JoTH Journal of Tropical Health

Case report

An Unusual Presentation of Systemic Lupus Erythematosus in a 70-Year-Old Female: A Case Report

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Abstract

Systemic lupus erythematosus (SLE) is uncommon in the elderly and often presents with subtle or atypical features that mimic malignancy, chronic infection, or other autoimmune disorders. We report a 70-year-old previously healthy woman with six months history of constitutional symptoms followed by photosensitive macular and malar rashes, generalized non-tender lymphadenopathy, and pancytopenia. Extensive infectious and malignant workup was negative. Autoimmune evaluation revealed ANA positivity (1:320, homogeneous), low complement levels, and positive anti-Ro and anti-Smith antibodies, confirming late-onset SLE despite negative anti-dsDNA. Her skin biopsy was also highly suggestive of subacute cutaneous lupus erythematosus. Treatment with prednisolone and hydroxychloroquine led to clinical and hematologic improvement with imaging evidence of marked resolution of lymphadenopathy. This case illustrates the diagnostic complexity of late-onset SLE and highlights the importance of considering SLE in elderly patients presenting with unexplained constitutional symptoms, lymphadenopathy, cutaneous manifestations and cytopenia.

Keywords: Systemic lupus erythematosus, elderly, anti-Ro antibodies, anti-Smith antibodies, photosensitive rash, case report

Funding: None
Competing interests: None
Received: 20 December 2025 **Accepted:** 10 May 2026 **Published:** 26 May 2026

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Cite this article as: Abeyrathna D et al. An Unusual presentation of systemic lupus erythematosus in a 70-year-old female: A case report. Journal of Tropical Health 2026;2 (1):24-29. DOI: https://doi.org/10.4038/joth.v2i1.48

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that predominantly affects women of childbearing age, with peak incidence between 20 and 40 years [1]. A subset of patients presents with late-onset SLE, defined as disease onset after age 50, accounting for approximately 12–18% of all SLE cases. Ageing is associated with morphological and functional changes in the immune system that may alter immune regulation and autoantibody production, leading to differences in disease expression compared with younger patients [1].

Late-onset SLE typically has an insidious and less aggressive clinical course. Initial symptoms are often nonspecific, such as arthralgia, myalgia, fatigue, weight loss, and low-grade fever, which can delay diagnosis [1,2]. Compared with early-onset disease, cutaneous manifestations, photosensitivity, renal involvement, neurological features, and arthritis are less common. In contrast, serositis, pulmonary involvement, and coexisting Sjögren's syndrome are observed more frequently in older patients [1,3].

Diagnosis in elderly individuals requires careful evaluation, as comorbidities and drug-induced lupus erythematosus (DIL) may mimic SLE [4]. Common drugs implicated in DIL include hydralazine,

procainamide, isoniazid, methyldopa, carbamazepine, acebutolol, and biologic agents such as interferon-alpha and TNF-alpha inhibitors [5]. Therefore, a thorough drug history is essential in all suspected cases.

Immunologically, late-onset SLE is characterised by a higher prevalence of rheumatoid factor (RF) and anti-Ro (SSA) and anti-La (SSB) antibody positivity [1]. Although anti-dsDNA antibodies may be present, their titers do not correlate strongly with organ damage or disease severity in this subgroup [4].

Overall, late-onset SLE tends to follow a more benign course, but prognosis is often influenced by comorbid conditions rather than lupus activity itself. Cardiovascular disease, malignancy, drug-related complications, and infections are the leading causes of morbidity and mortality in this population [4].

We present a case of a 70-year-old woman who presented with constitutional symptoms, photosensitive skin lesions, generalised lymphadenopathy, and pancytopenia, ultimately diagnosed with SLE. The significance of this case is in pointing out an uncommon presentation of SLE in an elderly patient and maintaining a high index of suspicion for autoimmune diseases, which can be easily missed in this population.

Case Presentation

A 70-year-old previously healthy woman presented with a six-month history of intermittent low-grade fever associated with chills, anorexia, and significant weight loss. Two months before hospital admission, she developed a generalised erythematous, non-pruritic, photosensitive macular rash predominantly affecting the anterior chest, lateral upper limbs, and fingertips. A classic malar rash appeared two weeks before admission.

