DEVOTION
Journal of Research and Community Service

Volume 7, Number 2, February 2026
e-ISSN: 2797-6068 and p-ISSN: 2777-0915

Acute Lupus Pneumonitis Mimicking Pneumonia: Diagnostic Challenges and Management in A 40-Year-old Sle Patient

Ni Made Dwi Wulandari[1], Putu Dyah Widyaningsih[2]

[1]Intern Physician, Division of Pulmonology and Respiratory Medicine, Sanjiwani General Hospital, Gianyar, Bali, Indonesia.

[2]Pulmonologist, Division of Pulmonology and Respiratory Medicine, Sanjiwani General Hospital, Gianyar, Bali, Indonesia.

Email: nimadedwiwulandari@gmail.com

KEYWORDS

Acute Lupus Pneumonitis, SLE, Gagal Napas, Kortikosteroid, ARDS.

ABSTRACT

Acute Lupus Pneumonitis (ALP) is a rare pulmonary complication of Systemic Lupus Erythematosus (SLE) with a mortality rate of up to 50%. The greatest challenge for clinicians is identifying this condition amid symptoms that closely resemble severe pneumonia or pulmonary edema, to ensure appropriate management. This case report aims to describe the diagnostic challenges, clinical presentation, and therapeutic response of ALP in an SLE patient presenting with acute respiratory failure, and to emphasize the importance of early high-dose corticosteroid therapy. A 40-year-old woman with SLE presented with progressive dyspnea, cough, and fever lasting one week. She rapidly developed severe hypoxemic respiratory failure, with an oxygen saturation of 58% on room air and a PaO_2/FiO_2 ratio of 42 mmHg, consistent with severe ARDS. Chest X-ray revealed extensive bilateral infiltrates with a normal cardiac silhouette. Laboratory findings showed thrombocytopenia and lymphopenia, suggesting active lupus disease activity, while negative culture results excluded bacterial infection. The patient received High-Flow Nasal Cannula (HFNC) therapy and immediate pulse-dose methylprednisolone. Within 24 hours, oxygenation improved markedly (PaO_2/FiO_2 ratio 162 mmHg). After 10 days, she achieved full recovery and was discharged without oxygen supplementation. Acute Lupus Pneumonitis should be suspected in SLE patients presenting with severe hypoxemia and bilateral infiltrates after excluding cardiogenic and infectious causes. Early initiation of high-dose corticosteroids is crucial to reducing mortality. A systematic diagnostic approach combined with prompt immunosuppressive therapy is essential for achieving optimal outcomes.

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is an autoimmune disease that can affect multiple organs, and its manifestations vary greatly among individuals (Keane & Lynch, 2000). Pulmonary involvement is not only frequently encountered but may also occur early and persist throughout the course of the disease, ranging from mild pleuritis to severe, life-threatening conditions. In a recent study, it was reported that 20–90% of SLE patients experience pulmonary disorders during their lifetime. (Shin et al., 2022) Based on research conducted by Prof. Handono Kalim and colleagues in Malang, the prevalence of Systemic Lupus Erythematosus (LES) in the community is reported to be 0.5% of the total population. [10] Systemic lupus erythematosus (SLE) is a complex disease influenced by various factors, including genetic predisposition, environmental exposure, and dysregulation of both innate and adaptive immune responses. The pleiotropic nature of SLE can result in diverse clinical

manifestations and multi-organ involvement, ranging from mild mucocutaneous symptoms to severe, life-threatening systemic complications (Cintawati, Yunivita, Hamijoyo, & Sahiratmadja, 2025).

One form of pulmonary complication is Acute Lupus Pneumonitis (ALP). Although its prevalence is relatively low—approximately 2–4% of SLE patients—this manifestation is associated with a high fatality rate, with mortality exceeding 50% in some case reports. ALP is often challenging for clinicians because its initial symptoms, such as fever, cough, dyspnea, and hypoxemia, closely resemble severe pneumonia or Acute Respiratory Distress Syndrome (ARDS). (Hannah & D'Cruz, 2019) Moreover, the radiological findings of ALP, including bilateral infiltrates, ground-glass opacities, and pleural effusion, are generally nonspecific (Mathai & Danoff, 2016).

