lupus International Collaborating Clinics (SLICC) in 2012 that methylprednisolone (40mg QD) with hydroxychloroquine (200mg BID) were used. Besides, because of syphilis and homosexual history, HIV examinations were checked. The HIV antibody revealed positive (9.61 S/CO) and HIV western blot showed indeterminate. The HIV viral load was 1.05 x 10^6copies/mL, and the CD4 T-cell count was 239 cells/mm3. After SLE treatment, without medications for HIV infection initially, the symptoms and the laboratory abnormalities improved gradually. Then, under stable condition, discharge was arranged.

After discharge, at our infection out-patient department (OPD), antiretroviral drugs with abacavir, dolutegravir, and lamivudine were prescribed for HIV infection. In rheumatology OPD, prednisolone was tapered gradually to 10mg daily with hydroxychloroquine (200mg BID) 2 months after discharge. Under combined SLE and HIV infection treatments, there were no more symptoms of recurrence. The laboratory abnormalities, including leukopenia and thrombocytopenia, became normal and the HIV viral load also decreased to 2.71 x 10^2 copies/mL one month after combination antiretroviral therapy.

Discussion

The cases of concomitant SLE and HIV infection are scarce and the first case was described in 1988. Recently, the nationwide cohort study from Taiwan revealed the patients with HIV infection had higher risks of incident autoimmune diseases, such as SLE. Herein, we report the first case with a concurrent diagnosis of SLE and HIV infection in Taiwan. These two diseases share several similar clinical manifestations, including fever, hematologic, renal and neurological abnormalities. SLE is characterized by the overproduction of autoantibodies. ANA has the greatest sensitivity and anti–double-stranded DNA and anti-Smith antibodies have the greatest specificity. However, some reports mentioned that HIV-infected patients may also produce autoantibodies, such as ANAs and anti-cardiolipin antibodies, which lead to diagnostic difficulties. It was reported that intravenous cyclophosphamide pulse therapy for SLE could result in an increase of HIV viral load. Thus aggressive immunosuppressant agents for SLE, such as cyclophosphamide, should be carefully used and monitored. Otherwise, highly active antiretroviral therapy (HAART) for HIV infection was ever found to be associated with SLE flare that combined patient care by rheumatologists and infectious disease specialists were suggested. In conclusion, although the coexistence of SLE and HIV infection is uncommon, the reported complicated cases indicate that we should be cautious about the diagnostic and therapeutic dilemmas between these two diseases.