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Case Report

A wolf in psoriatic skin: systemic lupus erythematosus masquerading as psoriasis

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disorder characterized by diverse clinical manifestations that may mimic several other diseases, posing significant diagnostic challenges. We report the case of a 55-year-old male who had been treated for presumed psoriasis for nearly 20 years and who presented to us with progressive symmetrical proximal muscle weakness involving all four limbs and truncal muscles and was associated with sensory disturbances. Clinical examination revealed depigmented and hyperpigmented plaques, scarring alopecia, and multiple oral ulcers. Nerve conduction studies revealed severe sensorimotor axonal polyneuropathy with secondary demyelination. Muscle biopsy demonstrated features of immune-mediated necrotizing inflammatory myopathy, while skin biopsy showed histopathological features consistent with discoid lupus erythematosus. These findings supported a diagnosis of SLE with muscular and neurological involvement. This case highlights the importance of reconsidering treatment-resistant psoriasiform skin lesions, particularly when accompanied by new systemic manifestations. Cutaneous manifestations of SLE can resemble chronic plaque psoriasis, potentially resulting in misdiagnosis and delayed recognition of the underlying autoimmune condition. The coexistence of inflammatory myopathy further underscores the heterogeneous and potentially severe nature of lupus-associated overlap syndromes.

Keywords: Systemic lupus erythematosus, Male, Psoriasis, Myopathy

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic multisystem, autoimmune disorder characterized by a broad spectrum of clinical manifestations.[1] The cutaneous manifestations of SLE are varied and may mimic a variety of dermatological conditions.[2] Psoriasiform lupus is one such condition, in which lesions resemble chronic plaque psoriasis, which often leads to misdiagnosis and delay in treatment.[3,4] SLE commonly affects women. The disease is relatively rare in males and when present is often associated with a more severe clinical presentation and delay in diagnosis.[5]

Here, we present a case of a middle-aged male who was initially diagnosed to have Psoriasis and was on treatment for nearly 20 years before a diagnosis of SLE was made.

CASE REPORT

A 55 year old male patient was admitted to the Department of General Medicine, Vydehi Institute of Medical Sciences, Bangalore, with complaints of chronic progressive, painless, symmetrical proximal muscle weakness of bilateral lower limbs since 3 months. Initially he had difficulty in climbing stairs, getting up from a squatting position and from a chair which later progressed such that he needed two people to help him stand and walk. He developed similar weakness in the proximal muscles of bilateral upper limbs in the form of difficulty in combing hair, washing hair and bringing food to mouth after one month. However, the distal motor power of all four limbs was normal. The patient also complained of sensory disturbance in both lower limbs in the form of reduced sensation to touch and temperature from 3 months.

He also complained of patchy alopecia and depigmented skin lesions over the scalp and multiple oral ulcers since 20 years which had progressed since 4 months. There was no history suggestive of cranial nerve involvement or bowel and bladder involvement.

The patient had a significant past history of having been diagnosed with Psoriasis and was on treatment since 20 years. The patient was prescribed topical creams for application, exact details of which were unavailable with the patient at the time of admission. However, the patient had no improvement in skin lesions.

There was no significant personal or family history.

On examination, the patient was moderately built and nourished and was pale with grade 3 pandigital clubbing (congenital) and bilateral pitting edema of the lower limbs. Dermatological examination showed multiple depigmented plaques with peripheral hyperpigmentation, crusted plaques and scarring alopecia.

Oral examination also showed multiple ulcers over hard palate and buccal mucosa and hyperpigmentation of the buccal mucosa with depigmentation of lips.

