Research Article

Management of Systemic Lupus Erythematosus Patients With Secondary HIV Infection

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Abstract

Objective: To describe the diagnostic and therapeutic challenges of concurrent systemic lupus erythematosus (SLE) and human immunodeficiency virus (HIV) infection, an uncommon coexistence marked by overlapping clinical features and complex immunological interactions, through a case report illustrating the bidirectional relationship between the two conditions. **Materials and Methods:** This report details the case of a 32-year-old woman with lupus nephritis who was diagnosed with advanced HIV infection. Her history of frequent hospitalizations and repeated blood transfusions was identified as a potential risk factor for HIV transmission. Clinical management encompassed immunosuppressive therapy, broad-spectrum antibiotics, and supportive care, alongside evaluation for opportunistic infection. **Results:** The patient subsequently developed severe complications, including acute decompensated heart failure, respiratory failure, opportunistic tuberculosis pleuritis, and profound immunosuppression with a critically low CD4 count. Despite intensive medical management, she experienced progressive respiratory deterioration during hospitalization, and the initiation of anti-tuberculosis therapy was further complicated by multiorgan dysfunction and severe immune compromise. **Conclusion:** This case underscores the complex bidirectional relationship between SLE and HIV, wherein immunosuppressive therapy for SLE may accelerate HIV progression, while HIV-associated immune dysregulation can exacerbate autoimmune disease activity. Early HIV screening in patients presenting with severe or atypical SLE manifestations is essential to optimize management strategies, and a multidisciplinary, individualized approach is required to balance immunosuppression and infection control in patients with concurrent SLE and HIV.

Keywords: Systemic Lupus Erythematosus, HIV Infection, Opportunistic Infection, Tuberculosis Pleuritis.

1 Introduction

The coexistence of systemic lupus erythematosus (SLE) and HIV infection is rare and presents significant diagnostic and management challenges. Over the past three decades, HIV has evolved from a fatal and highly stigmatized disease to a manageable chronic condition due to advances in antiviral therapy [1]. These treatments have markedly increased CD4+ cell counts and reduced AIDS-related complications [2]. However, as HIV becomes a chronic condition, healthcare providers are increasingly confronted with autoimmune diseases such as SLE, which can present complex symptoms and complicate management. Autoimmune diseases may affect multiple organs, including the lungs, thyroid, liver, and hematologic system, necessitating accurate diagnosis and treatment in the context of HIV [3], [4], [5].

SLE is a well-known autoimmune disorder with an unclear cause, although recent research suggests a potential link to various viruses, including retroviruses. In particular, recent findings suggest that Human Endogenous Retrovirus (HERV) can trigger SLE. In particular, there is significant overlap between HIV infection and SLE in terms of immune response characteristics and clinical symptoms, sometimes making it difficult to differentiate. However, it is still rare for a patient to have both HIV and SLE infections [6].

Systemic lupus erythematosus is driven by CD4-positive T cells, yet HIV-1 targets and eliminates this cellular population, potentially lowering SLE susceptibility on theoretical grounds. High mortality attributable to advancing immune compromise in the initial phase of the HIV-1 pandemic gave rise to the hypothesis that autoimmune diseases like SLE and Rheumatoid Arthritis (RA) become less active owing to declining CD4+ lymphocyte counts [7].

This case may provide insight into the immunological mechanisms of both conditions. Clinicians should carefully evaluate the safety of immunosuppressants for SLE in HIV patients. The coexistence of SLE and HIV presents challenges in diagnosis and treatment, as treatment for one condition can worsen the other [4], [6].

2 Case Report

A patient, Mrs. NY, aged 32 years, female, came to the Emergency Room of RSDS with complaints of shortness of breath since the morning of the SMRS. The shortness of breath was felt to be worse when in the room. Complaints were accompanied by heartburn and nausea, but no vomiting. The patient complained of a fever 1 day before the MRS. Cough and cold were denied. There were no complaints of urinary or fecal incontinence. Swelling in both legs had come and gone for the past 3 months.

