Lupus Pneumonitis Masquerading As Community Acquired Pneumonia And Pulmonary Tuberculosis: A Case Report

Mateus Pinheiro[1], Charlotte Hall[2,3], Dioneia Benevides Monteiro[1], Nelson Eugenio P.G. Monteiro[1], Terezinha F. P. Ribeiro[4].

[1]Internal medicine department Hospital National Guido Valadares, Dili, Timor Leste

[2]Radiology department Hospital National Guido Valadares, Dili, Timor Leste

[3]Menzies School of Health Research, Dili, Timor-Leste

[4]Leeds Teaching Hospitals NHS Trust, UK

DOI: https://doi.org/10.51244/IJRSI.2025.120800329

Received: 04 Sep 2025; Accepted: 10 Sep 2025; Published: 11 October 2025

ABSTRACT

Introduction

Systemic lupus erythematosus (SLE) is characterized by production of antibodies against various nuclear antigens with involvement of multiple organs. Pulmonary manifestations of SLE can include a wide spectrum of diseases such as pleuritis, pneumonia, pulmonary embolism, pneumothorax and pulmonary haemorrhage. Lupus pneumonitis (LP) has an incidence of 1-8% and may be difficult to distinguish from pulmonary infections. We report a case of LP mimicking community acquired pneumonia (CAP) and pulmonary tuberculosis (PTB) admitted to the Internal Medicine ward in Hospital National Guido Valadares, Dili, Timor Leste.

Case

A 43-year-old female school teacher presented with cough and shortness of breath. She also had a history of fever and malar rash along with significant hair loss. On examination she was febrile and dyspneic, with anaemia, hair loss, malar rash, tachycardia, tachypnoea, and coarse crepitations on chest auscultation. Sputum for Gene Xpert MTB/RIF Ultra test, bacterial culture, and fungal stains were negative. She was initially treated with broad-spectrum antibiotics for CAP, while being investigated for TB. However, as she failed to respond to intravenous antibiotics, further evaluation was done. Anti-nuclear antibodies (ANA) and ds-DNA were strongly positive. Urine analysis revealed nephrotic-range proteinuria. High resolution computed tomography (CT) showed bilateral ground glass changes suggestive of lupus pneumonitis (LP). A diagnosis of SLE with LP was made and the patient was commenced on corticosteroids (pulsed methylprednisolone for three consecutive days, followed by oral prednisolone) which led to a dramatic clinical and radiological response.

Conclusion

SLE has a wide range of presentations. Keeping this in mind, even in countries where tuberculosis is endemic, the differential diagnosis of SLE and LP should be considered. Many challenges exist in the diagnosis and management of patients with SLE and its complications in resource-limited settings.

Keyword's: autoimmune disease, systemic lupus erythematosus, lupus pneumonitis, community acquired pneumonia, pulmonary tuberculosis

INTRODUCTION

SLE (systemic lupus erythematosus) is a multisystem autoimmune disorder which has a waxing and waning course. The clinical manifestations of SLE are variable but include erythematous photosensitive malar rash, oral ulcers, non-erosive polyarthritis or polyarthralgia, polyserositis, immune-mediated cytopenias, renal, neurologic, pulmonary and cardiac abnormalities. Pulmonary manifestations of SLE were first described by Osler in 1904 who described a patient of SLE with persistent lower lobe infiltrates [1]. A wide spectrum of pulmonary

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue IX September 2025

presentations has since been described, including pleuritis, pneumonia, pulmonary embolism, pneumothorax and pulmonary haemorrhage [2]. Though infections are also a frequent cause of pulmonary infiltrates in patients with SLE, in many cases pulmonary infiltrates are not related to infection [1]. Lupus pneumonitis (LP) is an unusual

and potentially life-threatening complication of SLE which has an incidence of 1 % to 8 % (3,13) and usually occurs during SLE flare-ups, with a mortality rate of up to 50%. It is rare during the primary presentation of SLE and may mimic tuberculosis or other acute infectious pneumonia. LP results from immune complex deposition in the lung microvasculature with complement activation and inflammatory cell recruitment that then causes alveolar-capillary injury (12). A high index of clinical suspicion is needed when young adult females present with unexplained pulmonary infiltrates, especially in tuberculosis endemic countries like Timor Leste where use of empirical antituberculosis therapy is high. This is a report of LP as a presenting feature of SLE which mimicked CAP and pulmonary tuberculosis and the diagnostic and therapeutic challenges associated with in a setting of TB-endemic, resource-limited country (12,14).

