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HIV infection in lupus nephritis patients

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Abstract

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that predominantly affects women of childbearing age, with severe complications such as lupus nephritis often requiring long-term immunosuppressive therapy. On the other hand, HIV infection remains a global health problem because it causes progressive immune system decline and increases the risk of opportunistic infections. The coexistence of SLE and HIV is very rare, with fewer than 100 cases reported worldwide, posing complex diagnostic and therapeutic challenges. The study reports the case of a 32-year-old woman with a history of SLE and lupus nephritis who was later diagnosed with HIV, accompanied by symptoms of chronic diarrhea, weight loss, and opportunistic tuberculosis. Clinical and laboratory interpretation in this patient was quite difficult due to overlapping symptoms and the potential for false serological results in HIV testing. Management requires a careful balance between immunosuppressive therapy for lupus and antiretroviral therapy for HIV, with close monitoring to prevent Immune Reconstitution Inflammatory Syndrome (IRIS) and lupus flares. This case underscores the importance of comprehensive evaluation, multidisciplinary collaboration, and individually tailored therapeutic strategies to achieve optimal care for patients with concurrent SLE and HIV.

Keywords: Systemic lupus erythematosus, lupus nephritis, HIV infection, Opportunistic infection, Immune Reconstitution Inflammatory Syndrome (IRIS)

Introduction

Systemic Lupus Erythematosus (SLE) is a form of autoimmune disease, a condition in which the immune system mistakenly attacks the body's own tissues and organs. Although not as common as other chronic diseases, SLE occupies an important position because it is a relatively common autoimmune disease. Epidemiological data show that its prevalence reaches 130 per 100,000 people, a significant number for an autoimmune disease (Shaban and Leira, 2019). SLE primarily affects women of reproductive age, with a mean age ranging from 25.7 to 34.5 years (Sumantri *et al.*, 2021). SLE is much more common in women than in men, especially in Asia. Data shows that for every man with SLE, there are about 9 to 14 women who also have the disease. This imbalance is even more apparent in Indonesia, where a cohort study found that the number of women with the disease can be up to 22 times higher than men (Hamijoyo *et al.*, 2019).

Lupus nephritis is one of the most serious complications of SLE that affects the kidneys. Its diagnosis is based on the criteria of the American College of Rheumatology (ACR), including the presence of high levels of protein in the urine (proteinuria), either by laboratory measurement >0.5 g/24 hours or dipstick results ≥3+, as well as the presence of casts in the urine, which indicate damage to the kidneys. For treatment, the 2012 EULAR guidelines recommend the use of immunosuppressive drugs. Oral corticosteroids are administered in low to moderate doses to suppress inflammation, and in some cases may be combined with azathioprine (AZA) to enhance the immunosuppressive effect. This combination therapy is typically indicated for patients with significant proteinuria (>1 g/24 hours) accompanied by glomerular hematuria, as these conditions reflect more severe kidney damage (IRA, 2019).

Unlike SLE, HIV is a virus that attacks the immune system, making sufferers more susceptible to various infectious diseases and cancer. Globally, HIV remains a serious problem. The 2018 UNAIDS report estimates that nearly 38 million people worldwide are living with HIV, with a prevalence of about 0.8% in the adult population. The region with the highest burden is sub-Saharan Africa, due to high rates of transmission through unprotected sexual contact and limited access to health care. However, new HIV cases are still found in developed countries, although in smaller numbers, usually associated with risky sexual

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practices and intravenous drug use with shared needles (Okada, 2021).

HIV enters the human body through intact mucous membranes, broken skin, and through parenteral inoculation. Transmission of the virus from mother to child occurs primarily in the third trimester and through breast milk (Gorsch-Worner et al., 2000). Detection of the HIV can be done in two ways: serological and virological examinations. Serological methods can detect antibodies and antigens, consisting of rapid immunochromatography tests (RATs) and EIA (Etiology) *enzyme immunoassay* Virological testing is performed by examining HIV DNA and HIV RNA. The clinical stage of HIV can then be determined based on the clinical symptoms and opportunistic infections that appear (Ministry of Health of the Republic of Indonesia, 2019).

