RESEARCH PAPER

When Lupus Isn't to Blame: Disseminated Tuberculosis Masquerading as Disease Flare in Lupus Nephritis

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Abstract

Disseminated tuberculosis remains an important opportunistic infection among patients receiving prolonged immunosuppressive therapy for systemic autoimmune disorders. Distinguishing active infection from disease flare in systemic lupus erythematosus (SLE) is often difficult because both conditions may present with prolonged fever, constitutional symptoms, pulmonary infiltrates, and renal dysfunction. We report the case of a 40-year-old woman with biopsy-proven Class IV lupus nephritis, hypertension, and hypothyroidism, who presented with prolonged fever, dysuria, constitutional symptoms, and progressive respiratory distress. Radiological imaging demonstrated diffuse bilateral miliary pulmonary nodules with ground-glass opacities, while urine CBNAAT detected Mycobacterium tuberculosis. The coexistence of chronic kidney disease, persistent proteinuria, and long-term immunosuppressive therapy further complicated the clinical picture. Following multidisciplinary assessment, anti-tubercular therapy was initiated with supportive management. This case emphasizes the importance of maintaining a high index of suspicion for disseminated tuberculosis in patients with lupus nephritis presenting with prolonged fever, even when clinical manifestations overlap with lupus flare.

Keywords: Systemic lupus erythematosus, Lupus nephritis, Disseminated tuberculosis, Genitourinary tuberculosis, Immunosuppression, Persistent fever

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Introduction

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease characterized by recurrent periods of disease activity requiring prolonged immunosuppressive therapy. Although these medications improve long-term survival, they substantially increase susceptibility to opportunistic infections, particularly tuberculosis in endemic regions. Tuberculosis remains one of the leading infectious complications among patients with SLE. Infection may involve pulmonary, genitourinary, lymphatic, or disseminated forms and often mimics lupus flare because both conditions present with fever, fatigue, weight loss, elevated inflammatory markers, and multiorgan involvement. Consequently, establishing the diagnosis may be delayed, potentially increasing morbidity and mortality.

We report a patient with established lupus nephritis who developed prolonged fever and constitutional symptoms secondary to probable disseminated tuberculosis. The case highlights the diagnostic challenges encountered in differentiating infection from autoimmune disease activity in immunocompromised individuals.

Case Report

A 40-year-old woman presented with persistent fever for approximately three months, associated with chills and rigors. She also complained of dysuria, obstructive lower urinary tract symptoms, progressive loss of appetite, significant weight loss, nausea, intermittent cough, and increasing shortness of breath.

She had been diagnosed with systemic lupus erythematosus in 2019 and had biopsy-confirmed Class IV lupus nephritis. Her medical history was also significant for systemic hypertension and hypothyroidism. She had been receiving prednisolone, hydroxychloroquine, mycophenolate mofetil, antihypertensive medications, and thyroid hormone replacement.

On admission, she was conscious, alert, and oriented. Oxygen saturation was reduced, necessitating supplemental oxygen, and she subsequently required non-invasive ventilatory support with BiPAP because of worsening hypoxemia. Cardiovascular and abdominal examinations were otherwise unremarkable.

Laboratory Investigations

Baseline investigations demonstrated impaired renal function with serum creatinine of approximately 2.2 mg/dL and elevated blood urea levels. Urinalysis revealed persistent proteinuria, albumin positivity, and pyuria, raising suspicion of urinary tract involvement. These findings were interpreted in the context of known lupus nephritis with chronic kidney disease.

High-resolution computed tomography (HRCT) of the thorax demonstrated diffuse bilateral miliary nodules associated with patchy ground-glass opacities, findings highly suggestive of disseminated pulmonary tuberculosis. Urine culture showed sterile pyuria. Urine cartridge-based nucleic acid amplification test (CBNAAT) subsequently detected Mycobacterium

tuberculosis with a low bacterial load, supporting the diagnosis of genitourinary tuberculosis.