Case Report

A 43-year-old female school teacher was admitted to the Internal Medicine ward at Hospital National Guido Valadares in Timor Leste with a three-week history of low-grade fever and cough with mucoid expectoration. She had experienced difficulty in breathing for three days prior to admission. She had a history of intermittent swelling of hands and feet and associated joint pain, malar rash and hair loss for the past one year but had not sought medical attention. Systems review revealed history of irregular menstruation but no other features, including no history of weight loss or exposure to any drugs or toxins. She did give history of contact with a case of pulmonary tuberculosis in her family. Past medical history was unremarkable and there was no history of travel in the recent past. She did not use tobacco or drink alcohol.

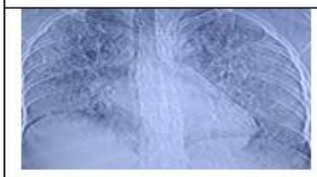
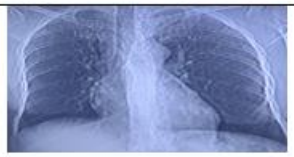
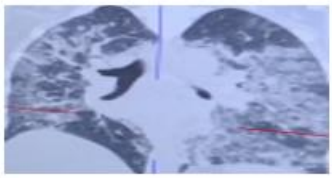
On examination she was fully conscious and oriented but appeared pale and febrile; oral temperature was 38 degrees Celsius. Her blood pressure was 100/60mmHg. She was tachycardic (pulse rate 106/minute), tachypnoeic (respiratory rate 32/minute) and in respiratory distress as evidenced by the use of accessory muscles of respiration. There was presence of grade-2 diffuse alopecia. On respiratory examination, there were diffuse bilateral coarse crepitations on auscultation of her chest. Cardiac examination revealed a sinus tachycardiac with a prominent pulmonary component of the second heart sound. There were no other positive findings on physical examination.

Table 1: Initial laboratory investigation results and ordered ANA and ds-DNA testing from Private clinic after strong suspicion on SLE with LP.

Parameter	Findings	Normal ranges
Laboratory results on admission	ESR 138 mm/hour	0-12 mm/hour
	CRP 80.0 mg/L	0-5 mg/L
	Total white blood cells $6.4x10^9$/L	3.5-10 $X10^9$/L
	Haemoglobin 8.5g/L	12-15 g/L
	Platelets: $95x10^9$/L	150-410 $X10^9$/L
	Albumin 29 g/L	35-50 g/L
	AST 38 U/L	17 -30 U/L
	Creatinine: 142 umol/L	45-90 umol/L
	Urea: 10.4 umol/L	3.0-8.0 mmol/L
	Arterial blood gases with O_2 6L/minute supplementary oxygen via face mask:	
	pH 7.45	7.35-7.45
	pO_2: 60 mmHg	80-105 mmHg
	pCO_2: 29 mmHg	35-45 mmHg
	HCO_3^- : 23 mmol/L	22-27 mmol/L
Antinuclear antibody test after strong suspicion of SLE was made	ANA 115.9 AU/mL	0-40 AU/mL
	dsDNA (435.4 IU/mL)	0-30 IU/mL

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue IX September 2025

Table 2: results of radiological studies on admission and follow up

Initial Imaging studies	Follow up imaging studies
 **Figure 1:** Initial CXR: Bilateral diffuse alveolar infiltrates.	 **Figure 2:** Follow up CXR two weeks after administration of pulsed methylprednisolone and commencement of high dose oral prednisolone: clear lung fields without diffuse or patchy alveolar infiltrates.
 **Figure 3:** Initial CT scan thorax: extensive bilateral ground glass opacities occupying lung fields with some air space consolidation.	 **Figure 4:** Follow-up CT scan taken after two weeks administration of pulsed methylprednisolone and commencement of high-dose oral prednisolone: marked reduction in ground glass opacities and consolidation, areas of clear parenchymal architecture with diminished inflammation with near- complete resolution of previous changes.

