International Journal of Advances in Medicine
Manikasari AS et al. Int J Adv Med. 2026 Jan;13(1):32-36
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20254140

Case Report

Systemic lupus erythematosus in a 20-year-old woman: a case report

Ayu S. Manikasari*, I. Gusti Ayu Wiradari Tedja, Made A. V. Prianggandanni

Department of Clinical Pathology, Wangaya Regional General Hospital, Denpasar, Bali, Indonesia

Received: 25 August 2025
Revised: 10 October 2025
Accepted: 17 October 2025

***Correspondence:**
Dr. Ayu S. Manikasari,
E-mail: ayusandra.warmadewa16@gmail.com

ABSTRACT

Systemic lupus erythematosus (SLE) is a complex autoimmune disease affecting multiple organ systems. It predominantly occurs in women of reproductive age, with a global prevalence ranging from approximately 20 to 150 cases per 100,000 individuals. The diagnosis of SLE can be supported by antinuclear antibody (ANA) testing, which includes an initial screening with immunofluorescence ANA (ANA-IF), followed by an ANA profile to identify specific autoantibodies. A 20-year-old female presented with complaints of easy fatigue, fever, joint pain, and hair loss. Laboratory investigations revealed anemia. Immunological testing demonstrated a positive ANA-IF result with a titer of 1:1000 and a delicate speckled pattern, meeting the EULAR/ACR criteria that consider a positive ANA-IF titer ≥1:80. Based on these clinical and laboratory findings, the patient was diagnosed with SLE and commenced on corticosteroid therapy. Subsequent follow-up showed clinical improvement. This case highlights the importance of careful interpretation of ANA-IF titers and patterns for the early detection of SLE. Additionally, specific autoantibody profiling remains essential to support a more precise diagnosis.

Keywords: Systemic lupus erythematosus, Antinuclear antibody

INTRODUCTION

Systemic lupus erythematosus (SLE) is a complex autoimmune disease that affects multiple organ systems.[1] It is characterized by chronic inflammation and tissue damage that can involve nearly every organ in the body. The exact etiology of SLE remains unclear; however, it is widely hypothesized that a combination of genetic, environmental, and hormonal factors contributes to the onset of the disease. SLE predominantly affects women, with a female-to-male ratio of approximately 9:1, and can occur at any age, though it is most commonly diagnosed between the ages of 10 and 50 years.

The prevalence of SLE varies globally, ranging between 20 and 150 cases per 100,000 individuals. The disease is more frequently observed among populations of African, American, Asian, Hispanic, and Native American descent compared to other ethnic groups worldwide.[2]

The clinical manifestations of SLE are diverse and may involve multiple organ systems. Common symptoms include cardiovascular complaints such as chest pain caused by pericardial friction; gastrointestinal issues including abdominal pain, nausea, vomiting, diarrhea, ascites, pancreatitis, and dysphagia; hematological abnormalities such as anemia, leukopenia, and thrombocytopenia; as well as cutaneous and mucosal involvement presenting as malar (butterfly) rash, alopecia, discoid lesions, photosensitivity, and oral ulcers.

Musculoskeletal manifestations, most notably arthralgia, are also frequently seen. Neuropsychiatric involvement can manifest as seizures, psychosis, myelopathy, movement disorders, cognitive dysfunction, cerebrovascular disease, anxiety disorders, aseptic meningitis, and delirium. In addition, pulmonary complications such as pneumonia, ocular manifestations including dry eye syndrome and retinal vasculitis, lupus nephritis affecting the kidneys, and reproductive issues such as infertility may also occur.[2,3]

The diagnosis of SLE is supported by laboratory investigations, including routine hematology, erythrocyte sedimentation rate (ESR), comprehensive urinalysis, and biochemical blood tests. Autoantibody assessment is a critical component, particularly the detection of antinuclear antibodies (ANA) by Indirect Immunofluorescence (ANA-IF), which serves as an initial screening tool for autoimmune processes. A positive ANA-IF result prompts further evaluation of specific ANA profiles, including anti-double-stranded DNA (anti-dsDNA), anti-Smith (anti-Sm), anti-Ro/SSA, and anti-La/SSB antibodies. Additional recommended tests include antiphospholipid antibodies, the direct Coombs test, and levels of complement components C3 and C4.[1]