The primary challenge in the early phase lies in distinguishing ALP from infectious and cardiogenic causes, particularly because SLE patients are frequently immunosuppressed and therefore highly susceptible to infections. Research by Mathai and Danoff emphasizes that the diagnostic workup should include thorough infection screening, bronchoalveolar lavage, and high-resolution imaging studies before initiating aggressive immunosuppressive therapy. Delays in administering high-dose corticosteroids can worsen the prognosis and increase the risk of permanent complications, such as pulmonary fibrosis. (Hannah & D'Cruz, 2019) Data regarding the histopathological features of acute lupus pneumonitis are limited; however, several reports have described lymphocytic infiltration and alveolar damage accompanied by interstitial edema, observed in both lung biopsy samples and post-mortem findings (Amarnani, Yeoh, Denneny, & Wincup, 2021).

Although ALP is a rare manifestation, patients may demonstrate a rapid and remarkable response to treatment. (Mohamed, Hammam, El Zohri, & Gheita, 2019) reported clinically significant improvement within 24 hours following methylprednisolone pulse therapy. These findings suggest that early symptom recognition, accurate interpretation of imaging studies, and prompt initiation of therapy are crucial determinants of successful outcomes (Lazovic, Zlatkovic-Svenda, Jasarovic, & Stevanovic, 2018). Despite the availability of several case reports on ALP, a significant gap remains in the literature regarding the detailed clinical trajectory, diagnostic reasoning process, and therapeutic response patterns—particularly within the Indonesian population. Most existing studies focus on Western cohorts or do not comprehensively describe the step-by-step exclusion of differential diagnoses, the integration of microbiological and echocardiographic evidence, and the objective quantification of respiratory improvement using serial PaO_2/FiO_2 ratio monitoring. Furthermore, no previous case report has explicitly demonstrated the correlation between the timing of corticosteroid administration and the rapid reversal of ARDS-equivalent severe respiratory failure in ALP within the first 24 hours, supported by objective hemodynamic and laboratory parameters (Serio, Arnaud, Mathian, Hausfater, & Amoura, 2014).

The novelty of this study lies in its comprehensive and systematic documentation of the diagnostic exclusion process, objective measurement of therapeutic response through serial PaO_2/FiO_2 ratio monitoring, and demonstration of a life-saving outcome achieved through timely high-dose corticosteroid administration in a resource-limited setting (Amarnani et al., 2021). This approach provides a replicable diagnostic and therapeutic framework for clinicians in similar contexts. This study aims to describe the clinical presentation and diagnostic

challenges of Acute Lupus Pneumonitis in a 40-year-old female patient with SLE who developed severe respiratory failure; to illustrate the step-by-step exclusion of infectious and cardiogenic etiologies through microbiological, echocardiographic, and radiological evaluations; to evaluate the therapeutic response to high-dose methylprednisolone and High-Flow Nasal Cannula (HFNC) support using objective parameters—particularly the PaO_2/FiO_2 ratio; and to propose a practical clinical algorithm for the early recognition and management of ALP in comparable healthcare settings. Theoretically, this case report enriches the limited body of knowledge on Acute Lupus Pneumonitis, especially within the Indonesian context, and provides objective evidence of the rapid reversibility of severe hypoxemia following timely immunosuppressive therapy. Practically, it offers clinicians a clear diagnostic and therapeutic workflow that emphasizes early corticosteroid administration once infection has been reasonably excluded. For future researchers, this case may serve as a foundation for subsequent investigations, including multicenter case series or cohort studies to identify predictors of ALP and optimal corticosteroid dosing strategies, while also underscoring the essential role of multidisciplinary collaboration among emergency physicians, intensivists, internists, and radiologists in managing complex autoimmune emergencies.

METHOD

This study is a case report describing the diagnostic process, clinical management, and therapeutic outcomes of a patient with Acute Lupus Pneumonitis (ALP) at RSUD Sanjiwani, Gianyar, Bali, during hospitalization from April 14 to April 24, 2024.