Differential Diagnosis

The major differential diagnoses included:

- Disseminated tuberculosis with pulmonary and genitourinary involvement
- Lupus flare with active lupus nephritis
- Pyelonephritis or complicated urinary tract infection
- Chronic kidney disease with persistent proteinuria
- Opportunistic pulmonary infection secondary to immunosuppression

Management

The patient initially received broad-spectrum antimicrobial therapy with intravenous meropenem along with supportive treatment including oxygen supplementation and non-invasive ventilation. Multidisciplinary evaluation was undertaken with consultations from nephrology, pulmonology, rheumatology, and surgical teams. Following microbiological confirmation of tuberculosis, anti-tubercular therapy was initiated while continuing close monitoring of renal function and lupus disease activity. A repeat renal biopsy was planned for further assessment of persistent renal dysfunction.

Discussion

Tuberculosis remains a major cause of morbidity among patients with SLE, particularly in countries where the disease is endemic. Long-term corticosteroid therapy and immunosuppressive agents impair cell-mediated immunity, increasing susceptibility to both primary infection and reactivation of latent tuberculosis.

The present patient had multiple risk factors for opportunistic infection, including lupus nephritis, chronic kidney disease, corticosteroid therapy, and mycophenolate mofetil. These factors likely contributed to the development of disseminated tuberculosis involving both the lungs and urinary tract.

One of the greatest diagnostic challenges was distinguishing active infection from lupus flare. Fever, constitutional symptoms, renal dysfunction, elevated inflammatory markers, and pulmonary infiltrates may occur in either condition. In such situations, relying solely on clinical manifestations can delay definitive diagnosis.

High-resolution CT played a crucial role by demonstrating diffuse bilateral miliary nodules with associated ground-glass opacities, findings strongly suggestive of hematogenous dissemination of Mycobacterium tuberculosis. Furthermore, urine CBNAAT provided rapid microbiological confirmation of genitourinary involvement, allowing timely initiation of anti-tubercular therapy.

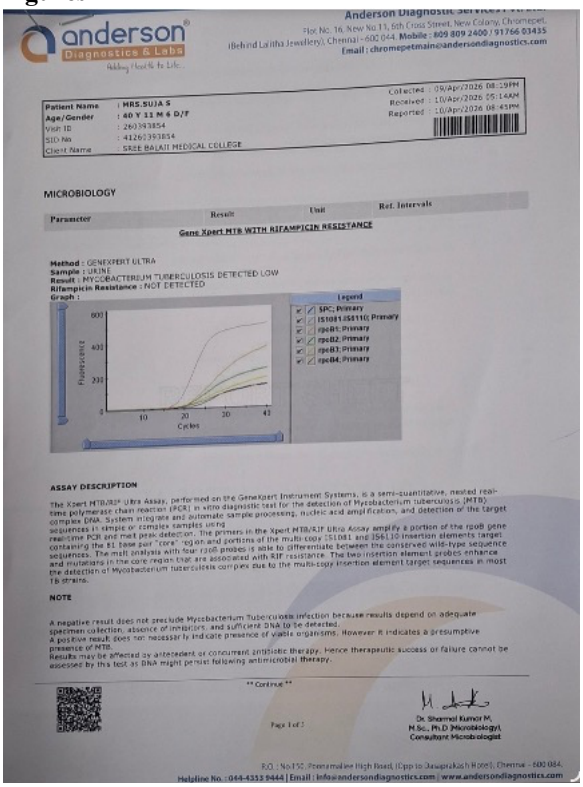
The coexistence of persistent proteinuria complicated therapeutic decision-making. Although worsening renal function may indicate lupus nephritis progression, infectious causes must be excluded before escalation of immunosuppressive therapy. Multidisciplinary collaboration among internists, nephrologists, pulmonologists, and rheumatologists was therefore essential in optimizing patient management.