She also experienced symmetrical, non-inflammatory small joint arthralgia for one month, which resolved spontaneously before presentation. She denied any large joint involvement or back pain and no history of oral ulcers, hair loss, dry eyes or mouth or Raynaud's phenomenon. There were no respiratory, gastrointestinal, or neurological complaints, nor hematuria or frothy urine. She had no past medical history of chronic illness, was not on long-term medications, and had no family history of autoimmune disorders or carcinoma. She had no history of pregnancy losses or thrombotic events.

On examination, she was mildly pale. She had a typical malar rash and widespread erythematous photosensitive rash mainly distributed in sun-exposed areas, including the upper limbs and upper chest. There was no alopecia, mucosal ulceration, arthritis or joint deformities. There was palpable, non-tender, generalised lymphadenopathy involving cervical, axillary, and inguinal regions. No hepatosplenomegaly. Her cardiovascular, respiratory, and neurological examinations were unremarkable.

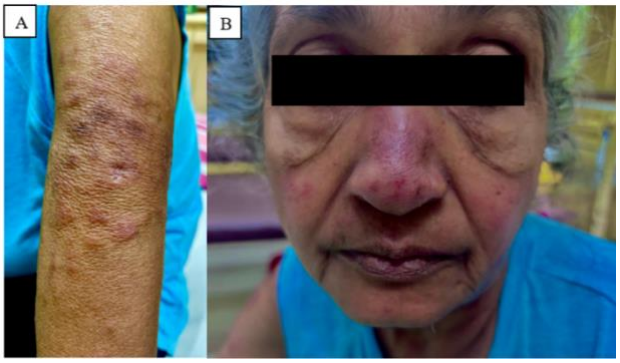

Figure 1A: Photosensitive rash on the lateral aspect of the left arm, **1B:** Malar rash

Diagnostic investigations and findings

The results of the laboratory investigations are summarised in Table 1. Her full blood count revealed pancytopenia, and the blood picture showed evidence of excessive rouleaux formation without any abnormal or immature cells. Her liver function tests, clotting profile and serum electrolytes were normal, and no evidence for renal impairment or significant tubular or glomerular pathology was noted. Positive antinuclear antibody (ANA) testing and reduced complement levels were identified. Histology of skin biopsy from her left arm was highly suggestive of subacute cutaneous lupus erythematosus. Hence, based on the clinical history and laboratory investigations, the patient fulfilled the diagnostic criteria for SLE. Contrast-enhanced computed tomography (CECT) of the chest, abdomen, and pelvis revealed generalised lymphadenopathy without evidence of any solid organ malignancy. Haematological malignancy was excluded by bone marrow examination and lymph node biopsy, both of which demonstrated only reactive changes. Other potential infectious causes of lymphadenopathy, including tuberculosis, were also excluded.

In view of these findings, a final diagnosis of SLE with haematological and cutaneous involvement was established. The patient was initiated on oral prednisolone at a dose of 1 mg/kg/day along with oral hydroxychloroquine. Following initiation of therapy, there was gradual improvement in her constitutional symptoms, photosensitive rashes and pancytopenia. Follow-up CECT of the chest, abdomen, and pelvis after 6 months of treatment showed marked resolution of lymphadenopathy.