Case Report

A 40-year-old woman came to the Emergency Installation with complaints of shortness of breath that had worsened since the last two days. Symptoms begin with cough with phlegm and fever that the patient has felt for one week before being admitted to the hospital. Patients do not complain of chest pain or swelling in the legs. A previous history of the disease indicates that the patient has systemic lupus erythematosus (SLE) who has been diagnosed since 2013 and routinely follows treatment under the supervision of a consultant internal medicine specialist. The routine medication taken by the patient is methylprednisolone 4 mg once a day orally.

At the initial physical examination in the emergency room, the patient appeared to be seriously ill with composing awareness (GCS E4V5M6). Vital signs indicate respiratory instability, with blood pressure of 120/80 mmHg, pulse of 146 times/minute, breathing rate of 30 times/minute, and critical oxygen saturation of 58% in room air. Thoracic examination showed vesicular breathing sounds with coarse wet rumblings in both lung chambers, without wheezing or heart murmurs. No edema was found on the extremities.

Diagnostic Assessment

A series of supporting examinations are performed to determine the cause of acute respiratory failure and rule out the possibility of an appeal diagnosis.

Blood Gas Analysis (AGD):

At the time of admission (14/04/2024), the AGD results showed severe type 1 respiratory failure with a pH value of 7.42, pCO_2 28.0 mmHg, pO_2 36.0 mmHg, and HCO_3 19.3 mmol/L. PaO_2/FiO_2 ratio of 42 mmHg, in accordance with the Severe ARDS criteria based on the Berlin Criteria, thus corroborating the suspicion of severe pulmonary parenchyma involvement.

Laboratory Examination:

Laboratory results support the suspected SLE flare with an active inflammatory response. There was thrombocytopenia with an initial platelet count of 104,000/µL which decreased to 76,000/µL on the second day of treatment. In addition, lymphopenia was found with a lymphocyte percentage of 4.3% and an absolute lymphocyte count of 0.36×10^3/µL.

Echocardiography Examination

The results of the echocardiography examination showed normal heart chamber dimensions without left ventricular hypertrophy. Systolic and diastolic function of the left ventricle was within normal limits (EF 67.6%), with good right ventricular function and global heart wall movement. The heart valves and pericardium appear normal, with an estimated right atrial pressure of 8 mmHg, according to the normal echocardiography picture.

Radiological Examination:

On the anteroposterior thoracic photo dated April 14, 2024, a diffuse infiltrate can be seen in both lung fields, with a normal heart silhouette. No signs of acute cardiogenic pulmonary edema were found, so the differential diagnosis could be ruled out. Another finding is the elongasio of the aorta, with the cosophrenic sinuses, diaphragm, and thoracic bones within normal limits.

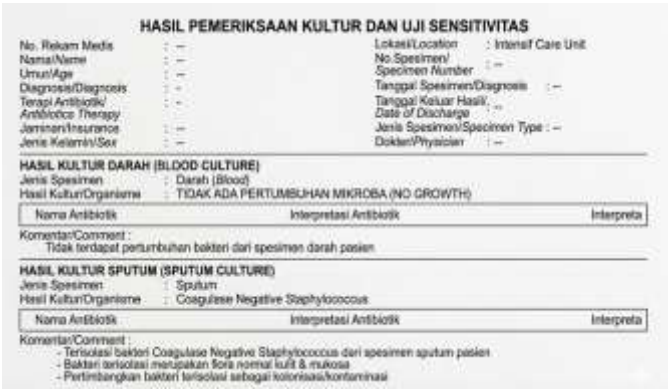


Figure 1. Chest X-ray on Initial Admission Showing Diffuse Bilateral Infiltrates
Source: Medical records of RSUD Sanjiwani, Gianyar, Bali (2024)

Microbiological Examination:

Sputum culture examination conducted on April 15, 2024 showed the growth of Coagulase-Negative Staphylococcus which was assessed as normal flora, so it was not

considered the cause of infection. Meanwhile, blood cultures showed no growth of pathogenic microorganisms (Figure 2).