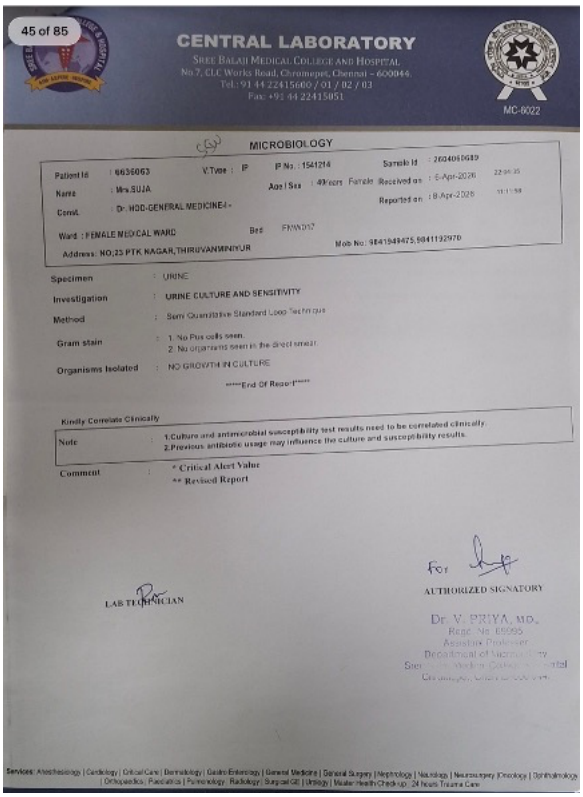
Early recognition of tuberculosis in immunocompromised individuals is particularly important because delayed diagnosis may lead to respiratory failure, progressive renal dysfunction, disseminated disease, and increased mortality. Careful clinical assessment combined with microbiological confirmation and radiological evaluation remains the cornerstone of diagnosis.

Conclusion

This case illustrates the diagnostic complexity of prolonged fever in patients with systemic lupus erythematosus receiving immunosuppressive therapy. Disseminated tuberculosis should always be considered in the differential diagnosis of persistent constitutional symptoms, pulmonary infiltrates, and renal dysfunction in endemic regions. Prompt microbiological confirmation, appropriate imaging, and coordinated multidisciplinary management are essential to improve clinical outcomes while avoiding unnecessary escalation of immunosuppressive therapy.

Figures





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CENTRAL LABORATORY

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MC-6022

MICROBIOLOGY

Patient Id	: 6636063	V.Type : IP	IP No. : 1541214
Name	: Mrs.SUJA	Age / Sex	: 40Years Female
Consl.	: Dr. HOD-GENERAL MEDICINE4 -		
Ward	: FEMALE MEDICAL WARD	Bed	: FNW057
Address	: NO;23 PTK NAGAR,THIRUVANMIYUR		

Sample Id : 2604060489
Received on : 6-Apr-2026
Reported on : 8-Apr-2026
Mob No. : 9841949475,9841192970

Specimen	:	URINE
Investigation	:	URINE CULTURE AND SENSITIVITY
Method	:	Semi Quantitative Standard Loop Technique
Gram stain	:	1. No Pus cells seen. 2. No organisms seen in the direct smear.
Organisms Isolated	:	NO GROWTH IN CULTURE

*****End Of Report*****

Kindly Correlate Clinically

Note : 1.Culture and antimicrobial susceptibility test results need to be correlated clinically.
2.Previous antibiotic usage may influence the culture and susceptibility results.

Comment : * Critical Alert Value
** Revised Report

LAB TECHNICIAN

For

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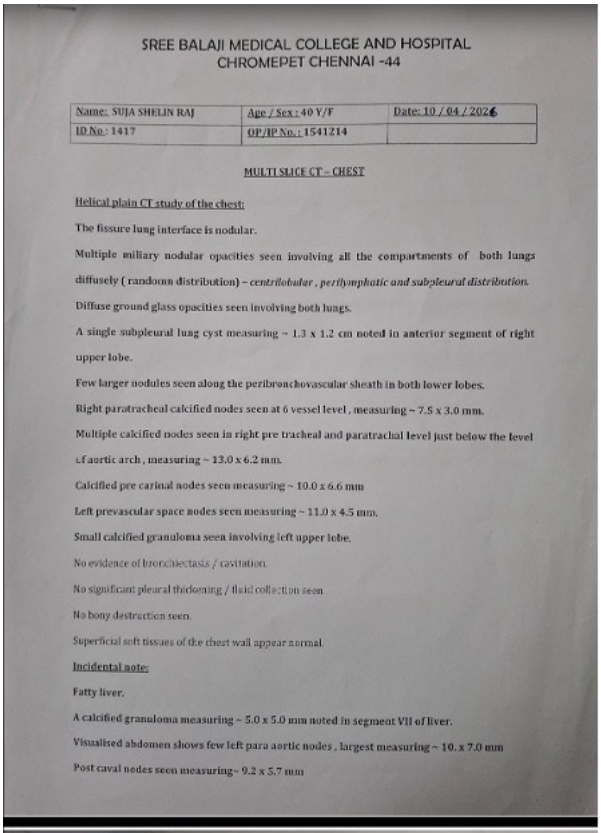