HIV infection is known to cause symptoms that resemble autoimmune diseases, sometimes leading to diagnostic confusion. However, cases of HIV occurring concurrently with SLE are very rare. Based on literature reports, the number of cases recorded globally is only around 80 (Liao, 2017). To date, how these two diseases influence each other remains a major question; there are indications that HIV may modify the course of SLE, or conversely, SLE may affect the progression of HIV, but the available evidence is still limited (Belgaumkar, 2019). These rare cases are important to study because they can provide new insights into the immunological mechanisms underlying both HIV and SLE. On the other hand, a major challenge arises in terms of therapy. SLE is usually treated with immunosuppressive drugs, but in patients with HIV, the use of immunosuppressants can be risky because it can exacerbate immune deficiency. Therefore, healthcare providers must carefully consider safe treatment options. This situation presents both diagnostic and therapeutic dilemmas (Liao, 2017). In this report, we describe a rare case of coexisting SLE and HIV and review the relevant literature to discuss the diagnostic, pathogenetic, and therapeutic implications of their relationship.

Case

A 32-year-old woman (previously married), Javanese, Muslim, employee at a *department store*, has 1 child aged 1 year, resides in Surabaya, came to the Emergency Room in February 2023 with complaints of chronic diarrhea for 1 month, weight loss since the last 2 months of 5 kg, accompanied by cough and shortness of breath since the last 1 month. Fever and night sweats have been felt for the last 1 month. Black stools or bloody mucus are denied, vomiting red or black blood is denied, coughing up blood is also denied. Mouth ulcers are denied, hair loss is denied, red face when exposed to sunlight is denied, seizures are denied, changes in consciousness are denied, and urination is felt to be normal.

The patient is a SLE sufferer with manifestations of Lupus Nephritis who was diagnosed at a Private Hospital in Surabaya since 2013 based on clinical complaints, namely complaints of joint pain, foamy urine, swelling of both legs, hair loss accompanied by supporting ANA *test* positive. From 2013 to 2018, the therapy received was methylprednisolone 1x16 mg, methotrexate 4 tablets/week. In 2018, the patient was treated at RSDS with complaints of swelling in both legs accompanied by shortness of breath, foamy urine, and was found to have a low albumin value of up to 1.8 mg/dL with proteinuria +4. The patient received remission induction therapy with cyclophosphamide six times (having previously undergone *screening* HIV-negative), and afterward, the patient experienced improvement in proteinuria to +2 and never experienced episodes of swelling or hypoalbuminemia. Outpatient maintenance therapy received from 2018 to February 2023 consisted of methylprednisolone 4 mg once, Immuran 50 mg twice, hydroxychloroquine 200 mg once, folic acid, and calc. In March 2021, the patient's first pregnancy was complicated by condyloma acuminata. HIV screening was performed and the results were positive *undefined*. Routine patient *ante natal which* at RSDS, and in October 2021 it was decided to terminate the pregnancy with *sectio caesaria* due to premature rupture of membranes. Repeat HIV screening was performed in October 2021, and the results were positive *undefined.* The patient continued to receive regular outpatient treatment at the dermatology clinic for vulvovaginal candidiasis and condyloma acuminata, as well as at the rheumatology clinic for SLE every month throughout 2022. In August 2022, the patient complained of a lump in her neck, and a FNAB examination was performed, which revealed lymphadenitis. The patient had sexual intercourse with her boyfriend in 2017 for two years, then with her deceased husband

from 2019 until they married in January 2021. The patient's late husband passed away in December 2021 due to a pulmonary illness; however, his HIV status was unknown. It was noted that he had a history of frequent sexual relations with commercial sex workers prior to 2019.

Physical examination upon arrival at the Emergency Room on 4/2/23 found a general condition of weakness, GCS 456, blood pressure 79/58 mmHg, pulse 115 x/minute, regular, weak to the touch, cold extremities *capillary refill time* more than 2 seconds, respiratory rate 26 x/minute, oxygen saturation 94% with free air and 98% with nasal tube oxygen 3 l/minute, axillary temperature 37.5^{the}C. Weight 46 kg, height 158 cm, body mass index 18.4 kg/m^2. On examination of the head and neck, alopecia and conjunctiva of the eyes were found *slight* anemic, sclera does not appear icteric, does not appear *oral thrush*, found enlargement of the right anterior colli lymph node with a diameter of 2x1 cm. Thoracic examination found symmetrical chest movement, retraction (-), rhonchi (+) in both lung fields, wheezing (-), single S1S2 heart sound, murmur (-), and gallop (-). On abdominal examination, it appeared supple, bowel sounds were normal, and no hepatosplenomegaly was found. Examination of the extremities found *pitting edema* on both legs. Examination of skin turgor showed decreased. Anogenital examination revealed condyloma acuminata.