HASIL PEMERIKSAAN KULTUR DAN UJI SENSITIVITAS

No. Rekam Medis : --
Nama/Name : --
Umur/Age : --
Diagnosis/Diagnosis : -
Terapi Antibiotik/ Antibiotics Therapy : -
Jaminan/Insurance : --
Jenis Kelamin/Sex : --

Lokasi/Location : Intensif Care Unit
No Spesimen/ Specimen Number : --
Tanggal Spesimen/Diagnosis : --
Tanggal Keluar Hasil/ Date of Discharge : --
Jenis Spesimen/Specimen Type : --
Dokter/Physician : --

HASIL KULTUR DARAH (BLOOD CULTURE)
Jenis Spesimen : Darah (Blood)
Hasil Kultur/Organisme : TIDAK ADA PERTUMBUHAN MIKROBA (NO GROWTH)

Nama Antibiotik	Interpretasi Antibiotik	Interpreta

Komentar/Comment :
Tidak terdapat pertumbuhan bakteri dari spesimen darah pasien

HASIL KULTUR SPUTUM (SPUTUM CULTURE)
Jenis Spesimen : Sputum
Hasil Kultur/Organisme : Coagulase Negative Staphylococcus

Nama Antibiotik	Interpretasi Antibiotik	Interpreta

Komentar/Comment :
- Terisolasi bakteri Coagulase Negative Staphylococcus dari spesimen sputum pasien
- Bakteri terisolasi merupakan flora normal kulit & mukosa
- Pertimbangkan bakteri terisolasi sebagai kolonisasi/kontaminasi

Figure 2. Sputum and Blood Culture Results Showing No Pathogenic Growth
Source: Clinical microbiology laboratory, RSUD Sanjiwani, Gianyar, Bali (2024)

The patient was admitted to the Intensive Care Unit (ICU) with a diagnosis of type 1 respiratory failure and a strong suspicion of acute lupus pneumonitis, with pneumonia considered as a differential diagnosis, accompanied by a moderate-degree SLE flare. The patient was then started on oxygen therapy using High-Flow Nasal Cannula (HFNC) with a flow setting of 50 L/min and a FiO_2 of 50–60% to help maintain respiratory function.

Pharmacological management was initiated based on the initial clinical condition and objective findings. Given the possibility of bacterial pneumonia or opportunistic infection as the cause of respiratory failure, the patient was administered empirical antibiotics in the form of Levofloxacin 750 mg intravenously every 24 hours and Ceftazidime 1 g intravenously every 6 hours. This therapy was continued until the eighth day and subsequently discontinued after blood and sputum culture results did not demonstrate the presence of pathogenic organisms.

In an effort to suppress the autoimmune inflammatory process involving the lungs, the patient was administered methylprednisolone at a stress dose (62.5 mg intravenously twice daily). In addition, Hydroxychloroquine 200 mg per day was given as maintenance therapy to control long-term disease activity, reduce the risk of recurrence, preserve organ function, and provide a protective effect against the risk of thrombosis. The role of this drug complements the effects of corticosteroids in suppressing inflammation during the acute phase.

Supportive therapy was also provided according to clinical needs. The patient received N-acetylcysteine as a mucolytic agent, nebulized bronchodilators to improve ventilation, and Furosemide when there were indications of fluid overload or signs of pulmonary congestion.

The patient's response to therapy was significant. The PaO_2/FiO_2 ratio graph illustrates the recovery pattern (Figure 3). From a markedly low value of 42 mmHg at the initiation of treatment, the PaO_2/FiO_2 ratio increased substantially to 162 mmHg on the second day after corticosteroid therapy. The patient's clinical condition showed progressive improvement, allowing gradual reduction of oxygen support to a nasal cannula by day 9. The patient was subsequently discharged on day 11 without the need for oxygen supplementation, maintaining an oxygen saturation of 98% on room air.