Initial investigation and radiology results are shown in Tables 1 and 2. Bloods at presentation showed anaemia, thrombocytopenia, elevated ESR and CRP and hypoalbuminaemia as well as elevated urea and serum creatinine. Coagulation profile was normal. Chest X-ray on admission showed patchy bilateral air space opacification, worse on right side involving all zones of the lung without any cranio-caudal gradient and cardiomegaly with prominent pulmonary conus (see Figure 1). Urinalysis showed gross proteinuria with occasional pus cells.

Sputum for bacteriological culture was sterile; sputum for Gene-Xpert MTB/RIF Ultra assay was repeatedly negative. Sputum microscopy and fungal stains did not reveal presence of fungi. Serological markers for HBV, HCV, HIV and TPHA were non-reactive.

Abdominal ultrasonography revealed mild splenomegaly. Echocardiography showed pulmonary arterial hypertension with a normal left ventricular function.

The patient was initially managed with empirical broad-spectrum antibiotics in line with local guidelines (intravenous ceftriaxone 2 grams daily with azithromycin 500 milligrams oral daily) and high flow oxygen (6L/minute) through face mask for suspected community acquired pneumonia. However, the patient's overall clinical condition failed to improve over the next 72 hours. On grounds of prolonged duration of her illness, history of contact with tuberculosis and radiographic findings of multiple pulmonary opacities, the patient was empirically put on anti-tubercular therapy, despite which her clinical condition did not improve over the following five days.

Further investigations were therefore ordered and tested at a private facility which showed positive (high, 115.9 Au/mL) antinuclear antibody (ANA) and anti-ds-DNA (high, 435.4 IU/mL) tests, following which a diagnosis of SLE was made. Rheumatoid factor was negative. Quantitative analysis of urine for protein showed nephrotic-range proteinuria (3.5grams/24 hours). High resolution CT thorax of the patient showed bilateral ground glass changes (right more than left) with relative sparing of the subpleural regions suggestive of LP (see Figure 3).

Based on these findings and her failure to respond to empirical IV and oral antibiotics and anti-tuberculosis treatment, we made a revised diagnosis of SLE with LP. Anti-tuberculosis treatment was stopped and intravenous pulsed methylprednisolone (1,000 mg/day) for three days was given, followed by oral prednisolone 40mg/day and oral Azathioprine 50mg/day. There was gradual improvement of the symptoms with disappearance of clinical signs. There was also marked resolution of radiographic findings in the follow up chest X- rays and CT scan thorax at the follow up (see Figures 2 and 4). The patient was discharged on steroids and Azathioprine at maintenance doses and continues to do well on follow up at the outpatient department (OPD); she has been able to return to work.

DISCUSSION

This case report describes a severe case of LP in a patient with no pre-existing diagnosis of SLE in a setting with high TB endemicity and limited diagnostic resources and demonstrates the importance of thorough clinical assessment and re-evaluation of diagnosis when response to treatment is not as expected.

LP is relatively rare, but is a serious complication of SLE. It necessitates prompt and effective intervention to prevent its potentially lethal outcomes (13). The diagnosis of LP presents significant challenges due to its nonspecific clinical manifestations that often mimic other pulmonary disorders and the fact it is not a common manifestation during the primary presentation of SLE. It generally presents with acute onset of fever, cough, tachypnea and hypoxia. The usual radiological features of LP are consolidation in one or more areas, usually basal and bilateral, and often associated with pleural effusion and pulmonary arterial hypertension [1]. The underlying histology in cases of LP are those of diffuse alveolar damage, bronchiolitis obliterans organizing pneumonia, non-specific interstitial pneumonia, or a combination of these [4]. The mortality of lupus pneumonitis is around 50%, with respiratory failure being the primary cause of death [5]. The diagnosis of LP is primarily by exclusion of other causes of lung infiltration such as infective pneumonia (bacterial, mycobacterial, fungal and viral), organizing pneumonia, alveolar hemorrhage, pulmonary embolism among others.