From the blood laboratory examination on 4/2/23, hemoglobin was 9.2, leukocytes 15,130 with neutrophils 94.5% and lymphocytes 2.4%, platelets 269,000, AST 124, ALT 45, total bilirubin 0.6, direct bilirubin 0.5, albumin 2.4, serum creatinine 0.8, BUN 17, sodium 132, potassium 3.9, chloride 103, magnesium 1.3, random blood sugar 58, pH 7.45, $pCO_2$32, $pO_2$102 (with nasal tube, PF *ratio* 329), HCO_3 22.2, BE -1.8, $SaO_2$98, C3 138 (normal), C4 58 (normal), and HIV 3 reactive method (807.1). Urine examination on 4/2/23 showed amber color, clear, BJ 1.023, pH 5.5, leukocytes 3+, albumin 150 mg/24 hours, proteinuria +1. Radiological examination on 4/2/23 showed reticular pattern in both lungs which is interstitial pneumonia dd TB miliary and no abnormalities visible. Microbiological examination of RT-PCR swab COVID was negative.

Based on the patient's history, physical examination, and laboratory findings, she was diagnosed with AIDS, low-activity SLE (SLEDAI score 2), sepsis associated with miliary tuberculosis versus Pneumocystis jirovecii pneumonia (PCP), hypoglycemia (58 mg/dL), and hypoalbuminemia (2.4 g/dL) related to reduced intake, chronic diarrhea, and condyloma acuminata. The patient was treated with oxygen therapy via nasal cannula at 3 L/min, intravenous rehydration with 0.9% NaCl 1500 mL/4 hours followed by 20 drops per minute, intravenous D40 (2 ampoules), intravenous 20% albumin infusion 100 mL/4 hours, intravenous norepinephrine infusion at 50 ng/kg/min, intravenous levofloxacin 750 mg every 24 hours, packed red cell (PRC) transfusion 1 unit, a high-calorie and high-protein diet plan (1,900 kcal/day), oral attapulgite 2 tablets every 8 hours, oral hydroxychloroquine 200 mg every 24 hours, oral cotrimoxazole 4800 mg every 24 hours, oral prednisone 40 mg every 12 hours, and discontinuation of azathioprine.

Disease course

On the 4th day of treatment (7/2/23), the patient's condition improved. Shortness of breath had begun to decrease, coughing was still present, diarrhea had stopped, fever was gone, appetite improved, weakness was reduced, nausea and vomiting were denied. Vital signs examination revealed a general condition of sufficient, GCS 456, blood pressure 103/85 mmHg (without vasopressors), pulse 89 x/minute with strong lifting, RR 20 x/minute, temp 36.5^{the}C, $SpO_2$97% with a nasal tube of 2 l/minute. Physical examination revealed that the conjunctiva was not anemic, the thorax showed rhonchi (reduced), turgor improved, and *pitting edema* has decreased. Blood laboratory showed improvement in hemoglobin after 1 cup of PRC transfusion, to 11.2, decrease in leukocytes to 12,290, platelets to 256,000, improvement in liver enzymes AST 24, ALT 18, increase in albumin after albumin transfusion to 2.8, improvement in random blood sugar to 113. Examination *imaging* a chest HRCT was performed for this patient, and was consistent with the picture of miliary TB. *Gene-Xpert* patient's sputum detected the presence of *Mycobacterium tuberculosis*. Based on clinical and supporting examinations, a diagnosis of miliary TB was confirmed and intensive phase anti-tuberculosis medication (OAT) was administered to this patient.