SREE BALAJI MEDICAL COLLEGE AND HOSPITAL
CHROMEPET CHENNAI -44

Name: SUJA SHELIN RAJ	Age / Sex : 40 Y/F	Date: 10 / 04 / 2026
ID No : 1417	OP/IP No. : 1541214	

MULTI SLICE CT – CHEST

Helical plain CT study of the chest:

The fissure lung interface is nodular.

Multiple miliary nodular opacities seen involving all the compartments of both lungs diffusely (randomn distribution) – *centrilobular , perilymphatic and subpleural distribution.*

Diffuse ground glass opacities seen involving both lungs.

A single subpleural lung cyst measuring ~ 1.3 x 1.2 cm noted in anterior segment of right upper lobe.

Few larger nodules seen along the peribronchovascular sheath in both lower lobes.

Right paratracheal calcified nodes seen at 6 vessel level , measuring ~ 7.5 x 3.0 mm.

Multiple calcified nodes seen in right pre tracheal and paratrachal level just below the level of aortic arch , measuring ~ 13.0 x 6.2 mm.

Calcified pre carinal nodes seen measuring ~ 10.0 x 6.6 mm

Left prevascular space nodes seen measuring ~ 11.0 x 4.5 mm.

Small calcified granuloma seen involving left upper lobe.

No evidence of bronchiectasis / cavitation.

No significant pleural thickening / fluid collection seen.

No bony destruction seen.

Superficial soft tissues of the chest wall appear normal.

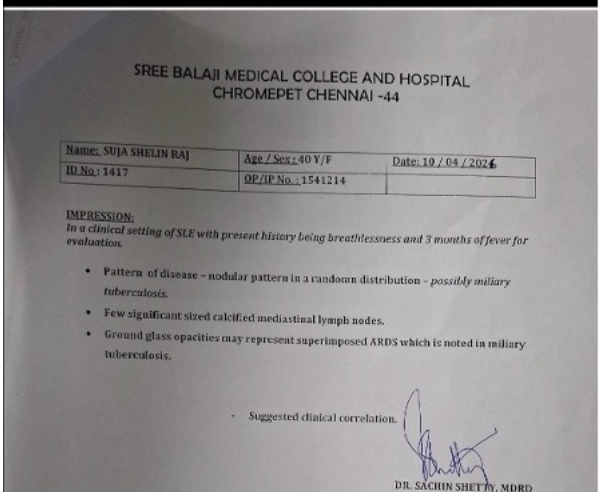
Incidental note:

Fatty liver.

A calcified granuloma measuring ~ 5.0 x 5.0 mm noted in segment VII of liver.

Visualised abdomen shows few left para aortic nodes , largest measuring ~ 10. x 7.0 mm

Post caval nodes seen measuring ~ 9.2 x 5.7 mm



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IMPRESSION:
In a clinical setting of SLE with present history being breathlessness and 3 months of fever for evaluation.

- Pattern of disease – nodular pattern in a randomn distribution - *possibly miliary tuberculosis.*
- Few significant sized calcified mediastinal lymph nodes.
- Ground glass opacities may represent superimposed ARDS which is noted in miliary tuberculosis.

- Suggested clinical correlation.

DR. SACHIN SHETTY, MDRD