Several classification criteria have been developed to aid in the diagnosis of SLE. One commonly used set is the 1997 American College of Rheumatology (ACR) criteria, which comprises 11 clinical and laboratory parameters; a diagnosis of SLE is established when at least four criteria are met. Another widely accepted system is the 2012 Systemic Lupus International Collaborating Clinics (SLICC) criteria, which includes 17 parameters; SLE can be diagnosed if a patient meets four criteria, with at least one clinical and one immunologic criterion, or through a kidney biopsy confirming lupus nephritis accompanied by positive ANA or anti-dsDNA antibodies in the absence of other criteria.[3] More recently, the 2019 EULAR/ACR classification criteria were introduced, requiring a positive ANA-IF at a titer of ≥1:80 (or equivalent positive result by other methods) with no alternative explanation for the patient's presentation. Diagnosis is confirmed when the total weighted score reaches 10 points or more, provided that at least one clinical criterion is present.[3]

CASE REPORT

A 20-year-old female presented with complaints of weakness and fever persisting for four days, accompanied by persistent joint pain, increased hair loss beyond her usual amount, and nausea. Physical examination revealed a patient who was alert and oriented (compos mentis), with a body temperature of 37.5 °C, blood pressure of 107/72 mmHg, a pulse rate of 98 beats per minute, and a respiratory rate of 20 breaths per minute. Pallor of the conjunctiva and bilateral exophthalmos were also noted.

To support the diagnosis, laboratory investigations were performed, including complete blood count, iron profile, urinalysis, and blood chemistry, with results detailed in the Tables 1-4. Blood typing identified the patient's blood group as A, Rhesus positive. The peripheral blood smear showed hypochromic microcytic erythrocytes with poikilocytosis, including pencil cells, teardrop cells, helmet cells, and target cells, suggestive of iron deficiency anemia versus chronic disease anemia (Figure 1).

Table 1: Hematology.

Variables	5/6/2025	10/6/2025 (Post-transfusion)	Normal value
Leukocytes (10^3/µl)	6.28	18.17 (H)	4.0-10.0
Erythrocytes (10^6/µl)	3.17 (L)	4.82	4.20-5.40
Hemoglobin (g/dl)	5.0 (L)	10.4 (L)	12.0-16.0
Hematocrit (%)	19.0 (L)	33.8 (L)	37.0-47.0
Mean corpuscular volume (fl)	59.9 (L)	70.1 (L)	81.0-96.0
Mean corpuscular hemoglobin (pg)	15.8 (L)	21.6 (L)	27.0-36.0
Mean corpuscular hemoglobin concentration (g/l)	26.3 (L)	30.8 (L)	31.0-37.0
Platelets (10^3/µl)	748 (H)	485 (H)	150-400
RDW-SD (fl)	43.8	65.2 (H)	37-54
RDW-CV (%)	21.5 (H)	26.5 (H)	11-16
Neutrophils (%)	65.1	89.3 (H)	50-70
Lymphocytes (%)	24.8	6.5 (L)	20-40
Monocytes (%)	8.0	4.0	2-8
Eosinophils (%)	2.1	0.1	0-4
Basophils (%)	0.0	0.1	0-1
Neutrophil-to-lymphocyte ratio	2.62	13.76 (H)	<3.13

L-low, H-high

Ultrasonography revealed splenomegaly. Anti-Streptolysin O (ASO) level was elevated at 800 IU/ml. The direct Coombs test was positive, indicating in vivo sensitization of red blood cells by IgG antibodies. In contrast, the indirect Coombs test was negative, indicating the absence of incomplete antibodies or complement in plasma following in vitro incubation with red blood cells. Qualitative ANA immunofluorescence (ANA-IF) testing demonstrated a delicate speckled pattern with a titer of 1:1000. The patient was diagnosed with SLE, hypochromic microcytic anemia likely related to chronic disease with a possible component of iron deficiency, rheumatic fever, and urinary tract infection (UTI).

Treatment included intravenous administration of Methylprednisolone at 62.5 mg twice daily with tapering to 31.25 mg twice daily, followed by oral

Methylprednisolone 16 mg twice daily. The patient also received five units of packed red blood cells (PRBC) transfusion, one unit per day, without any significant transfusion reactions. Post-transfusion results showed an increase in hemoglobin concentration and mean corpuscular volume (MCV), although values remained below the normal reference range.

Table 2: Blood chemistry.