On the 8th day of treatment (10/2/23), the patient's clinical and supporting symptoms improved, and he was discharged from the hospital. The patient received intensive phase OAT outpatient therapy of 4 tablets every 24 hours (for TB), prednisone 40 mg every 24 hours (for TB and SLE), hydroxychloroquine 200 mg every 24 hours, cotrimoxazole 4800 mg every 24 hours (for PCP for 21 days), folic acid 1 tablet every 8 hours, and calcium carbonate 1 tablet every 8 hours orally. The patient returned to the UPIPI clinic on the 14th day after being discharged from the hospital (24/10/23) for a viral load evaluation and 1,488,679 copies/ml were detected *copy* HIV. The patient was given ARV (Anti-Retroviral) in the form of TLD (Tenovovir – Lamivudine – Dolutegrafir) 1 tablet every 24 hours because she was actively breastfeeding. The patient was scheduled for CD4 blood level evaluation and routine monthly check-ups at the UPIPI and Rheumatology clinics.

Discussion

HIV infection occurring concurrently with SLE is a rare clinical phenomenon that raises many questions. Both diseases affect multiple organs and are related to the immune system, particularly T cell function, cytokine regulation, and B cell activation (Sekigawa, 2002). The history of reports on this case is quite long. Kopelman and Zolla-Pazner first documented cases of HIV and SLE coexistence in 1988 (Kopelman, 1988). Years later, Mody and colleagues reported the largest series of cases at that time, involving 13 patients in South Africa (Mody, 2014).

However, to date, the number of documented cases remains low, with only about 80 cases worldwide, and the exact relationship between the two diseases remains unclear (Liao, 2017). According to the literature, there are three main clinical patterns that describe the coexistence of HIV and SLE: HIV appears after the diagnosis of SLE, SLE appears after HIV, or both are diagnosed at the same time (Gindea, 2010). Of these three patterns, the most common is patients with SLE who later become infected with HIV. Interestingly, several studies have shown that HIV infection can reduce SLE activity, possibly due to the virus's effect on the immune system, particularly the reduction in T cell counts, which also play a role in the autoimmune process (Furie, 1991).

In this case, the patient first suffered from SLE (2013) and was then diagnosed with HIV in 2021.

SLE and HIV both have a wide spectrum of symptoms, with many similarities in both clinical and laboratory aspects. This often leads to diagnostic difficulties, and not infrequently results in misdiagnosis or delayed diagnosis (Carugati, 2013). HIV itself can mimic autoimmune symptoms such as fever, enlarged lymph nodes, skin rashes, kidney dysfunction, nervous system disorders, blood disorders, sicca syndrome, and joint pain (Calabrese, 2005). In addition, both SLE and HIV patients have a high risk of opportunistic infections due to immunosuppression, either as a result of the disease itself or due to treatment (Sekigawa, 2002).

Tuberculosis (TB) is the most commonly reported opportunistic infection, especially in developing countries. TB can be found in HIV patients (WHO, 2013) and SLE patients (Zandman, 2005). This risk increases when both diseases are present simultaneously. A study by Mody (2014) showed that 7 out of 13 patients with HIV and SLE had TB. Similar results were shown by a cohort study by Hax (2018), which found 20 cases of TB in HIV patients. In addition to TB, other opportunistic infections such as candidiasis are also commonly found in both SLE patients (Ginzler, 2002) and HIV patients (WHO, 2013). Therefore, HIV testing is strongly recommended for SLE patients, as the use of immunosuppressants can mask HIV symptoms and increase the risk of fatal disease progression. Therefore, clinicians need to be aware that the progression of HIV and SLE can follow similar clinical patterns, requiring careful diagnosis (Okada, 2021).

In this case, the patient's clinical manifestations and opportunistic infections overlapping with HIV and SLE symptoms were polyarthralgia, vulvovaginal candidiasis, colli lymphadenopathy, and condyloma acuminata before HIV was discovered. After HIV was discovered, another opportunistic infection was miliary tuberculosis.

HIV diagnosis in patients with SLE is often challenging due to the possibility of misleading test results. In SLE patients, HIV ELISA tests can produce false positive results due to the presence of autoantibodies and cross-reactivity with viral antigens (Jian, 2015). The frequency of false positives varies considerably, ranging from 0% to 45%,

depending on the quality of the kit used, although newer generation kits show better specificity. Even the Western blot test, which is usually used for confirmation, can produce inconclusive results in up to 35% of SLE patients (Barthel, 1993).

