A Patient with Human Immune Deficiency Virus Mimicking Systemic Lupus Erythematosus

Sistemik Lupus Eritematozusu Taklit Eden İnsan İmmün Yetmezlik Virüsü Pozitif Bir Olgu

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ABSTRACT

Background: Human immunodeficiency virus (HIV) can cause rheumatological manifestations due to infectious itself or treatment. Among these findings take part arthralgia, arthritis, photosensitivity, oral ulcer, sicca syndrome, fever, hypergammaglobulinemia, cytopenia, neuropathy and anti-nuclear antibody (ANA) positivity, which have been also observed in systemic lupus erythematosus (SLE). Not only does HIV imitate SLE, but also it might coexist with.

Case Report: A 40-year-old man who presented with oral lesions, blurry vision, vertigo, speech difficulty and ataxic gait admitted to the clinic. Cerebellar tests were abnormal. He could not walk in tandem gait. After observing leukopenia, lymphocytopenia and mildly increased liver function tests in laboratory and encephalitis on magnetic resonance imaging (MRI), autoantibodies and viral panel were requested. While autoantibodies were negative, HIV Enzyme-Linked Immunosorbert Assay (ELISA) was found positive.

Conclusion: HIV can lead to systemic autoimmune and autoinflammatory conditions. Notably, ruling out HIV in patients who have rheumatological findings is necessary.

Keywords: cytopenia, human immune deficiency virus, oral ulcer, systemic lupus erythematosus

ÖZ

Amaç: İnsan immune yetmezlik virüsünün (HIV) kendisi veya HIV tedavisinde kullanılan ajanlar romatizmal bulgulara neden olabilir. Bu bulgular arasında, sistemik lupus eritematozus (SLE) hastalarında da görülen artalji, artrit, fotosensitivite, oral ülser, sıkka sendromu, ateş, hipergammaglobulinemi, sitopeni, nöropati ve anti-nükleer antikor (ANA) pozitifliği yer almaktadır. HIV, sadece SLE’yi taklit etmez, Ayrıca SLE ile birlikte de olabilir.


Anahtar Kelimeler: sitopeni, insan immune yetmezlik virüsü, oral ülser, sistemik lupus eritematozus

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INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by various clinical presentations. Fever, oral ulcer, delirium, psychosis, seizure, non-scarring alopecia, acute, subacute and discoid cutaneous lupus erythematosus, pleural and pericardial effusion, acute pericarditis, joint involvement, nephritis might be seen in patients with SLE as clinical manifestations. Additionally, leukopenia, thrombocytopenia, autoimmune hemolysis, low level of C3 and/or C4, anti-ds DNA, anti-Sm, and antiphospholipid autoantibodies might also be detected in those patients (1).

Several diseases including other connective tissue diseases, infectious diseases, hematological malignancies, solid tumors and IgG4-related disease can imitate SLE (2). Accordingly, different kind of infections such as viruses, bacteria, parasites and other infectious agents can cause or exacerbate those above-mentioned symptoms mimicking SLE. Among viruses, cytomegalovirus, ebstein-Barr virus, hepatitis C, enterovirus, and human immunodeficiency virus (HIV) are mostly well defined viruses associated with autoimmunediseases (3).

Human immunodeficiency virusis a blood-borne virus targeting immune cells and causes acquired immunodeficiency syndrome (AIDS) in case of progression of the infection (4). Nowadays, the clinical presentation of HIV/AIDS has altered from mostly fatal condition due to opportunistic infections to a chronic disease in which cardiovascular, renal, autoimmune disorders, diabetes mellitus and malignancies have been revealed (5). HIV-related rheumatic findings including arthralgia, arthritis, myalgia, myositis, vasculitis and gout can be observed. Even though highly active antiretroviral treatment (HAART) has improved the rheumatological manifestations regarding HIV, it can also rapidly ameliorate immune system derived paradoxical inflammatory reactions against the preexisting infection. This entity is known as immune reconstitution inflammatory syndrome (IRIS) (5,6).

According to the above-mentioned literature, various rheumatological findings can be determined related to HIV; however, manifestations regarding systemic autoimmune diseases such as SLE are not generally common unless patients with HIV use HAART. We herein present a patient mimicking SLE who was diagnosed with HIV.

CASE

A 40-year-old man presented with 3 months history of oral lesions and 2 months history of blurry vision, vertigo, speech difficulty and ataxic gait. His past medical history was unremarkable. According to physical examination, his vital findings were within normal ranges. There were two oral lesions that were greater than 10 mm in diameter localized on the hard palate and tongue. Cerebellar tests including gait, finger-to-nose and heel-to-shin were abnormal. He could not walk in tandem gait. However, muscle strength and sensory exam were normal.