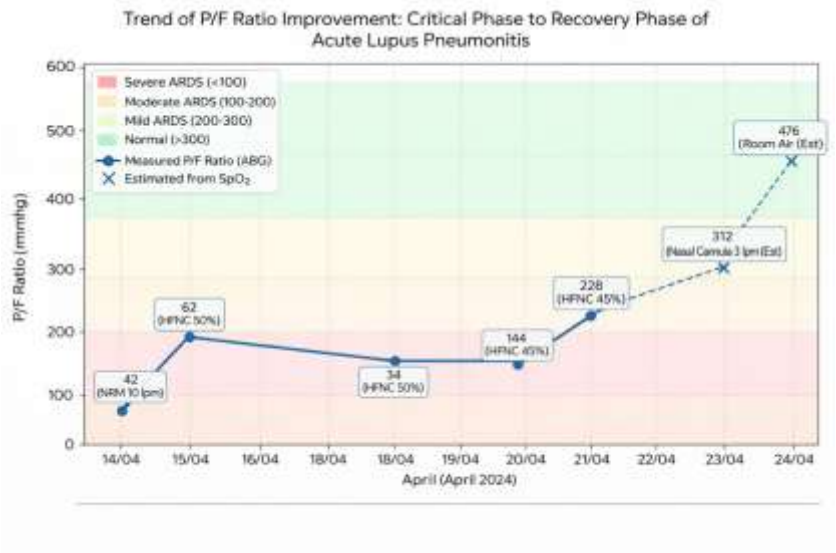


Figure 3. Graph of the Patient's P/F Ratio (PaO_2/FiO_2) During Hospitalization
Source: Primary data processed by the author (2024).

The graph shows a significant improvement from Severe ARDS (Ratio 42) on the first day to Moderate ARDS (Ratio 162) within 24 hours after initiation of high-dose corticosteroids. Although there were slight fluctuations in the weaning phase (April 18-20), it appeared that improvement continued until it reached normal values (>300) on the 10th day (April 23). (NRM: Non-Rebreathing Mask; HFNC: High Flow Nasal Cannula; NK: Nasal Kanul).

RESULT AND DISCUSSION

Acute Lupus Pneumonitis (ALP) is a rare but potentially fatal pulmonary manifestation in patients with Systemic Lupus Erythematosus (SLE). The incidence of this condition is reported in fewer than 2–4% of SLE patients; however, it carries a high mortality rate and may exceed 50% if not treated promptly and appropriately. This condition is often difficult to distinguish from pneumonia, Acute Respiratory Distress Syndrome (ARDS) of infectious etiology, or cardiogenic pulmonary edema due to overlapping clinical symptoms and radiological findings. This diagnostic challenge becomes even more complex because SLE patients are frequently immunosuppressed; therefore, infection remains a competing diagnosis that must be carefully excluded. The clinical presentation of ALP is nonspecific and often resembles pneumonia, with an acute onset of fever, cough, dyspnea, pleuritic chest pain, and, in some cases, hemoptysis.

Physical examination may reveal tachycardia, tachypnea, hypoxemia, hypocapnia, and pulmonary rhonchi. In certain circumstances, these manifestations can progress to acute respiratory failure requiring mechanical ventilation support. (Chattopadhyay, Chatterjee, Maiti, & Debnath, 2015) Chest radiographs may demonstrate bilateral, multiple infiltrates predominantly affecting the lower lobes, with or without pleural effusion. However, in some cases—especially in the early phase of the disease—the chest radiograph may appear normal or show only pulmonary nodules. Although these findings are nonspecific, thoracic computed

tomography (CT) scans may reveal ground-glass opacities and areas of consolidation predominantly located in the lower lobes. In this context, bronchoscopy with analysis of bronchoalveolar lavage fluid (BALF) is recommended, followed by microbiological examination to detect both common and opportunistic pathogens (Emmanuelli, Fernández, & Jiménez, 2020).

Histopathologically, acute lupus pneumonitis (ALP) demonstrates a pattern of diffuse alveolar damage (DAD) characterized by inflammatory cell infiltration, damage and necrosis of alveolar–capillary units, edema, formation of hyaline membranes, and alveolar hemorrhage. In addition, capillary obstruction and thrombosis have also been reported. Alveolar damage is thought to be mediated by the deposition of immune complexes and complement activation. However, no specific histological findings are diagnostic or pathognomonic for ALP.