In our case the primary suspicion was indeed of an infective origin, especially CAP and/or TB, on account of the chronic temporal pattern of the symptoms and signs with a positive history of contact with tuberculosis. However, when repeated sputum analysis ruled out a possible infective aetiology the index of suspicion shifted towards other possible causes and finally the diagnosis of LP was established by the clinical manifestations, HRCT findings, anti-nuclear antibody panel and dramatic response to corticosteroids.

Managing SLE by acute lupus pneumonitis in resource-limited countries is particularly challenging due to multiple clinical, logistical, and systemic factors. Diagnostic challenges, limited access to advanced imaging such HRCT is the gold standard to detect ALP, but many hospitals may only have X-ray capabilities, and early subtle lung changes may be missed leading to delayed diagnosis. ALP may mimic pneumonia or TB which are prevalent in resource-limited settings, and misdiagnosis often leads to inappropriate antibiotic therapy instead of immunosuppression. Timor Leste faces a high TB burden; WHO, in 2022 global TB report stated Democratic republic of Timor-Leste ranking 6th with incidence rate of 498 cases per 100,000 population, making it the highest in the SEA region.

Another important diagnostic challenge in resource-limited settings is limited laboratory testing; autoantibody panels (ANA, anti-dsDNA) and complement levels may not be readily available. Bronchoscopy and bronchoalveolar lavage for ruling out infection are often unavailable. Such as in our case, complement levels, bronchoscopy and bronchoalveolar lavage were not done because they are not available in our setting; tests for ANA and dsDNA are only available in the private setting, not in all government hospitals.

Meanwhile, limited or lack of availability of immunosuppressive drugs, high-dose IV corticosteroids and biologics/targeted therapies such as Belimumab and Rituximab in resource-limited settings make the management of SLE very challenging. Still another challenge is the decision to commence immunosuppression therapy in areas with high of TB or other endemic infection (e.g. strongyloides), along with the increased risk of opportunistic infection to the patient, with limited capacity for infection monitoring and prophylaxis, limitation of supportive care such as Intensive care unit (ICU) with oxygen therapy, monitoring and rapid response to respiratory failure as compare to high resource setting in developed countries (10,11,12,13,14)

When connective tissue disorders have a presentation which mimic an infective process the diagnostic challenges for the clinician become manifold. The possibilities of co-existence of a connective tissue disorder with an infection, or an infection which trigger an auto-immune response further complicate the diagnostic process. While on the one hand, patients with SLE can be complicated with superimposed infections, on the other hand, others may present with immune mediated processes which very closely resemble an infectious process, as was found in our case. Pulmonary infections in patients with SLE are also quite common. A previous study reported that pulmonary infections are seen in 20% of cases with SLE [6]. As discussed in our case, it may be difficult to differentiate pulmonary infection from LP, especially in countries like Timor Leste which are highly endemic for TB and there being limited resources for carrying out sophisticated immunological tests to diagnose or exclude connective tissue diseases. In our hospital there is not available test for dsDNA and ANA for connective tissue disorders (SLE), in cases where there is a strong suspicion, it is important to recommend the patient to be tested through a private clinic, such as in this case. In such cases, it sometimes becomes imperative to administer broad spectrum antibiotics and also to consider a trial of anti-tuberculous therapy prior to establishing a definitive diagnosis. However, other signs of active lupus like rash, arthralgias, malar rash, alopecia, elevated ESR and CRP with high urinary protein and casts may help in differentiating LP from pulmonary infections. The other possibilities that come in the differential diagnosis of lupus pneumonitis include are Goodpasture's syndrome and granulomatosis with polyangiitis (GPA), because all three share overlapping clinical, radiological and pathological features. In our case these entities could not be excluded as there was no availability of testing for ANCA and anti-GBM autoantibodies (8,9,10).

Because acute LP is a rare entity there is no definite consensus on the optimal modality of treatment [7]. Patients are generally treated with high dose corticosteroids. In cases which respond poorly to corticosteroids (methylprednisolone/prednisolone), immunosuppression (cyclophosphamide, azathioprine, methotrexate and mycophenolate mofetil (MMF) and biologics/ targeted therapy (Belimumab or Rituximab) are used. Hydroxychloroquine (HCQ) has also been used to some effect [11,12,13,14].