Past medical history: The patient has a history of lupus nephritis since January 2024 and has had regular check-ups at the Rheumatology Clinic at Dr. Soetomo Hospital, with a history of therapy Hydroxychloroquine (HCQ) 1x200 mg, Furosemide 1x40 mg, Spironolakton 1x25 mg, Bisoprolol 1x2.5 mg, ISDN 1x5 mg for pain, Ramipril 1x5 mg, Atorvastatin 1x20 mg, Asam folat 1x1 mg, Methylprednisolon 3x16 mg dan mycophenolic acid2x360 mg. History of hypertension for 2 years, no regular check-ups, and a history of heart disease known since February 2024. The patient has a history of more than 5 hospitalizations related to anemia and frequent transfusions. The patient was last hospitalized in the Pandanwangi ward from May 29, 2024, to June 12, 2024, with a diagnosis of SLE, SLEDAI score 4 + CAP PSI Score 42 Risk Score II +, Bilateral Pleural Effusion +, Hypoalbuminemia (Alb 2.38; 2.47; 2.55) +, Anemia normokrom normositer (Hb 8.4; 9; 11) +, DCM (HFrEF, EF 35%) + HT Grade 1 +, candidiasis oris + HIV Naive.

Based on social history, the patient and her husband denied any risky sexual behavior, and her husband had not been tested for HIV. The patient had a history of blood transfusions during her hospitalization. She had been married since 2019 and had no children. She was a housewife.

The patient presented with generalized weakness but remained alert and oriented, achieving a GCS of 456 on examination. Hemodynamic parameters demonstrated hypertension with a reading of 130/106 mmHg, tachycardia at 100 beats per minute with a regular, normally amplified pulse, tachypnea at 28 respirations per minute, afebrile status at 36.7°C axillary, and adequate oxygenation at 99% SpO_2 while receiving 15 LPM via NRM. Inspection of the head and neck revealed neither pallor nor icterus, no cyanosis or jugular venous distention, and absent cervical lymphadenopathy; respiratory distress, however, was evident. Symmetric thoracic movement was observed, and no intercostal or supraclavicular retractions were evident. On cardiac examination, S1 and S2 were audible as single sounds, free of murmurs, gallops, or pericardial rubs, while the cardiac silhouette fell within standard dimensions. Pulmonary examination found vesicular breath sounds in both hemithorax; rhonchi were found but no wheezing in both lung fields. The abdomen presented with a supple contour, normal peristaltic activity, no fluid wave, and neither hepatosplenomegaly nor abdominal pain on palpation. The extremities demonstrated warm, dry, red acral areas with capillary refill of less than two seconds; however, edema was observed in both lower limbs.

Laboratory examination at the Emergency Room of Dr. Soetomo Hospital on June 22, 2024, showed Hb 11.4 g/dL, HCT 34.8%, MCV 84.7fL, MCH 27.7pg, WBC 9,810/mm3, Neutrophils 81.6%, Lymphocytes 9.7%, Platelets 535,000/mm3, BUN 38.4, SK 1.2, GDA 65, Sodium 135, Potassium 4.4, Chloride 100, SGOT 59, SGPT 90, Albumin 2.45. Procalcitonin >100, BGA examination with SM 8 lpm, namely pH 7.50, pCO2 31, pO2 137, HCO3 24.4, BE 1.1, SO2 99, PF Ratio 274. Urinalysis examination: pH 5, Specific Gravity 1.029, Erythrocytes 3+, Glucose negative, Ketone negative, Leukocytes negative, Nitrite negative, Bilirubin negative, Urobilinogen normal, Protein 4+, Amber color, Clear clarity, AC ratio >=300, PC ratio >=0.5. HIV examination using 3 methods on June 11, 2024, was reactive, CD4 15. The results of the pleural fluid analysis were pH 8, WBC 0.25, RBC 0.005, MN 87.2%, PMN 12.8%, Cell count 0.251, Glucose 59.2, Protein 0.71, LDH 116.78.