Some data suggest a possible pathogenic role of anti-Ro/SSA antibodies, based on the association between ALP and the presence of these autoantibodies. In the absence of specific clinical or radiological findings, the diagnosis of ALP is established through an exclusion approach, considering a broad range of differential diagnoses, including infection, organizing pneumonia, malignancy, diffuse alveolar hemorrhage, pulmonary edema, and drug-induced lung toxicity. Infection must always be ruled out because it can present with a similar clinical picture, and the immunosuppressive therapy required in ALP may potentially worsen an underlying infection (Aarabi & McGinn, 2024).

Damage to the alveolar–capillary unit constitutes the principal pathological mechanism in acute lupus pneumonitis (ALP), as this structure plays a critical role in pulmonary gas exchange. The alveolar–capillary unit consists of alveolar epithelium, a basement membrane, and capillary endothelium, which normally form a thin, selective barrier. In ALP, active autoimmune processes trigger immune system activation and diffuse inflammation at the pulmonary microvascular level (Gomez et al., 2024). Deposition of immune complexes and complement activation within the alveolar–capillary unit lead to infiltration of inflammatory cells—particularly neutrophils and lymphocytes—which release proinflammatory mediators and proteolytic enzymes. This process results in endothelial and epithelial injury, increased capillary permeability, and leakage of fluid and proteins into the alveolar space (Martinez-Taboada, Blanco, Armona, Fernandez-Sueiro, & Rodriguez-Valverde, 1995). As a consequence of this injury, alveolar edema, hyaline membrane formation, and, in some cases, alveolar hemorrhage may occur, which histologically correspond to diffuse alveolar damage (DAD). Injury to the alveolar–capillary unit directly impairs gas exchange, resulting in severe hypoxemia that may progress to acute respiratory failure. Thus, acute damage to the alveolar–capillary unit represents a central pathological mechanism in ALP, explaining the link between lupus-related autoimmune activity, the histopathological pattern of DAD, and the rapid onset of severe oxygenation impairment (Mok & Ying, 2004).

This case report highlights a common diagnostic challenge in SLE patients presenting with acute pulmonary infiltrates, namely determining whether the underlying cause is infectious or autoimmune. Acute Lupus Pneumonitis (ALP) is a diagnosis established through the exclusion of other potential causes. In this case, three principal findings support the diagnosis of ALP.

The first consideration was the mismatch between radiological findings and hemodynamic status when compared with cardiogenic pulmonary edema. Although the patient exhibited severe hypoxemia (PaO_2/FiO_2 ratio of 42), initial chest radiographs demonstrated a normal

cardiac size without signs of pulmonary venous congestion, such as Kerley B lines. Hypoxemia that cannot be adequately explained by arterial blood gas analysis in the absence of clear pulmonary parenchymal abnormalities has been reported in hospitalized SLE patients. In a case report by Abramson et al., 22 inpatients were evaluated; six experienced nine episodes of hypoxemia and/or hypocapnia. Improvement in gas exchange was observed within 72 hours following corticosteroid therapy (Sun & Kaplan, 2001).

The second supporting point was the absence of evidence of active infection. Although SLE patients are at high risk of infection due to immune dysregulation and immunosuppressive therapy, microbiological examinations in this case did not support an infectious etiology.

Blood cultures were negative, and sputum cultures revealed only normal flora (Coagulase-negative Staphylococcus). Furthermore, no significant clinical improvement occurred following empirical antibiotic therapy, in contrast to the rapid and marked response observed after corticosteroid administration. These findings strongly suggest an autoimmune process as the primary cause.

The third point supporting the diagnosis of ALP was the excellent clinical response to corticosteroids. High-dose corticosteroids, such as intravenous methylprednisolone, represent first-line therapy in ALP.