CONCLUSION

Connective tissue disorders have a wide range of presentation. Our case shows a rare presentation of SLE

presenting as acute LP which was initially considered as community acquired pneumonia and pulmonary tuberculosis. Even in tuberculosis endemic country like Timor-Leste where clinical findings and chest X-rays are sometimes taken as sufficient evidence for initiation of anti-tuberculous therapy in the absence of an alternative diagnosis, the differential diagnosis of SLE, especially LP should be borne in mind and appropriate investigations should be carried out so that the possibility of LP is not missed in these patients. In resource-limited settings, there are range of challenges in managing SLE and LP, including delays in diagnosis, lack of immunosuppressive drugs, high infection risk, and limited ICU support make ALP management extremely difficult, contributing to high morbidity and mortality as compared to high resource countries.

Conflict Of Interest Statement

None declared.

Ethics Statement

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or National institute of Public Health Timor-Leste (INSPTL). Informed consent was obtained from all individual participants included in the study. The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request from Radiology department and medical record hospital national Guido Valadares.

REFERENCES

1. Amirhossein Aarabi, Stella McGinn (2024), Severe lupus pneumonitis: A case of life-saving multimodal therapy with rituximab and corticosteroids. https://pmc.ncbi.nlm.nih.gov/articles/PMC11221880/2024 Jul 3;12(7): e01426. doi: 10.1002/rcr2.1426. Access 18/8/2025
2. Beresford MW, Cleary AG, Sills JA, Couriel J, Davidson JE (2005) Cardio-pulmonary involvement in juvenile systemic lupus erythematosus. Lupus 14: 152-158.
3. Ciftçi E, Yalçinkaya F, Ince E, Ekim M, Ileri M, et al. (2004) Pulmonary involvement in childhood-onset systemic lupus erythematosus: a report of five cases. Rheumatology (Oxford) 43: 587-591.
4. Fanouriakis A, et al. 2021 EULAR recommendation for the management of SLE. Ann. Rheum Dis. 2021; 80:1-9.
5. Hahn BH, et al. American College of Rheumatology guideline for screening, treatment, and management SLE. Arthrtis Rheum. 2012; 64:364-387.
6. Huang YC, Lin YT, Yang YH, Wang SJ, Yang CM, et al. (2001) Acute lupus pneumonitis mimicking pulmonary tuberculosis: a case report. J Microbiol Immunol Infect 34: 143-146.
7. Keane MP, Lynch JP 3rd (2000) Pleuropulmonary manifestations of systemic lupus erythematosus. Thorax 55: 159-166.
8. Lazovic B, Zlatkovic-Svenda M, Jasarovic D, Stevanovic D. Systemic lupus erythematosus presenting as acute lupus pneumonitis. Arch bronchoneumol. 2018; 54 (7): 377-379
9. Mathian A, Dorgham K, Parizot C, et al. Acute lupus pneumonitis: a rare but manifestation of systemic lupus erythematosus. Front Med (Lausanne). 2020; 7:610257.
10. Matthay RA, Schwarz MI, Petty TL, Stanford RE, Gupta RC, et al. (1975). Pulmonary manifestations of systemic lupus erythematosus: Review of twelve cases of acute lupus pneumonitis. Medicine (Baltimore) 54: 397–409.
11. Rai DK, Upadhyaya SK, Sharma A, Kuldeep K (2012) A case of lupus pneumonitis mimicking as infective pneumonia. IOSR Journal of Pharmacy 2: 11-14
12. Ramnathan P, Rathnasamy R (2011) Infections in a Rheumatology setting- Chennai experience. Indian J Rheumatol 6: 31-35.
13. Timor Leste Antimicrobial Guideline, Timor Leste, 2022
14. Tsokos GC. Systemic lupus erythematosus. N Engl J Med. 2011; 365:2110-2121.
15. Wiedemann HP, Matthay RA (1992) Pulmonary manifestations of systemic lupus erythematosus. J Thorac Imaging 7: 1-18.