Table 1: Laboratory investigations

Category	Investigation	Result	Reference range with units
Hematological investigations and clotting profile	White blood cells	**2610**	4000-10000
	Neutrophil	41	40-80%
	Lymphocytes	43.7	20-40%
	Eosinophils	3.4	1.0-6.0%
	Hemoglobin	**9.7**	11-17 g/dl
	Hematocrit	29.8	36- 50 %
	Mean corpuscular volume	91.7	82-96 fl
	Platelet	**64000**	150000-400000
	Reticulocyte count	2%	0.5 – 2.5%
	PT	17.8	11-13.5 seconds
	INR	0.9	<1.1
	APTT	31	25-35 seconds
	Blood picture	Pancytopenia, excessive rouleaux formation	
Inflammatory markers	C-reactive protein	7	0-5 mg/dl
	ESR	**123**	
	Procalcitonin	0.203	< 0.5 ng/ml
Liver and renal function tests	Aspartate aminotransferase	28	<40 U/L
	Alanine aminotransferase	29	<40 U/L
	Albumin	38	35-50 g/L
	Globulin	22	20-35 g/L
	Total bilirubin	0.78	0.1-1.2 mg/dl
	Direct bilirubin		
	Alkaline phosphatase	175	64-306 U/L
	Gamma glutamyl transferase	40.9	9-39 U/L
	Serum creatinine	62	70 – 120 µ mol / L
	Urine albumin/creatinine	24.58	<30 mg Albumin/ Creatinine
Infectious workup	Ebstein Barr virus IgM	Non-reactive	
	Ebstein Barr virus IgG	Reactive	
	Cytomegalovirus IgM	Non-reactive	
	Cytomegalovirus IgG	Reactive	
	HIV 1 and 2 Ag/ Ab combination assay	Negative	
	Hepatitis Bs antigen	Negative	
	Hepatitis C IgM	Negative	
	Hepatitis C IgG	Negative	
	Parvo virus B19 DNA	Negative	
	IGRA	Negative	
Antibody panel	**Anti-nuclear antibodies**	**Positive with titer of 1:320 Pattern: Homogenous**	
	Anti cytoplasmic antibody	Negative	
	Anti mitotic antibody	Negative	
	Anti dsDNA	Negative	
	Jo-1 antibody	Negative	
	RNP antibody	Negative	
	Scl-70 antibody	Negative	
	Ro (SSA) antibody	**Positive**	
	La (SSB) antibody	Negative	
	Anti Smith antibody	**Positive**	
	Complement C3	**37**	83-177 mg/dl
	Complement C4	**7**	12-36 mg/dl
Invasive investigations	Bone marrow aspirate	Moderately hypocellular bone marrow with no detectable myelodysplastic features. Marrow hypocellularity could be a part of autoimmune nature of a disease	
	Immunohistochemistry of bone marrow aspirate	No evidence of lymphoproliferative disorder	

	Real time PCR of *Mycobacterium tuberculosis* of bone marrow aspirate	Negative	
	Mycobacterial tuberculosis culture of bone marrow aspirate	Negative	
	Skin biopsy from left arm	Features are highly suggestive of Subacute cutaneous lupus erythematosus	
	Right axillary lymph node biopsy and immunohistochemistry	Reactive lymph node	
Imaging	2D Echocardiogram	Normal ejection fraction, No structural abnormalities or valvular lesions	
	CECT chest, abdomen and pelvis, at diagnosis	Pathologically enlarged lymph nodes in the cervical, axillary, subpectoral, mediastinal, hila, abdominal, pelvic and inguinal groups. No hepatosplenomegaly or destructive osseous lesions. Overall appearances suggestive of lymphoma; suggest lymph node biopsy for definitive diagnosis	
	CECT Chest, abdomen and pelvis, six months after treatment	Prominent bilateral axillary and external iliac lymph nodes are nonspecific. No suspicious mass or lymphadenopathy	
Other	Serum protein electrophoresis	Polyclonal gammopathy	
	Serum cryoglobulins	Negative	
	Serum IgG4 level	0.69 g/L	0.03-2.0 g/L
	Angiotensin converting enzyme	50 U/L	8-52 U/L
	Rheumatoid factor	8 IU/ml	<20 IU/ml
	HbA1C	5.5%	<5.6%

Discussion

SLE predominantly affects women of reproductive age; however, a distinct subset of patients develops the disease later in life [1]. Late-onset SLE often presents with atypical clinical and immunological features that delay diagnosis [1]. This case illustrates the diagnostic complexity of late-onset SLE in a 70-year-old woman whose presentation closely mimicked malignancy or chronic infection.

This patient presented with prolonged constitutional symptoms, including low-grade fever, anorexia, and significant weight loss, followed by photosensitive cutaneous manifestations, generalised lymphadenopathy, and pancytopenia. Such nonspecific symptoms are well described in elderly-onset SLE and frequently lead clinicians to prioritise infectious or malignant etiologies at this age [1]. In this case, the presence of generalised, non-tender lymphadenopathy and cytopenia prompted extensive evaluation for lymphoproliferative disorders, tuberculosis, and viral infections, all of which were systematically excluded. Lymph node histology demonstrated reactive changes, and bone marrow examination revealed hypocellularity without dysplasia or malignant infiltration, supporting an autoimmune rather than neoplastic process.