Conversely, patients with active HIV can also produce autoantibodies, such as antinuclear antibodies (ANA) and anticardiolipin antibodies, further complicating the differentiation between HIV and SLE (Massabki, 1995). The situation becomes more complex when SLE patients are undergoing intensive or long-term immunosuppressive therapy, as suppression of the immune system can reduce the sensitivity of HIV screening tests and cause false negative results (Tsoi, 2022). Therefore, physicians need to rely on comprehensive clinical evaluation, consider patient risk factors, and not solely depend on screening test results. To confirm the diagnosis, real-time HIV RNA PCR testing is the most sensitive method and can serve as the gold standard for confirmation (Lam, 2022).

In this case, the first HIV screening was performed prior to cyclophosphamide therapy in 2018, with a negative result. During antenatal care in March 2021, HIV screening yielded an indeterminate result, which remained indeterminate when repeated in October 2021. In February 2023, the HIV screening test returned a reactive result (807.1), subsequently confirmed on February 24, 2023, by PCR viral load, which detected 1,488,679 copies of HIV. As risk factors for HIV infection, the patient had a history of sexual relations with a partner in 2017 for two years, followed by her late husband from 2019 until their marriage in January 2021. The husband passed away in December 2021 due to a pulmonary illness; however, his HIV status was unknown. It was noted that he had a history of frequent sexual relations with commercial sex workers prior to 2019.

Systemic Lupus Erythematosus (SLE) is a systemic autoimmune disease characterized by inflammation in various organs of the body. This condition arises due to the production of pathogenic autoantibodies that target nucleic acids and their binding proteins, reflecting a global loss of self-tolerance (Shlomchik, 2001). In its pathogenesis, neutrophils and apoptotic cells play an important role because they present antigens that then trigger plasmacytoid dendritic cells (pDC) to express high levels of type I interferon (IFN-I) (Tsoko, 2016). These self-antigens are then recognized by B cells and CD4+ T cells in secondary lymphoid organs, which subsequently produce pathogenic autoantibodies. Autoantibodies, together with inflammatory cytokines, play a role in exacerbating tissue damage in lupus. Dendritic cells play a crucial role as they act as a link between the innate and adaptive immune systems through antigen presentation and proinflammatory cytokine secretion. The continuous formation of autoantibodies and cytokines activates the innate immune system, thereby reinforcing the inflammatory cycle.

Genetic predisposition is a fundamental factor that enables the abnormal activation of this immune system. However, environmental factors and random events also contribute to triggering the course of the disease (Craft, 2016). In active SLE, a relatively high increase in the number of CD4+ cells is found, for example, reaching 361 cells/µl. In addition, the involvement of pDCs also plays an important role because these cells are strong producers of interferon and are capable of activating immature dendritic cells to increase the presentation of self-antigens to autoreactive T and B cells, thereby reinforcing the autoimmune cycle that underlies tissue damage in SLE (Tsoko, 2016; Jam et al., 2025).

The pathogenesis of HIV infection differs from the mechanism in SLE because HIV directly reduces the number of CD4+ T cells. One of the main ways HIV weakens the immune system is by infecting and destroying CD4+ T cells, which ultimately leads to immunodeficiency in the late stages of the disease. The HIV virus attaches to the CD4+ protein on the surface of these cells and other cells in order to enter them. However, the presence of CD4+ molecules alone is not sufficient for HIV to infect other cells, such as monocytes and dendritic cells, so additional factors are required for infection to occur (Chun, 2012).

In addition, a decrease in the number of Plasmacytoid Dendritic Cells (pDCs), which produce interferon gamma (IFN-γ), in HIV infection may contribute to the protective effect of HIV against the development of SLE. Dendritic cells themselves can be targets of HIV infection, and changes in the TLR9 signaling pathway that affect pDC function may also be influenced by the HIV gp120 protein. The loss of pDCs

during HIV infection generally parallels the reduction in CD4+ T cells, although the rate of recovery between the two may differ. Interestingly, the emergence of SLE after PegIFNα therapy in patients with HIV/HCV infection highlights the importance of pDCs in the immune mechanisms linking the two conditions (Gindea, 2010).