Neurological examination revealed hypotonia of all four limbs with normal neck movements. There was symmetrical proximal muscle weakness predominantly in the lower limbs. Shoulder power was grade 4/5 bilaterally, elbow flexion and extension was grade 4/5 while wrist movements and hand grip were normal. Truncal muscle weakness was also present as the patient was unable to roll from one side to the other side in bed or sit up without help. Lower limb examination showed weakness of hip flexion (grade 2/5), hip extension, abduction and adduction were grade 3/5, knee flexion and extension was grade 3/5.Ankle movements were relatively preserved (grade 4/5) and toe movements were normal. Deep tendon reflexes were absent in biceps, knee and ankle jerk bilaterally while triceps and supinator reflexes were decreased (1+). Superficial reflexes examination showed absent abdominal reflexes, mute plantar responses, and normal corneal and conjunctival reflexes. Sensory examination showed decreased fine touch over the dorsum of both feet and ankles with impaired position sensation at toe and ankles. Crude touch, pain, temperature and vibration sense were normal bilaterally. Thus, the patient had symmetrical proximal predominant quadriparesis with truncal weakness, grossly decreased reflexes, and distal large fibre sensory neuropathy.

ECG showed normal sinus rhythm with poor progression of R wave in V1-V5, QS in V4, T wave inversions in V2-V6 and aVL (no dynamic ST-T changes on serial ECG). 2D Echo showed mild concentric LVH, no PAH, and EF of 55%. Ultrasonography of abdomen showed bilateral mild increased cortical echotexture of kidneys. The chest X ray was normal.

Nerve conduction studies showed severe symmetrical sensorimotor axonal polyneuropathy with secondary demyelination of all four limbs, more predominant in the lower limbs. Electromyography confirmed chronic neurogenic reinnervation changes.

Muscle biopsy was done from the vastus lateralis which showed features of necrotizing inflammatory myopathy, characterized by pipestem capillaries and mild endomysial, interstitial, and perivascular inflammatory infiltrates without fibrosis, which is consistent with immune-mediated necrotizing inflammatory myopathy.

Sural nerve biopsy was also done, which showed no histopathological evidence of vasculitis.

Skin biopsy was done from the right forearm, which showed a thinned epidermis, basal vacuolar degeneration, thickened basement membrane, mild to moderate superficial perivascular inflammation composed of lymphocytes, histiocytes, plasma cells, and a few neutrophils, along with periadnexal inflammatory infiltrates, which were suggestive of discoid lupus erythematosus.

In summary, a middle-aged male, who was a diagnosed case of Psoriasis for 20 years presented with progressive proximal muscle weakness, initially involving the lower limbs and later progressed to involve the upper limbs and trunk muscles. Investigations revealed positive Anti-Smith and U1-SNRNP antibody. Nerve conduction studies showed severe symmetrical sensorimotor axonal polyneuropathy with secondary demyelination of all four limbs, more predominant in the lower limbs. Muscle biopsy showed immune-mediated necrotizing inflammatory myopathy. These above findings, along with the history of non-resolving Psoriasis, prompted us to perform a skin biopsy which showed features of DLE. Hence a Diagnosis of SLE with inflammatory myopathy was made. In view of significant proteinuria, the patient was advised to undergo renal biopsy to look for Lupus Nephritis; however, the patient was unwilling. The patient

was initiated on Tab. Prednisolone 60MG 1-0-0 in tapering doses, Tab. Hydroxychloroquine 200MG 1-0-1 and Tab. Sulphasalazine 500 mg 1-0-1 and other supportive measures (TAB. Calcium 500MG + Vitamin D3 2000IU 0-1-0, Clobetasol + Salicylic 3% lotion 0-0-1 and Tacrolimus ointment 0.1% 1-0-1). At the time of discharge, the power of upper limb improved to shoulder abduction and adduction 5/5, shoulder flexion, extension, internal rotation and external rotation to 4+/5, elbow flexion and extension to 5/5 and wrist flexion and extension to 5/5. However, lower limb power did not show any improvement. He was discharged after 20 days with advice of strict adherence to medications and regular follow up. Patient was followed up after 1 month and he reported consistent improvement in neurological symptoms. However, no improvement in skin lesions was noted. Patient had not yet undergone renal biopsy. Importance of renal biopsy was reiterated.