In this patient, rapid improvement in oxygenation was observed within the first 24 hours after steroid administration, with the PaO_2/FiO_2 ratio increasing by more than 300%. This response is consistent with the pathophysiology of ALP, which involves immune complex deposition and alveolar vasculitis responsive to potent anti-inflammatory therapy. In addition, the presence of thrombocytopenia and lymphopenia suggests active systemic disease (systemic flare) with prominent pulmonary involvement. High-dose corticosteroids remain the cornerstone of treatment. In severe cases, methylprednisolone may be administered at doses of up to 1,000 mg per day for 3 days, followed by oral prednisone at 1–2 mg/kg/day with gradual tapering according to clinical response. In refractory severe cases, the addition of immunosuppressive agents such as cyclophosphamide and azathioprine; biologic therapies such as rituximab (anti-CD20 monoclonal antibody); intravenous immunoglobulin; or plasma exchange may be considered, although evidence regarding their efficacy remains limited. Broad-spectrum antibiotics are recommended at the initiation of therapy until infection has been reasonably excluded and may also be considered for prophylaxis against opportunistic pathogens, such as Pneumocystis jirovecii, during immunosuppressive treatment. [16]

The use of antimalarial agents, particularly hydroxychloroquine and chloroquine, provides multiple clinical benefits in patients with Systemic Lupus Erythematosus (LES), including improved survival and remission rates, reduced disease activity and infection risk, favorable effects on lipid profiles, prevention of thrombosis, and reduced risk of organ damage. Additionally, hydroxychloroquine has been associated with improved bone mineral density (BMD). Although the patient experienced fluctuations in oxygen saturation between days 5 and 7—possibly related to oxygen weaning or respiratory muscle fatigue—the overall clinical course demonstrated sustained improvement until complete recovery, and the patient was discharged without the need for supplemental oxygen.

CONCLUSION

The third point supporting the diagnosis of Acute Lupus Pneumonitis (ALP) is the excellent clinical response to corticosteroids. High-dose corticosteroids, such as intravenous methylprednisolone, constitute first-line therapy in ALP. In this patient, rapid improvement in oxygenation was observed within the first 24 hours after steroid administration, with the PaO_2/FiO_2 ratio increasing by more than 300%. This finding is consistent with the pathophysiology of ALP, which involves immune complex deposition and alveolar vasculitis that respond to potent anti-inflammatory therapy. Additionally, the concurrent presence of thrombocytopenia and lymphopenia indicates active systemic disease (systemic flare) with significant pulmonary involvement. High-dose corticosteroids remain the cornerstone of treatment. In severe cases, methylprednisolone may be administered at doses of up to 1,000 mg per day for 3 consecutive days, followed by oral prednisone at 1–2 mg/kg/day with gradual tapering according to the clinical response. In refractory severe cases, adjunctive immunosuppressive agents such as cyclophosphamide and azathioprine; biologic therapies such as rituximab (anti-CD20 monoclonal antibody); intravenous immunoglobulin; or plasma exchange may be considered, although scientific evidence regarding their efficacy remains limited. Broad-spectrum antibiotics are recommended at the initiation of therapy until infection has been reasonably excluded and may also be considered for prophylaxis against opportunistic pathogens, such as Pneumocystis jirovecii, during immunosuppressive treatment.

The use of antimalarial agents, particularly hydroxychloroquine and chloroquine, provides multiple clinical benefits in patients with Systemic Lupus Erythematosus (LES), including improved survival and remission rates, reduced disease activity and infection risk, favorable effects on lipid profiles, prevention of thrombosis, and a decreased risk of organ damage. In addition, hydroxychloroquine has been associated with improved bone mineral density (BMD). Although the patient experienced fluctuations in oxygen saturation between days 5 and 7—possibly related to oxygen weaning or respiratory muscle fatigue—the overall clinical course continued to demonstrate significant improvement until complete recovery, and the patient was discharged without the need for supplemental oxygen.

REFERENCES

Aarabi, Amirhossein, & McGinn, Stella. (2024). Severe lupus pneumonitis: A case of life-saving multimodal therapy with rituximab and corticosteroids. *Respirology Case Reports*, *12*(7), e01426.