In addition, in SLE, the autoimmunity mechanism involves autoreactive T cells that produce IL-17, a proinflammatory cytokine that recruits and activates neutrophils to attack body tissues, as well as autoreactive B cells that produce autoantibodies, which then form immune complexes and cause organ damage (Tsokos, 2011). However, in the active phase of HIV, there is a general decline in immune function, including damage to CD4+ T cells and impaired function of plasma dendritic cells (pDCs), which should play a role in activating the immune response (Indrajit, 2005). Therefore, theoretically, autoimmune activity such as in SLE may be reduced due to a weakened adaptive immune response. Although this logic makes sense, clinical evidence regarding the relationship between HIV and SLE is still rare. Epidemiological studies show that the coexistence of HIV and SLE is very rare, making it difficult to draw definitive conclusions about how the two diseases influence each other (Iordache, 2014). Interestingly, case reports from Calabrese (2005) found that in 11 patients with severe HIV, SLE symptoms actually experienced spontaneous remission, as if the suppression of the immune system by HIV "inhibited" the autoimmune process.

In this case, the patient presented with active HIV, evidenced by the opportunistic infection miliary TB as a manifestation of AIDS. However, the patient's current SLE activity, based on the SLEDAI score, was low at 2, supported by normal C3 158 and C4 58 levels.

Management of patients with SLE who also have HIV is complex because the underlying mechanisms of these two diseases are opposite. In SLE, standard therapy generally uses immunosuppressive drugs to suppress autoimmune activity, such as corticosteroids or cytotoxic agents. However, in HIV patients, the use of immunosuppressants can actually worsen the condition because their immune systems are already compromised (Lopez, 2012).

Several studies emphasize that immunosuppressive treatment for SLE in HIV patients must be carefully evaluated, particularly regarding the risk of accelerating HIV progression or increasing susceptibility to opportunistic infections. On the other hand, immunosuppressants still have the potential benefit of suppressing persistent autoimmune activity. Therefore, further research is needed to assess the balance between controlling autoimmune symptoms and maintaining HIV viremia control (Carugati, 2013). Since there is no international consensus on treatment guidelines for SLE with HIV, therapeutic decisions must be individualized, taking into account specific clinical conditions, immunological status, viral load, and potential drug interactions with antiretroviral therapy (ART). With this approach, it is hoped that physicians can balance the risks and benefits in choosing the safest and most effective strategy (Gindea, 2010).

Corticosteroids are the primary therapy for suppressing inflammation and autoimmune activity, but the metabolism of these drugs is greatly influenced by the CYP3A4 enzyme in the liver. Some ARV drugs, including protease inhibitors and NNRTIs, can inhibit or modulate CYP3A4 activity, resulting in significant drug interactions. For example, delavirdine and some protease inhibitors can increase systemic corticosteroid levels by inhibiting metabolism, potentially causing systemic side effects (Christiaans, 2001). Conversely, dexamethasone itself induces CYP3A4, thereby decreasing the plasma concentration of certain ARVs, requiring dose adjustments to maintain the effectiveness of both drugs (De Maat, 2003). The clinical impact of high-dose corticosteroid use in HIV patients is quite serious. Studies in HIV transplant recipients have shown an increased risk of opportunistic infections, osteoporosis, and vascular necrosis of the femur, due to the immunosuppressive and metabolic effects of glucocorticoids (Izzedine, 2004). Therefore, a safer strategy is to use low-dose corticosteroids, but with close monitoring of patients with severe immune suppression due to AIDS, so that side effects can be minimized without compromising control of autoimmune diseases (Lopez, 2012).

In HIV-positive patients requiring immunosuppression, such as those with SLE or solid organ transplantation, drug selection must consider

the effects on viral replication and potential interactions with ART. Cyclosporine A and MMF are relatively safe for use in HIV patients, as they do not significantly worsen viral control and have been successfully used in transplant patients with ART (Stock, 2003). In contrast, azathioprine can stimulate HIV replication and should therefore be used with caution (Lang, 2003). However, because azathioprine is metabolized primarily in tissues and plasma, the possibility of metabolic interactions with ARVs in the liver is very low (Anstey, 1998).

MMF has a unique mechanism that not only suppresses lymphocyte and monocyte proliferation through IMPDH inhibition, but also exhibits anti-HIV activity in vitro. The IMPDH isoform in active lymphocytes is more sensitive to MMF, making this drug more effective at suppressing immune cells that play a role in viral replication (Carr, 1993). The combination of MMF and abacavir produces a synergistic effect against HIV-1, especially in resistant viruses, as both drugs interfere with intracellular guanosine metabolism. However, MMF can reduce the effectiveness of other drugs such as zidovudine and stavudine, so combination therapy must be carefully considered (Margolis, 2002).