Figure 1: Depigmented patches with peripheral hyperpigmentation and scarring alopecia.

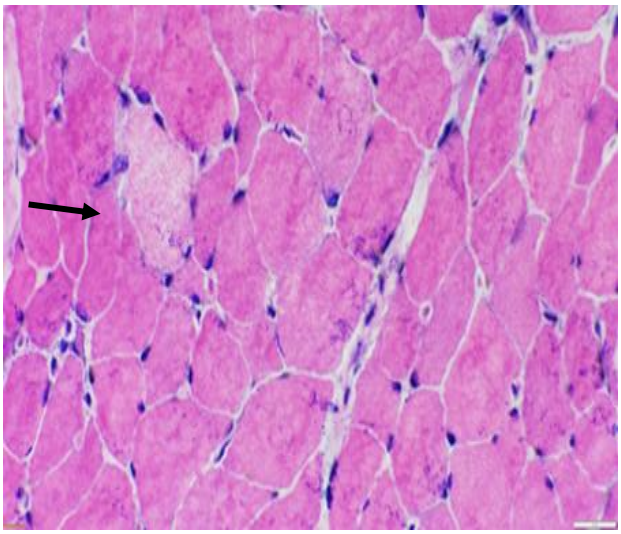


Figure 2: Muscle biopsy showing necrotic fibre (arrow) seen.

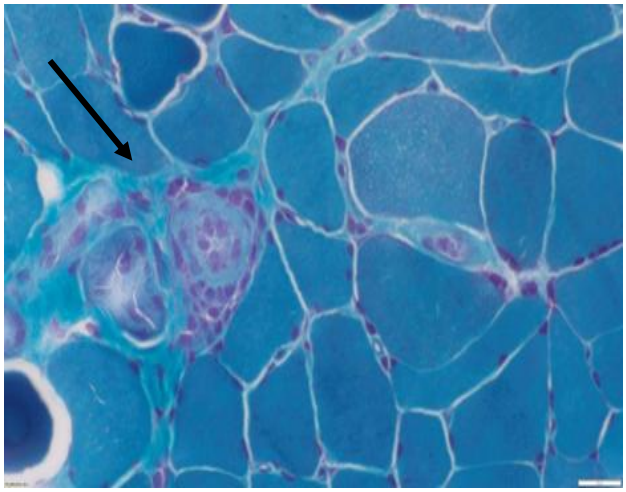


Figure 3: Muscle biopsy showing focus of mild lymphocytic endomysial perivascular inflammation (arrow).

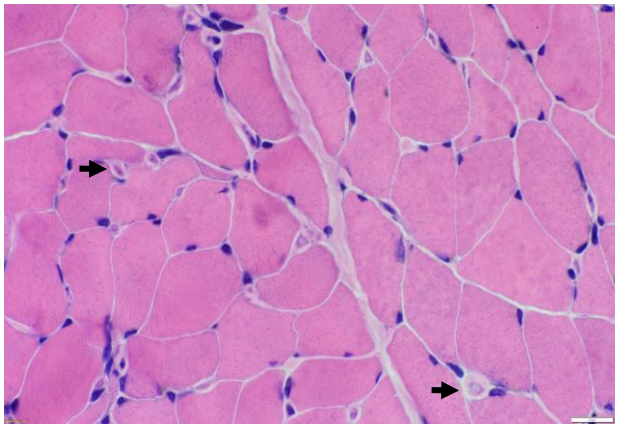


Figure 4: Endomysial capillaries show prominent hyalinization resembling pipestem capillaries (arrow) (muscle biopsy).