Variables	6/6/2025	Normal value
SGPT (µ/l)	6	0-42
SGOT (µ/l)	17	0-37
Urea (mg/dl)	15	10-50
Blood creatinine (mg/dl)	0.8	0.3-1.2

Table 3: Iron profile.

Variables	6/6/2025	Normal value
Iron ferozzine (SI) (mg/l)	31 (L)	50-170
TIBC (µg/dl)	309	149-491

L-low

Table 4: Urinalysis.

Variables	6/6/2025
Color	Yellow
Leukocyte esterase	500 Leu/ul
Blood	2+
Bacteria	+

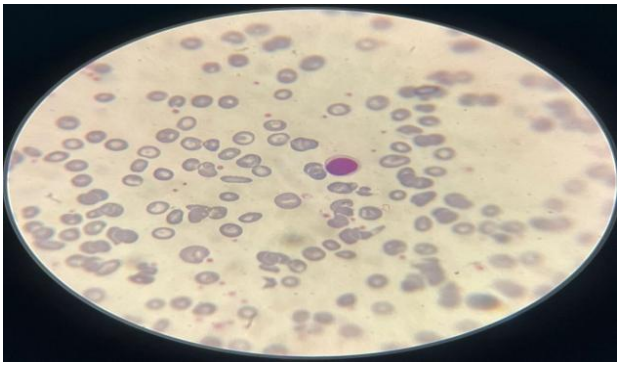

Figure 1: Peripheral blood smear with 100x magnification found lymphocyte nuclei larger than erythrocytes, pencil cells, target cells, helmet cells, and tear drops cells.

DISCUSSION

A 20-year-old female patient presented on the fourth day of symptoms with complaints of weakness, fever, persistent joint pain, increased hair loss, and nausea. Physical examination revealed a body temperature of 37.5 °C, pale conjunctiva, and bilateral exophthalmos. The patient's history and clinical findings revealed several manifestations consistent with SLE, including musculoskeletal involvement with arthralgia and cutaneous symptoms such as alopecia. The presence of pale conjunctiva suggested a potential anemia.

Hematological investigations demonstrated a significant decrease in hemoglobin levels by 5.0 mg/dl, accompanied by reductions in mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and hematocrit. A peripheral blood smear revealed hypochromic microcytic erythrocytes with poikilocytosis, including pencil cells, teardrop cells, helmet cells, and target cells. These findings are consistent with anemia commonly observed in SLE, particularly anemia of chronic disease. In this condition, persistent immune activation and chronic inflammation caused by SLE lead to increased production of inflammatory cytokines, altered iron metabolism, and impaired bone marrow function. Cytokines involved include Interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ), which stimulate hepcidin production and inhibit erythropoiesis. Hepcidin restricts iron release from macrophages and reduces intestinal iron absorption. Chronic inflammation also affects erythropoiesis by inducing resistance to erythropoietin (EPO). Renal EPO production decreases in response to inflammation, while erythroid progenitor cells in the bone marrow exhibit a diminished response. TNF-α and IFN-γ contribute to ineffective erythropoiesis by inducing apoptosis of erythroid precursors. Anemia of chronic disease is typically normocytic or microcytic.4 It is characterized by decreased serum iron and total iron-binding capacity (TIBC), standard or elevated ferritin levels, and low transferrin saturation. Iron deficiency anemia may also occur secondary to chronic inflammation in SLE, which impedes iron absorption. This condition typically presents with low serum iron, low ferritin, reduced transferrin saturation, and increased TIBC. In the present case, a decreased serum iron with normal TIBC was noted; hence, ferritin measurement should be considered as an additional parameter to aid diagnosis.[4]

Another hematologic abnormality observed was thrombocytosis. While SLE commonly induces thrombocytopenia due to autoantibodies targeting platelets (immune thrombocytopenia), in which antibody-platelet complexes are cleared by the spleen, leading to decreased platelet count, thrombocytosis can arise in the context of concurrent infection, iron deficiency, or significant hemorrhage. During severe inflammation and infection, cytokines such as IL-6 stimulate the liver to produce thrombopoietin, which in turn enhances platelet production, resulting in reactive thrombocytosis.[5]