Amarnani, Raj, Yeoh, Su Ann, Denneny, Emma K., & Wincup, Chris. (2021). Lupus and the lungs: the assessment and management of pulmonary manifestations of systemic lupus erythematosus. *Frontiers in Medicine*, *7*, 610257.

Chattopadhyay, B., Chatterjee, A., Maiti, A., & Debnath, N. B. (2015). Systemic lupus erythematosus presenting as acute lupus pneumonitis in a young female. *Journal of Postgraduate Medicine*, *61*(2), 129–130.

Cintawati, Yane Lis, Yunivita, Vycke, Hamijoyo, Laniyati, & Sahiratmadja, Edhyana. (2025). Pharmacokinetic Profile of Isoniazid and Acetylator Status in Patients with Systemic Lupus Erythematosus: Implications for Tuberculosis Prevention Therapy in Indonesia. *Infection and Drug Resistance*, 3637–3646.

Emmanuelli, Melba Feliciano, Fernández, Ricardo, & Jiménez, Ivonne Z. (2020). *2020*

CLINICAL VIGNETTES COMPETITION.

Gomez, Christian J., Naeem, Muhammad, Bechel, Meagan A., Battle, Wilson, Amin, Sagar, Hamid, Aws, Berkowitz, Eugene, Veeraraghavan, Srihari, & Filev, Peter. (2024). Thoracic and cardiovascular imaging manifestations of lupus. *Radiographics*, *45*(1), e240114.

Hannah, Jennifer R., & D'Cruz, David P. (2019). Pulmonary complications of systemic lupus erythematosus. *Seminars in Respiratory and Critical Care Medicine*, *40*(02), 227–234. Thieme Medical Publishers.

Keane, Michael P., & Lynch, Joseph P. (2000). Pleuropulmonary manifestations of systemic lupus erythematosus. *Thorax*, *55*(2), 159–166.

Lazovic, Biljana, Zlatkovic-Svenda, Mirjana, Jasarovic, Damir, & Stevanovic, D. (2018). Systemic lupus erythematosus presenting as acute lupus pneumonitis. *Arch Bronconeumol*, *54*(4), 220–234.

Martinez-Taboada, V. M., Blanco, R., Armona, J., Fernandez-Sueiro, J. L., & Rodriguez-Valverde, V. (1995). Acute reversible hypoxemia in systemic lupus erythematosus: a new syndrome or an index of disease activity? *Lupus*, *4*(4), 259–262.

Mathai, Stephen C., & Danoff, Sonye K. (2016). Management of interstitial lung disease associated with connective tissue disease. *Bmj*, *352*.

Mohamed, Alaa A. A., Hammam, Nevin, El Zohri, Mona H., & Gheita, Tamer A. (2019). Cardiac manifestations in systemic lupus erythematosus: clinical correlates of subclinical echocardiographic features. *BioMed Research International*, *2019*(1), 2437105.

Mok, C. C., & Ying, K. Y. (2004). Lupus pneumonitis or severe acute respiratory syndrome? *Lupus*, *13*(7), 549–553.

Serio, Ilaria, Arnaud, Laurent, Mathian, Alexis, Hausfater, Pierre, & Amoura, Zahir. (2014). Can procalcitonin be used to distinguish between disease flare and infection in patients with systemic lupus erythematosus: a systematic literature review. *Clinical Rheumatology*, *33*(9), 1209–1215.

Shin, Jae Il, Lee, Keum Hwa, Park, Seoyeon, Yang, Jae Won, Kim, Hyung Ju, Song, Kwanhyuk, Lee, Seungyeon, Na, Hyeyoung, Jang, Yong Jun, & Nam, Ju Yun. (2022). Systemic lupus erythematosus and lung involvement: a comprehensive review. *Journal of Clinical Medicine*, *11*(22), 6714.

Sun, Michelle V, & Kaplan, Peter J. (2001). Acute pneumonia and systemic lupus erythematosus. *Baylor University Medical Center Proceedings*, *14*(1), 88–93. Taylor & Francis.