A chest X-ray on June 22, 2024, revealed pneumonia and right pleural effusion. An abdominal ultrasound on June 3, 2024, revealed bilateral parenchymal kidney disease, ascites, and bilateral pleural effusion. No abnormalities were seen in the liver, spleen, pancreas, bladder, uterus, and right and left adnexa. A chest X-ray on June 22, 2024, revealed Left ventricular dimensions were dilated (LVIDd 5.9 cm) with eccentric LVH (LVDMi 140 g/m², RWT 0.39). Left ventricular systolic function was decreased (LVEF by TEICH 29%, by A4C 31%; by MOD A2C 28% and Biplane 28%). Global left ventricular segmental analysis was hypokinetic. Left ventricular diastolic function was grade I diastolic dysfunction; right ventricular dimensions were normal. Right ventricular systolic function was decreased (TAPSE 1.2 cm), left atrial dimension dilation, normal right atrial dimensions, no thrombus or intracardiac vegetation was found, heart valves: mild MR, mild AR, TR trivial, PR trivial, mild pericardial effusion was found in the basal (0.85 cm) and posterior (0.6 cm) and right pleural effusion. LUS B lines are positive in the left lung base.

The results of the consultation to the Lung TS in the Emergency Room currently obtained a patient with bilateral pleural effusion on the right with an organized impression, on the left with a serous impression that can be related to the underlying disease of SLE, CAP PSI score 92 class IV, HIV naïve. The examination and therapy advice from the Lung TS is O2 SM 8-10 lpm target SpO2 94-98%, Cefoperazone sulbactam injection 2x2 g intravenously,

Ventolin Nebulizer 2x5 mcg every 8 hours, Oral NAC 3x200mg, Oral Cotrimoxazole forte 1x960mg and maintaining bronchial toilet.

Based on anamnesis, physical examination and supporting, currently the patient is diagnosed as Type 1 Respiratory Failure + HIV naive + SLE with Lupus nephritis + ADHF wet and warm + DCM with HFrEF (29%) + RV failure (TAPSE 1.2 cm) + CAP PSI Score 92 Risk Class IV + Organized right pleural effusion with empyema + Left pleural effusion + Minimal pericardial effusion + HT stage II + Hypoalbuminemia (Alb 2.45) + Respiratory alkalosis. The treatment carried out was the administration of 8 lpm mask oxygen, 1900 kcal/day TKTPRG diet (high-protein, high-calorie diet (TKTPRG)), drinking a maximum of 1000 ml/day, Furosemide pump 5 mg/hour, methylprednisolone injection 62.5 mg every 24 hours intravenously, Lansoprazole capsule 30 mg every 24 hours orally, Hydroxychloroquine tablet 200 mg every 24 hours orally, Paracetamol tablet 500 mg every 8 hours orally, acetylcysteine capsule 200 mg every 8 hours orally, Cotrimoxazole forte tablet 960 mg every 24 hours orally, Spironolactone tablet 25 mg every 24 hours orally, Adalat oros tablet 30 mg every 24 hours orally, Sucralfate syrup 15 ml every 8 hours orally and Ventolin nebulizer 1 respule every 8 hours.

3 Results

3.1 Disease Course

Deterioration in breathing difficulty emerged on day three of hospitalization. Clinical findings indicated a generally weakened yet alert patient (GCS 456), with blood pressure 100/64 mmHg without hemodynamic support, pulse 100/min, respiratory frequency 32/min, temperature 37.2°C axillary, and SpO_2 98% while receiving NRM at 15 liters per minute. Pulmonary examination found rhonchi in both lung fields. The patient was catheterized with urine output of 1000 ml/24 hours. Laboratory results: CD4 8.14, CD4 Absolute 15, HIV viral load 780,556, Log 5.89. The results of the TS Cardio consultation in the room, the patient was diagnosed with ADHF wet and warm + DCM with HFrEF (29%) + RV failure (TAPSE 1.2 cm) + HT stg II + Minimal pericardial effusion and received advice on Furosemide 2 amp bolus > PU evaluation in 2 hours (target 200-300 ml) > if no response, repeat bolus furo 4 ampoules > If response, continue furosemide pump 2.5 mg/hour, delayed bisoprolol, uperio 2x50 mg, atorvastatin 1x40mg, spironolactone 1x25mg, adalat oros 1x30mg. Furosemide pump therapy was continued, and the patient received additional Cefoperazone sulbactam 2x2 grams intravenous injection therapy.