The leukocytosis observed on the fifth day of hospitalization is likely attributable to nosocomial infection. Common infections in SLE patients include urinary tract infections, predominantly caused by *Escherichia coli*, and pneumonia, most frequently due to *Staphylococcus aureus*.[6] The patient's elevated neutrophil-to-lymphocyte ratio (NLR) may reflect disease activity of

SLE, corticosteroid therapy, presence of circulating immune complexes, and type I interferon activity that promotes granulopoiesis in the bone marrow, thereby increasing neutrophil and leukocyte counts.[7]

Direct antiglobulin test (DAT) or direct Coombs test returned positive, indicating in vivo sensitization of red blood cells by IgG antibodies. The indirect Coombs test was negative, indicating that incomplete antibodies or complement were not detected in the plasma following in vitro incubation with red blood cells. This testing is essential for identifying autoimmune hemolytic anemia (AIHA), a known complication of SLE. The positive direct Coombs test suggests antibody-mediated red cell destruction; however, complementary tests such as lactate dehydrogenase (LDH), haptoglobin, direct bilirubin, and reticulocyte count should be performed to confirm hemolysis.[8,9]

Urinalysis revealed leukocyte esterase at 500 leukocytes/µl, a positive blood reaction (+2), and the presence of bacteria, indicating a urinary tract infection (UTI). The incidence of UTI in SLE patients is approximately 28.57%, with *Escherichia coli* involved in 52.2% of cases. UTIs in SLE are generally secondary and precipitated by factors including autoantibodies that impair phagocytic function, reduced complement levels (C3 and C4) that hamper bacterial clearance, immunosuppressive therapy, urinary catheterization, urinary retention due to neuropathy or medication side effects, inadequate hydration, nosocomial infections during hospitalization, and steroid-induced diabetes mellitus.[10]

To further evaluate clinical complaints, qualitative ANA immunofluorescence (ANA-IF) was performed using indirect immunofluorescence assay (IFA) on human epithelial cells (HEp-2) and primate liver substrate, with IgG as the secondary antibody. The test demonstrated a delicate speckled pattern with a titer of 1:1000, fulfilling the EULAR/ACR criteria requiring a positive ANA-IF at ≥1:80. Follow-up testing with an ANA profile is recommended, as the delicate speckled pattern is commonly associated with autoantibodies such as anti-Ro/SSA, anti-La/SSB, anti-Sm, and anti-U1RNP, which are linked with diseases including SLE, polymyositis, Sjögren's syndrome, Mixed connective tissue disease (MCTD), and dermatomyositis. Therefore, specific autoantibody testing is crucial for establishing a more accurate diagnosis and prognosis.[11,12] The criteria for diagnosis of SLE is described in Table 5.[5]

Table 5: Criteria for diagnosis of SLE.

System	ACR (1997)	SLICC (2012)	EULAR/ACR (2019)
Cardiovascular/ pulmonary	Pleuritis or pericarditis	Seroitis	Pleural or pericardial effusion, acute pericarditis
Constitutional	-	-	Fever 100.9 °F (38.3 °C)
Hematology	Hemolytic anemia, leukopenia (<4,000 cells per mm^3), lymphopenia (<1,500 cells per mm^3), or thrombocytopenia (<100,000 cells per mm^3)	Hemolytic anemia, leukopenia (<4,000 cells per mm^3), lymphopenia (<1,500 cells per mm^3), or thrombocytopenia (<100,000 cells per mm^3)	Leukopenia, thrombocytopenia, autoimmune hemolysis
Immunology	Positive results on nuclear antibodies, elevated anti-dsDNA, anti-smith, or antiphospholipid antibodies; discoid rash; photosensitivity; mouth or nose ulcers	Positive results on nuclear antibodies, elevated anti-dsDNA, anti-Smith, or antiphospholipid antibody, low complement (C3, C4, or CH50) or direct Coombs test (without hemolytic anemia); chronic cutaneous lupus; alopecia without scarring; mouth or nose ulcers	Anticardiolipin immunoglobulin G or anti-beta2-glycoprotein 1 antibodies or lupus anticoagulant; low C3 or C4; low C3 and C4; anti-dsDNA or anti-Smith antibodies
Integument/ mucosal	Malar rash	Acute cutaneous lupus or subacute cutaneous lupus	Alopecia without scarring, mouth ulcers, subcutaneous or discoid lupus, cutaneous acute lupus
Musculoskeletal	Nonerosive arthritis involving two or more joints	Synovitis involving two or more joints or tenderness in two or more joints and stiffness for at least 30 minutes in the morning	Joint involvement
Neuropsychiatry	Seizures or psychosis	Seizures, psychosis, mononeuritis complex, myelitis, or peripheral or cranial neuropathy	Delirium, psychosis, seizures

Continued.