Haematological abnormalities are common in SLE and may include anaemia, leukopenia, thrombocytopenia, or pancytopenia [2,6]. In late-onset disease, cytopenia may be particularly prominent and multifactorial, resulting from immune-mediated peripheral destruction, bone marrow suppression, or chronic inflammation [1]. In our patient, bone marrow hypocellularity in the absence of myelodysplasia or infiltration, combined with low complement levels and polyclonal hypergammaglobulinemia, favoured immune-mediated marrow involvement. The subsequent improvement in blood counts following immunosuppressive therapy further supports this mechanism.

Cutaneous manifestations in late-onset SLE are reported less frequently than in younger patients; however, photosensitivity and malar rash remain important diagnostic clues [1]. Several age-related changes in the immune system, including reduced cutaneous immune responsiveness due to decreased keratinocyte cytokine production and decreased density and function of T and B cells, may contribute to a lower prevalence of cutaneous manifestations in elderly SLE [7,8]. Our patient developed a classical malar rash and photosensitive macular lesions, with skin biopsy demonstrating changes of cutaneous lupus

erythematosus, which is an atypical presentation in this age group.

Immunologically, this patient demonstrated a characteristic late-onset SLE profile, with high-titer antinuclear antibodies, low complement levels, and positivity for anti-Ro (SSA) and anti-Smith antibodies, despite negative anti–double-stranded DNA antibodies [1,2]. Previous studies have shown that anti-dsDNA antibodies are less consistently present in late-onset SLE and may not correlate strongly with disease activity or organ involvement in this population [2]. In contrast, anti-Ro antibodies are more prevalent and may be associated with cutaneous manifestations and cytopenia [9]. Anti-Smith antibodies, although highly specific for SLE, are less frequently reported in late-onset disease, making their presence in this patient particularly notable and supportive of a definitive diagnosis [1,3].

Drug-induced lupus erythematosus is an important differential diagnosis in elderly patients; however, this was considered unlikely in our case, as the patient had no history of exposure to medications commonly implicated in drug-induced lupus, such as hydralazine, procainamide, isoniazid, or biologic agents [3,4,5]. Additionally, the presence of hypocomplementemia and anti-Smith antibody positivity favours idiopathic SLE over a drug-induced aetiology [3].

Generalised lymphadenopathy is an uncommon but recognised manifestation of active SLE and is thought to result from immune complex deposition and reactive lymphoid hyperplasia [2,10,11]. In older patients, this finding often raises concern for alternative diagnoses, including lymphoma, other malignancies, or chronic infections. In the present case, lymph node biopsy revealed only reactive follicular hyperplasia without evidence of malignancy or specific infectious processes, thereby excluding these conditions. Additionally, serum IgG4 and angiotensin-converting enzyme (ACE) levels were within normal limits, effectively ruling out IgG4-related disease and sarcoidosis as causes of the lymphadenopathy. The marked regression of lymphadenopathy following initiation of immunosuppressive therapy for SLE provides strong supportive evidence for its attribution to lupus activity.

Despite the traditionally perceived milder disease course of late-onset SLE, prognosis is often influenced by comorbidities, infections, and treatment-related complications rather than lupus activity itself [3]. Notably, our patient had no significant comorbid conditions and responded favourably to systemic corticosteroids and hydroxychloroquine, with resolution of constitutional symptoms, improvement in pancytopenia, and regression of lymphadenopathy. This highlights that timely recognition and appropriate therapy can result in excellent outcomes even in elderly patients.

Conclusion

This case highlights the importance of considering SLE in the differential diagnosis of elderly patients presenting with unexplained constitutional symptoms, cytopenia, lymphadenopathy, and cutaneous manifestations. Awareness of the atypical presentations and distinct immunological profiles of late-onset SLE is essential to avoid diagnostic and treatment delay, which remain key to improving outcomes in this challenging patient population.

Patient perspective

"I have visited so many hospitals over a long time, going through numerous tests, but no one could identify the cause of my condition. I often felt frustrated and worried about my health. When I came to the Prof. Unit at Anuradhapura Hospital, the ward team took the time to understand my situation, care for me attentively, and manage my condition effectively. Since then, my symptoms have improved significantly, and I feel much better both physically and emotionally. I am truly grateful to the entire ward team for their kindness, dedication, and support. They have made a real difference in my life."

Consent

Informed written consent was obtained from the patient for publication of her clinical data and related images.

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