Day five of treatment found the patient still experiencing shortness of breath. Clinical evaluation demonstrated a generally weakened but cognitively intact state (GCS 456), with blood pressure 107/69 mmHg without inotropic support, pulse 98/min, respiratory frequency 28/min, temperature 37°C axillary, and SpO_2 98% while receiving NRM at 15 liters per minute. The patient was catheterized with urine production of 900 ml/24 hours. From the examination results, procalcitonin>100, BUN 105.8, creatinine 2.3, SGOT 80, SGPT 149. Blood gas analysis: pH 7.39, pCO2 36, pO2 143, HCO3 21.8, BE -3.2, SO2 99, PF ratio 158.9. The patient was consulted to TS anesthesia related to type 1 respiratory failure, but the ICU was full.

The patient noted improvement in respiratory effort on day six of hospitalization. Clinical findings demonstrated a debilitated but fully oriented individual, maintaining a GCS of 456. Blood pressure was 104/68 mmHg without support, pulse 92x/minute, respiratory rate 24x/minute, axillary temperature 37.1°C, SpO2 98% with NRM 10 lpm. The patient was catheterized with urine output of 1100 ml/24 hours. The results of the plain chest X-ray showed right pleural effusion (relatively the same impression), reticulogranular pattern in both lungs can be interstitial pneumonia or interstitial lung edema. Blood and urine culture results were sterile. Pleural fluid culture results showed acid-fast rod formation, so the pulmonary TS was diagnosed as TB Pleuritis. Additional OAT therapy was recommended for BW 50 kg: 3 tablets of 4-FDC, then adjusted to R 600 mg/H 300 mg every 24 hours and Z 1250 mg/750 mg every 48 hours (or three times per week).

By day eight of treatment, the patient experienced worsening respiratory symptoms. Clinical findings included a debilitated but alert status (GCS 456), blood pressure 101/63 mmHg without inotropic support, pulse 94 beats per minute, respiratory rate 26/min, temperature 36.9°C, and SpO_2 96% on NRM at 15 LPM. Urine output through Foley catheter totaled 700 mL per 24 hours. Arterial blood gas analysis demonstrated pH 7.30, pCO_2 45, pO_2 59, HCO_3 22.1, BE -4.3, SO_2 87%, and a PF ratio of 65.6. The intensive care unit was reported as fully occupied. The patient experienced a worsening of condition on day ten of hospitalization, with reduced alertness and exacerbated respiratory distress. Resuscitation was performed in the ward, but he was unresponsive and pronounced dead.

4 Discussion

SLE is an autoimmune condition characterized by organ and cell damage primarily caused by autoantibodies and tissue-bound immune complexes. In most cases, autoantibodies can be detected several years before the onset of clinical symptoms. Although SLE can occur in persons of any gender, age, or racial background, the majority of patients are reproductive-age women [8]. In the American population, the prevalence rate in women ranges from 20 to 150 per 100,000 individuals [9].

The severity of SLE can range from mild and intermittent to severe and life-threatening. Approximately 85% of patients experience ongoing active disease (even with treatment) or have one or more episodes of active disease each year. Complete and permanent remission, where symptoms disappear without treatment, is rare. Systemic symptoms such as fatigue and muscle/joint pain are common. In severe cases, the disease can cause significant systemic disease requiring glucocorticoid therapy, characterized by symptoms such as fever, extreme weakness, weight loss, and anemia, sometimes accompanied by other organ-specific manifestations. [9].