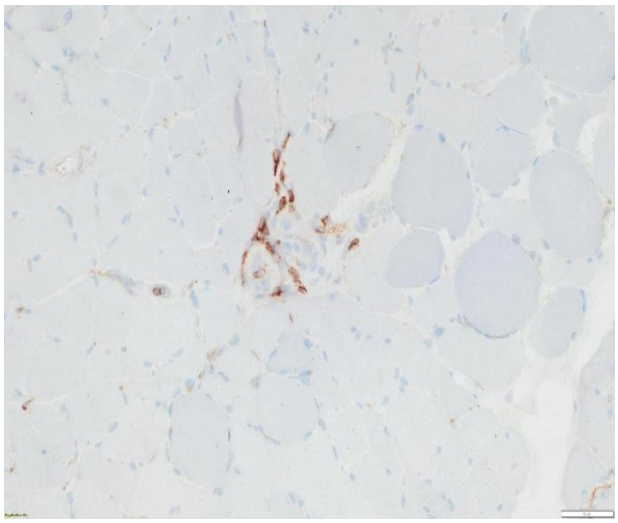


Figure 5: On muscle biopsy the inflammation consisted predominantly of CD8 predominant lymphocytes.

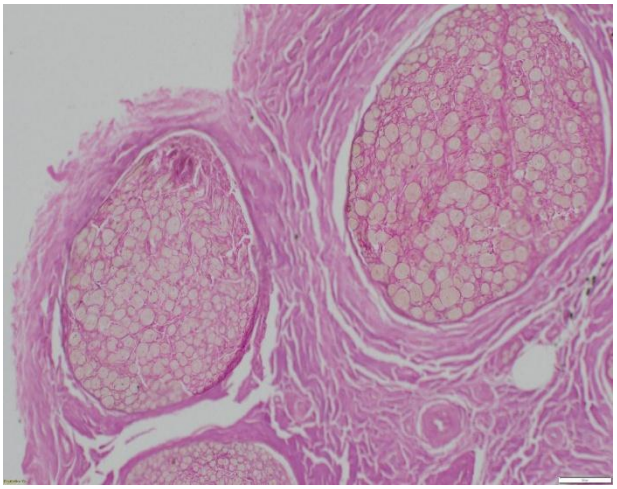


Figure 6: Nerve funicles with extensive pinch artefacts (H and E).

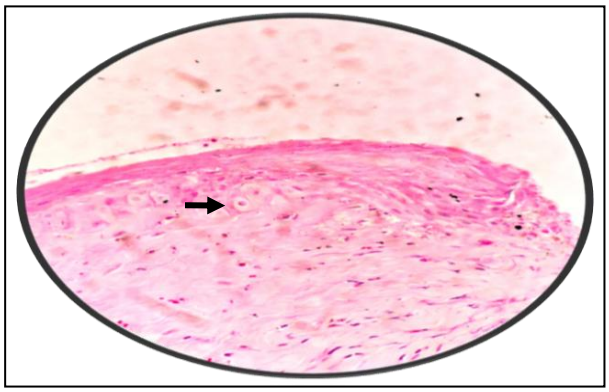


Figure 7: Basal vacuolar degeneration (arrow).

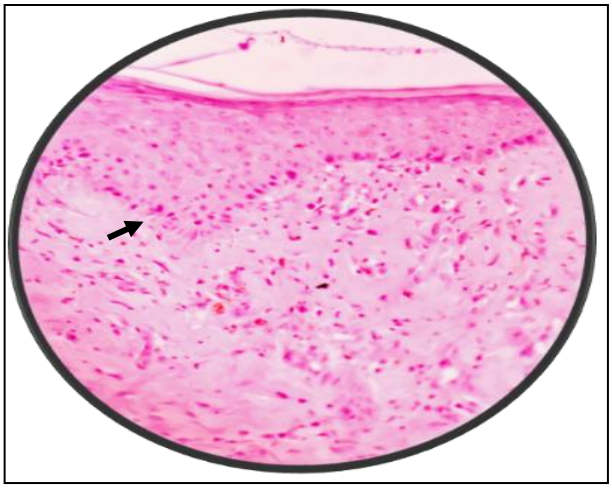


Figure 8: Thickened basement membrane (arrow).