On laboratory outcomes, fasting blood glucose, blood urea nitrogen, creatinine, and thyroid-function tests were normal. The rest of laboratory data were as follows: alanine aminotransferase 129 U/L (normal, ≤35 U/L), aspartate aminotransferase 102 U/L (normal, ≤35 U/L), gamma-glutamyltransferase 72 U/L (normal, ≤38 U/L), hemoglobin 12.3 g/dl (normal, 12-16.3 g/dl), white blood cell count 3.0x10^3/mcL (normal, 0.9-3.2x10^3/mcL), neutrophil count 1.8x10^3/ mcL (normal, 1.7-7.6x10^3/mcL), lymphocyte count 0.9x10^3/mcL (normal, 1-3.2x10^3/mcL), C-reactive protein 1.5 mg/L (normal, ≤5 mg/L), erythrocyte sedimentation rate 42 mm/h (normal, ≤20 mm/h). All reference ranges are based on our university’s laboratory values. According to laboratory results, leukopenia, lymphocytopenia and mildly increased liver function tests were observed. Thus, he was screened in terms of hepatitis, human immunodeficiency virus, cytomegalovirus, Epstein barr virus, parvovirus B 19, as well.

The patient was scanned by cerebral magnetic resonance imaging (MRI). It was reported as multiple hyperintense spots located on
supratentorial white matter, corpus callosum and brainstem on standard T2-weighted MRI sequence. These lesions were interpreted as encephalitis with a neurological consultation. Additionally, he was consulted to ophthalmologist due to blurry vision. No pathological finding was found. He was also evaluated for autoimmune disorders causing cerebral involvement. Thus, anti-nuclear antibody (ANA), extractable nuclear antigen antibodies (ENA), anti-double stranded DNA (anti ds DNA), antineutrophil cytoplasmic antibodies (ANCA), myeloperoxidase (MPO) and proteinase 3 (PR3) antibodies were investigated. All autoantibodies, hepatitis screening, cytomegalovirus, epsteinbarr virus, parvovirus B19 were found negative. On the other hand, HIV positivity was detected by ELISA. HIV confirmation test was also positive. Consequently, the patient was transferred to department of infectious diseases.

**DISCUSSION**

Some manifestations and findings including photosensitivity, oral ulcer, sicca syndrome, arthritis, fever, hypergammaglobulinemia, cytopenia, neuropathy, and ANA positivity can be observed in both SLE and HIV infection. As a result, the differentiation between both diseases might be difficult (6). HIV causes autoimmune and systemic disorders via triggering immune dysregulation. The frequency of these autoimmune diseases has ranged from 1% to 60% according to literature (6,7). Furthermore, HAART, the drugs using for AIDS treatment, can ameliorate CD4+Tcells and it can cause IRIS presenting with various rheumatologic manifestations. Thus, the frequency of HIV related connective tissue disorders might be varied depending on pre-HAART and post- HAART era. For instance, SLE and rheumatoid arthritis (RA), which have CD4+ T cell-mediated pathogenesis, might be determined less in patients with HIV that targets those cells. On the other hand, systemic autoimmune, such as SLE and RA and autoinflammatory diseases have observed moreunder the HAART treatment, even though the prevalence of rheumatic manifestations regarding HIV havedecreased (5,8). Accordingly, indeed, both HIV and the treatment of HIV can cause autoimmune diseases (6).

Apart from HIV or HAART related SLE, coexisting SLE and HIV cases are also reported in the literature. Non-erosive arthritis (68.3%), nephritis (63%), neurological manifestations (12.5%), and hemolytic anemia are reported more common in patients with both diseases. Autoantibodies including ANA and ds DNA can be useful to differentiate SLE from HIV; however, those autoantibodies might also be produced in HIV patients. Sharing similar clinical and laboratory findings has started physicians to think about the association between them. According to the literature, concomitant SLE and HIV are rare; nevertheless, it can be more common than we know (9-11).

In conclusion, the differential diagnosis of SLE consists of autoimmune connectivetissue disorders, vasculitis,such as Behcet disease, viral infections including HIV, hepatitis B and C, and malignancies (2,3). Therefore, the patient who had oral ulcer, neurological findings, and cytopenia was hospitalized with an initial diagnosis of SLE, but, after follow-up, he was diagnosed with HIV infection. Consequently, when diagnosing SLE, differential diagnosis is fundamental in order not to overlook the underlying diseases mimicking SLE.

**REFERENCES**