The patient has a history of lupus nephritis since January 2024 and regularly undergoes check-ups at the Rheumatology Clinic of Dr. Soetomo Hospital. Treatment includes HCQ 200 mg, Furosemide 40 mg, Spironolactone 25 mg, Bisoprolol 2.5 mg, ISDN 5 mg for pain, Ramipril 5 mg, Atorvastatin 20 mg, Folic acid 1 mg, Methylprednisolone 48 mg (3x16 mg), and Mycophenolic acid 720 mg (2x360 mg). The patient also has a history of hypertension for 2 years, but does not undergo regular check-ups, and a history of heart disease that was only discovered in February 2024.

HIV-related clinical status and CD4+ T lymphocyte counts form the dual basis of the CDC classification scheme for adolescents and adults with HIV infection. Per this system, an AIDS diagnosis is assigned to any HIV-infected person whose CD4+ T cell count falls below 200 cells/µL, irrespective of whether symptoms or opportunistic infections are manifest [9].

The AIDS definition is very detailed and primarily designed for surveillance rather than practical patient care. Doctors are encouraged to conceptualize HIV as a disease continuum extending from primary infection through advanced stages, rather than concentrating solely on whether a patient fulfills discrete AIDS criteria [9].

HIV can be spread through contaminated blood transfusions, blood products, and shared drug use equipment. This includes all types of blood components, such as whole blood and blood plasma. In the United States and most developed countries, the risk of HIV transmission from blood transfusions is very low. To reduce the risk of HIV transmission through transfusions, blood is tested for HIV and related markers, donors are asked to avoid donating if they engage in high-risk behaviors, and they are screened for other infections such as hepatitis B and C that can indicate HIV risk. They are also tested for syphilis [9].

Patients were frequently hospitalized more than 5 times due to anemia and frequently underwent blood transfusions.

Immune abnormalities in HIV infection and SLE have several similarities [6]

1. T cell disorders: Both of these conditions are characterized by a reduction in CD4+ T cells and dysfunction of both CD4+ and CD8+ T cells. This results in a lower CD4/CD8 ratio.
2. Polyclonal B Cell Activation (PBA): Both HIV and SLE show increased polyclonal B cell activation.
3. Cytokine and Chemokine Changes: Similar patterns of cytokine and chemokine changes were observed. Specifically, both diseases showed increased production of T helper type 2 (Th2) cytokines (such as IL-4, IL-6, and IL-10) and decreased production of T helper type 1 (Th1) cytokines (such as IL-2).
4. Chemokine Production: In both SLE and HIV, exacerbations are associated with changes in chemokine levels. For example, RANTES levels decrease while MCP-1 levels increase with disease progression or higher HIV viral loads.
5. Role of IL-16: IL-16 is a cytokine that is elevated in both conditions and may contribute to CD4+ T cell abnormalities similar to the HIV envelope protein gp120. IL-16 can enhance CD4+ T cell activation and anergy, and its levels correlate with disease activity in SLE.
6. HLA-DR expression: In both diseases, increased HLA-DR expression on CD8+ T cells correlates with decreased CD4+ T cell numbers. The exact mechanism of this relationship is not fully understood but suggests a link between decreased CD4+ T cells and HLA-DR expression.

Although prevalence data estimate that approximately 400 people in the US have both SLE and HIV-1, only about 20 cases have been reported. HIV viral load has increased dramatically in some SLE patients after immunosuppressive therapy, suggesting a potential protective factor against HIV in SLE [6].

Elevated IL-16 levels in SLE patients may inhibit HIV infection by suppressing HIV-1 transcription through CD4-mediated signaling. Furthermore, high levels of autoantibodies in SLE, which can mimic HIV proteins, may protect against HIV. However, SLE symptoms can sometimes worsen with HIV infection or its treatment, and highly active antiretroviral therapy (HAART) can trigger SLE manifestations due to changes in CD4+ T cells [6].

A substantial overlap exists between SLE clinical features and those attributable to primary or secondary HIV complications: cutaneous signs including alopecia, oral ulcers, and malar rash; systemic symptoms like fever and fatigue; musculoskeletal complaints encompassing arthralgia, arthritis, and myalgia; urinary abnormalities such as hematuria and proteinuria; neuropsychiatric conditions including seizures and psychosis; hematopoietic derangements comprising anemia, leukopenia, lymphopenia, and thrombocytopenia; and serological findings including hypergammaglobulinemia and ANA positivity [3].