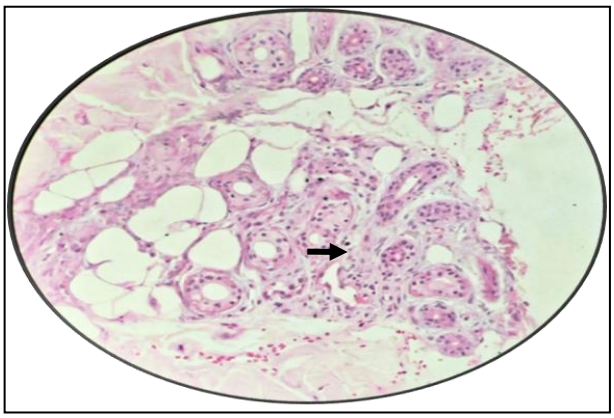


Figure 9: Periadnexal inflammatory infiltrates (arrow).

Table 1: Summary of investigations conducted with patient's results and normal reference range.

Investigations	Patient value	Normal / reference value
Hemoglobin	10.2 g/dl	13-17 g/dl
MCV	79.9 fL	83-101 fL
TLC/ WBC count	5,600 /µl	4,000-11,000 /µl
Platelet count	3,14,000 /µl	1,50,000-4,10,000 /µl
Serum iron	35 µg/dl	65-170 µg/dl
TIBC	113 µg/dl	255-450 µg/dl
Serum transferrin	<80 mg/dl	200-360 mg/dl
Serum ferritin	227 ng/ml	20-250 ng/ml
ESR	100 mm/hr	0-20 mm/hr
Urine protein	1000 mg/dl	<30 mg/dl
Urine protein/creatinine ratio	8.55	<0.15 g/g (normal)
Serum total bilirubin	0.29 mg/dl	0.3-1.2 mg/dl
Serum direct bilirubin	0.23 mg/dl	0.0-0.4 mg/dl
Serum total protein	6.80 g/dl	6.4-8.3 g/dl
Serum albumin	2.10 g/dl	3.5-5.2 g/dl
Serum aspartate transaminase (AST/SGOT)	61 U/l	15-40 U/l
Serum alanine transaminase (ALT/SGPT)	29 U/l	10-40 U/l
Serum alkaline phosphatase (ALP)	170 U/l	38-126 U/l
Serum globulin	4.70 g/dl	1.8-3.6 g/dl
Serum lactate dehydrogenase (LDH)	395 U/l	100-190 U/l

Continued.

Investigations	Patient value	Normal / reference value
Serum creatinine	0.40 mg/dl	0.66-1.25 mg/dl
Serum urea	18.0 mg/dl	16-40 mg/dl
BUN	8.41 mg/dl	6-20 mg/dl
Serum sodium (Na^+)	129 mEq/l	136-145 mEq/l
Serum potassium (K^+)	4.50 mEq/l	3.5-5.1 mEq/l
Serum chloride (Cl^-)	102 mEq/l	98-107 mEq/l
Serum calcium (Ca^{2+})	7.40 mg/dl	8.6-10.0 mg/dl
FBS	94 mg/dl	<100 mg/dl
PPBS	153mg/dl	<140 mg/dl
HIV antibody	Negative	Negative
HBsAg	Negative	Negative
Anti HCV antibody	Negative	Negative
ANA	3+; fine speckled nuclear pattern	Negative
Anti-Smith antibody	Positive	Negative
U1-snRNP antibody	Positive	Negative
ANCA	Weakly positive	Negative
MPO (p-ANCA)	Negative	Negative
PR3 (c-ANCA)	Negative	Negative
CPK	289 U/l	20-200 U/l

DISCUSSION

SLE is a heterogeneous autoimmune disorder with a wide range of manifestations which often mimic other inflammatory conditions, thus posing diagnostic challenges.[1,2]