Hydroxychloroquine (HCQ) is often used in autoimmune diseases such as SLE due to its anti-inflammatory and mild immunosuppressive effects. In patients who are also infected with HIV, HCQ is considered safe because it does not increase the risk of opportunistic infections, which is a major concern when using immunosuppressive drugs in this population (Sperber, 1996). In addition to its anti-inflammatory effects, HCQ has antiviral activity against HIV. In vitro, this drug can inhibit the post-transcription stage of HIV-1, which reduces viral production in monocytes and T cells—the main cells infected by HIV (Sperber, 1993). Furthermore, in HIV patients undergoing effective antiretroviral therapy (ART), HCQ can reduce residual inflammation that persists even when the virus is controlled, thereby providing additional benefits in reducing long-term immunological complications (Picconi, 2011).

In this case, the immunosuppressant regimen chosen was with low-dose steroids (1mg/kg) namely prednisone 40mg-0-0, discontinuation of the azathioprine regimen, and continuation of hydroxychloroquine 200mg-0-0.

Immune Reconstitution Inflammatory Syndrome (IRIS) is a clinical condition that occurs when the previously compromised immune system of HIV patients recovers due to ART and causes an excessive immune response to antigens, both from pathogens and autoantigens. In patients with SLE, IRIS can trigger the recurrence of autoimmune diseases or cause new symptoms, which can be local (e.g., on the skin) or systemic (Carugati, 2013).

Several studies support this phenomenon. Calabrese (2005) reported that after starting ART, four patients experienced a flare-up of SLE, while eight patients showed new symptoms of SLE, indicating that the restoration of immune function can trigger autoimmune reactivation. IRIS can also mask previously undiagnosed autoimmune diseases, so that symptoms that appear after ART can be misinterpreted if not suspected (French, 2004). Although IRIS most commonly occurs within the first three months of ART (Dhasmana, 2008), long-term reports indicate that recurrence or the onset of new SLE can occur up to several years after ART (Mody, 2014). Therefore, routine and careful clinical monitoring is necessary for HIV patients with autoimmune diseases to detect flare-ups or the onset of new autoimmune diseases due to IRIS, and to adjust treatment strategies if necessary.

In this case, the ART used was the TLD (Tenovofir – Lamivudine – Dolutegrafir) regimen because the patient was actively breastfeeding, with regular monitoring of the patient so that they were asked to have regular check-ups every 2-3 weeks at the clinic to monitor the possibility of IRIS side effects or flare-ups of SLE.

In a cohort study, the long-term prognosis of SLE patients with and without AIDS infection was assessed. The results showed that medium to long-term survival rates (5–10 years) were similar between SLE patients with AIDS and those without AIDS. This suggests that the presence of HIV/AIDS does not always worsen the prognosis of SLE patients in the context of modern treatment (Hax, 2018). However, the SLICC (Systemic Lupus International Collaborating Clinics Damage Index) score, which measures cumulative organ damage due to disease or therapy, was higher in SLE patients with AIDS. This indicates that although patients may live for a similar length of time, the burden of organ damage may be

greater in patients who are also infected with HIV/AIDS. Although this difference is statistically significant, its clinical implications remain uncertain, as it does not directly translate into differences in survival. In other words, patients may experience more complications or organ damage without directly affecting survival rates.

Conclusion

A 32-year-old woman presented to the emergency department complaining of chronic diarrhea, loss of appetite, weight loss of 5 kilograms over 2 months, and cough and shortness of breath for 1 month. The patient had a history of SLE since 2013 and HIV risk factors, including a deceased partner and a history of repeated contact with commercial sex workers. The HIV diagnosis was established through antibody serology, which was then confirmed by viral load PCR. Currently, there are no standard guidelines for managing patients with AIDS concurrent with SLE, as HIV treatment tends to increase immune activity, while SLE treatment is immunosuppressive, so therapy for these two conditions can be contradictory. Therefore, an individualized treatment strategy based on the patient's unique condition is required. Additionally, since HIV and SLE symptoms often overlap, and HIV serology testing in autoimmune patients can yield false-positive or false-negative results, clinicians should continue to consider HIV as a differential diagnosis or comorbidity in patients with autoimmune diseases, taking into account existing risk factors.

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