Opportunistic infections, which contribute substantially to morbidity and mortality, occur at elevated risk in both SLE and HIV populations. In SLE, opportunistic infections can occur even with stable disease and low-dose steroids or immunosuppressants, suggesting that lymphocyte dysfunction, particularly of T cells, may play a role. Similarly, in HIV, immune restoration disease (IRD) resulting from an increase in CD4+ T cells after HAART can lead to opportunistic infections [6].

In SLE, high levels of ANA and anti-dsDNA antibodies can lead to false-positive HIV test results due to cross-reactivity. Conversely, HIV infection can produce autoantibodies that complicate diagnosis. To prevent misdiagnosis, it is important to use a combination of tests, including Western blot, PCR, and CD4 T cell count. For SLE patients with confirmed HIV, prioritize anti-HIV therapy before initiating immunosuppressive medications, and monitor CD4 T cell counts to adjust treatment dosages. Accurate diagnosis requires careful consideration, with additional tests such as viral load assessment, virus isolation, and CD4/CD8 ratio measurement necessary to differentiate between active SLE, HIV infection, or SLE-like symptoms [4]. Laboratory results: CD4 8.14, CD4 Absolute 15, HIV viral load 780.556 Log 5.89.

Immune deficiencies are divided into primary and secondary, with certain microbes being able to manipulate or deplete the immune system, increasing the risk of secondary infections. HIV, for example, depletes protective immune responses, leading to serious infections by other pathogens. Important components of the immune defense include suppressive cytokines such as IL-10 and TGF-β, as well as regulatory T cells that prevent damage but can cause secondary immune deficiencies by suppressing important responses. The balance between immune activation and suppression, influenced by IL-10 levels and genetic variation, influences susceptibility to secondary infections. An effective immune response to primary infection is crucial for preventing secondary infections, which can be compromised by both microbes and the host's immune response [10].

The results of the pleural fluid culture examination showed the formation of acid-fast rods, so the pulmonary TS was diagnosed as TB Pleuritis. Additional OAT therapy with BW 50kg, 3 tabs, 4 FDC, was proposed, with an adjusted dose of R600mg/H300mg every 24 hours and Z1250mg/750mg every 48 hours or 3x a week.

All HIV-tuberculosis coinfected individuals are advised to initiate antiretroviral therapy (ART) within two months of commencing tuberculosis treatment, whereas those with CD4 counts below 50 cells per µL should begin ART within two weeks. Although early ART initiation at this two-week interval improves survival outcomes in the severely immunocompromised subgroup, it concurrently elevates the risk of paradoxical tuberculosis-associated immune reconstitution inflammatory syndrome (IRIS) [11].

The administration of glucocorticoids and immunosuppressants in SLE is complicated by their capacity to stimulate viral replication and hasten clinical deterioration. Among SLE patients with concurrent HIV, the introduction of HAART may elicit an amplified immune reaction, which can in turn induce disease exacerbations and progression. Thus, treatment must be tailored to each patient to strike a balance between managing HIV infection and suppressing lupus activity [12], [13].

Despite the current paucity of published data, rheumatologic therapy appears to be safely administerable provided that viral activity remains fully suppressed and the CD4 count exceeds 200 cells/mm^3 [14].

5 Conclusion

A 32-year-old woman presented with severe SLE and stage 4 HIV/AIDS with the opportunistic infection tuberculosis. Early diagnosis of HIV is crucial, especially in cases with severe manifestations and when increased immunosuppression is necessary. The management of SLE in HIV-infected individuals presents considerable therapeutic difficulty. Introduction of highly active antiretroviral therapy (HAART) in this population may provoke a robust inflammatory immune reaction, with the potential to induce lupus exacerbations and clinical deterioration. This complex interplay between SLE and HIV arises from overlapping clinical and pathogenic characteristics inherent to both diseases. It is hoped that studying the relationship between these two diseases can lead to improved treatment for each disease, including potential preventive or therapeutic strategies for HIV.