System	ACR (1997)	SLICC (2012)	EULAR/ACR (2019)
Renal	Persistent/permanent proteinuria >0.5 g in 24 hours or >3+ without quantitative examination or cellular cylinders: may be erythrocyte, hemoglobin, granular, tubular or mixed cylinders	Urine creatinine (or 24-hour urine protein) >500 mg or, erythrocyte cylinders	Proteinuria >0.5 g in 24 hours, lupus nephritis class II or V, lupus nephritis class III or IV

CONCLUSION

The patient was diagnosed with SLE based on the results of history taking, physical examination, and supporting examination. The patient's laboratory findings that support the patient's diagnosis are ANA IF examination, but to determine specific autoantibodies in patients, it is necessary to consider ANA profile examination to support the diagnosis and prognosis of the patient's disease.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Perhimpunan Reumatologi Indonesia. Diagnosis dan Pengelolaan Lupus Eritematosus Sistemik. Jakarta: Perhimpunan Reumatologi Indonesia; 2019. Available at: https://reumatologi.or.id/download/diagnosis-dan-pengelolaan-lupus-eritematosus-sistemik/. Accessed on 2 August 2025.
2. Selvaraja M, Abdullah M, Md Shah A, Arip M, Amin Nordin S. Systemic lupus erythematosus (SLE): a review on the prevalence, clinical manifestation, and disease assessment. Prog Microbes Mol Biol. 2020;3(1).
3. Lam NV, Brown JA, Sharma R. Systemic Lupus Erythematosus: Diagnosis and Treatment. Am Fam Physician. 2023;107(4):383-95.
4. Tugonza Akiro F. Anemia of Inflammation vs. Iron-Deficiency Anemia: Diagnostic Challenges and Overlapping Mechanisms. Newport Int J Res Med Sci. 2025;6(2):53-8.
5. Santacruz JC, Mantilla MJ, Rueda I, Pulido S, Rodriguez-Salas G, Londono J. A Practical Perspective of the Hematologic Manifestations of Systemic Lupus Erythematosus. Cureus. 2022;14(3):e22938.
6. Restrepo-Escobar M, Granda-Carvajal PA, Aguirre-Acevedo DC, Jaimes F, Vásquez GM. Predictive factors of hospital-acquired bacterial infections in patients with systemic lupus erythematosus. J Clin Rheumatol. 2023;29(5):240-4.
7. Han BK, Wysham KD, Cain KC, Tyden H, Bengtsson AA, Lood C. Neutrophil and lymphocyte counts are associated with different immunopathological mechanisms in systemic lupus erythematosus. Lupus Sci Med. 2020;7(1):e000382.
8. Berentsen S, Barcellini W. Autoimmune Hemolytic Anemias. N Engl J Med. 2021;385(15):1407-19.
9. Ramesh TV, Ayyaswamy G, Sangeetha Z, Kumar VP. Autoimmune Hemolytic Anemia in Systemic Lupus Erythematosus: Clinical Presentation, Diagnostic Challenges, and Management Strategies. J Angiother. 2022;6(1):1-5.
10. Azad MAK, Islam MN, Choudhury MR, Haq SA, Shahin A, Rashid FB, Sharmin F. Urinary Tract Infection in Systemic Lupus Erythematosus: Report from A Developing Country. Mymensingh Med J. 2025;34(1):141-52.
11. Fanouriakis A, Tziolos N, Bertsias G, Boumpas DT. Update on the diagnosis and management of systemic lupus erythematosus. Ann Rheum Dis. 2021;80(1):14-25.
12. Tešija Kuna A, Đerek L, Drvar V, Kozmar A, Gugo K. Assessment of antinuclear antibodies (ANA): National recommendations on behalf of the Croatian society of medical biochemistry and laboratory medicine. Biochem Med (Zagreb). 2021;31(2):020502.

Cite this article as: Manikasari AS, Tedja IGAW, Prianggandanni MAV. Systemic lupus erythematosus (SLE) in a 20-year-old woman: case report. Int J Adv Med 2026;13:32-6.