References

[1] F. Apriliyani and U. Wijaya, “Education With Booklets Media on HIV Patients in the Hospital: A Quasi-

Experimental Clinical Trial," Pharmacol. Med. Reports, Orthop. Illn. Details, vol. 1, no. 2, pp. 19–26, 2022, doi: 10.55047/comorbid.v1i2.82.
[2] O. A. Odebiyi, J. K. Oladejo, T. O. Oluyo, and S. W. Omotayo, "Existence and Optimal Control Analysis of HIV/AIDS Model," TRANSPUBLIKA Int. Res. EXACT Sci., vol. 3, no. 4, pp. 44–62, Nov. 2024, doi: 10.55047/tires.v3i4.1493.
[3] V. Hax, A. L. D. Moro, R. R. Piovesan, L. Z. Goldani, R. M. Xavier, and O. A. Monticielo, "Human immunodeficiency virus in a cohort of systemic lupus erythematosus patients," Adv. Rheumatol., vol. 58, no. 1, 2018, doi: 10.1186/s42358-018-0003-2.
[4] H.-Y. Liao, C.-M. Tao, and J. Su, "Concomitant systemic lupus erythematosus and HIV infection," Medicine (Baltimore)., vol. 96, no. 51, p. e9337, 2017, doi: 10.1097/md.0000000000009337.
[5] J. Roszkiewicz and E. Smolewska, "Kaleidoscope of autoimmune diseases in HIV infection," Rheumatol. Int., vol. 36, no. 11, pp. 1481–1491, 2016, doi: 10.1007/s00296-016-3555-7.
[6] I. Sekigawa et al., "Lessons from Similarities between SLE and HIV Infection," J. Infect., vol. 44, no. 2, pp. 67–72, 2002, doi: 10.1053/jinf.2001.0959.
[7] B. S. Naovarat, J. D. Reveille, G. A. Salazar, F. M. Williams, and B. Y. Nguyen, "Systemic lupus erythematosus in the setting of HIV-1 infection: a longitudinal analysis," Clin. Rheumatol., vol. 39, no. 2, pp. 413–418, 2020, doi: 10.1007/s10067-019-04867-w.
[8] S. Y. Purba and H. Hiswani, "The Relationship Between Sociodemographic and HIV Incidence Among 17-65 Age Group at the VCT Clinic of Padang Bulan Community Health Center Medan in 2025," TRANSPUBLIKA Int. Res. EXACT Sci., vol. 4, no. 3, pp. 126–142, Jul. 2025, doi: 10.55047/tires.v4i3.1844.
[9] A. S. Fauci and C. A. Langford, Harrison's Rheumatology. McGraw Hill, 2016.
[10] V. R. Bonagura and D. W. Rosenthal, "Infections that cause secondary immune deficiency," 2020, Elsevier. doi: 10.1016/b978-0-12-816768-7.00049-1.
[11] G. Meintjes, J. C. M. Brust, J. Nuttall, and G. Maartens, "Management of active tuberculosis in adults with HIV," Lancet HIV, vol. 6, no. 7, pp. e463–e474, 2019, doi: 10.1016/s2352-3018(19)30154-7.
[12] N. Manohar and S. S. Prasad, "Use of ChatGPT in Academic Publishing: A Rare Case of Seronegative Systemic Lupus Erythematosus in a Patient With HIV Infection," Cureus, 2023, doi: 10.7759/cureus.34616.
[13] L. E. Vega and L. R. Espinoza, "HIV infection and its effects on the development of autoimmune disorders," Pharmacol. Res., vol. 129, pp. 1–9, 2018, doi: 10.1016/j.phrs.2018.01.005.
[14] K. Walker-Bone, E. Doherty, K. Sanyal, and D. Churchill, "Assessment and management of musculoskeletal disorders among patients living with HIV," Rheumatology, p. kew418, 2016, doi: 10.1093/rheumatology/kew418.