It predominantly affects females, with a female to male ratio of 9:1 with male patients often presenting with more severe disease than females at diagnosis.[5]

The rarity of this disease in males along with atypical presentations leads to delays in diagnosis which can be detrimental considering the multisystem involvement.[5]

SLE is often referred to as the "great mimicker" because the lesions can resemble several inflammatory dermatological conditions, including Psoriasis.[2] Psoriasiform lupus is an uncommon but well recognized variant, in which the erythematous, scaly plaques closely resemble chronic plaque psoriasis.[3,4] Clinical distinction between the two is difficult, especially in chronic cases. In such cases, biopsy is helpful. Histopathological findings of basal vacuolar degeneration, thickened basement membrane, and perivascular and periadnexal inflammatory infiltrate favors the diagnosis of lupus over psoriasis.[4]

Our patient presented several diagnostic challenges. Firstly, he had been treated as having Psoriasis for nearly 20 years before the diagnosis of SLE was made. Secondly, it was his presentation with neurological complaints which prompted the evaluation for SLE. This highlights how SLE may be missed until systemic manifestations appear.[3,4]

Our patient fulfilled the diagnostic criteria for SLE based on biopsy proven lupus and positive serology (positive anti-Smith). Anti-Smith antibodies have high specificity for SLE.[1,2]

Another unique aspect of this case was the finding of biopsy proven myopathy. Musculoskeletal manifestations are one of the most prevalent presenting features in SLE. Arthralgia, myalgia, non-erosive arthritis and myositis are present in more than 90% of SLE patients. They significantly affect the patient's quality of life and daily functioning.[7] Myopathy is an uncommon feature of SLE, with reported prevalence ranging from 4% to 16%.[8] Muscle symptoms range from nonspecific myalgia and fatigue to clinically evident inflammatory myopathy. Although true SLE-associated myopathy is relatively uncommon, affected patients may present with symmetrical proximal muscle weakness, myalgia and elevated serum creatine kinase, with electromyographic, magnetic resonance imaging and histopathological evidence of muscle inflammation. Muscle pathology in SLE is heterogeneous and may include inflammatory myositis, dermatomyositis-like changes and necrotising myopathy, with necrotising myopathy being the predominant biopsy pattern in the cohort reported by Tiniakou et al.[8] Muscle weakness in SLE may also result from non-inflammatory mechanisms such as type 2 muscle fibre atrophy, disuse atrophy and corticosteroid-induced myopathy.[9] Muscle biopsy becomes essential in such cases to rule out other causes such as steroid induced myopathy, diabetic amyotrophy, metabolic myopathy or drug induced myopathy. In our patient, muscle biopsy done from the vastus lateralis showed features of necrotizing inflammatory myopathy, characterized by pipestem capillaries and mild endomysial, interstitial, and perivascular inflammatory infiltrates without fibrosis, which is consistent with immune-mediated necrotizing inflammatory myopathy.

This case highlights the importance of reevaluating treatment resistant dermatological conditions when new systemic manifestations arise.[3,4]

A multidisciplinary approach is integral to such cases to allow early diagnosis and institution of appropriate treatment to prevent morbidity and mortality.

CONCLUSION

This case highlights the broad clinical spectrum of lupus and the importance of reconsidering the diagnosis in patients with atypical or treatment resistant psoriasiform lesions, especially when accompanied by systemic manifestations.

Clinicians should maintain a high index of suspicion for connective tissue diseases in such patients. Early multidisciplinary evaluation involving dermatology, rheumatology, neurology, and pathology, combined with timely autoimmune serology and tissue biopsy, can facilitate earlier diagnosis, appropriate immunosuppressive therapy, and improved long-term outcomes.

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REFERENCES

1. Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, et al. 2019 European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. Ann Rheum Dis. 2019;78(9):1151-9.
2. Kuhn A, Sticherling M, Bonsmann G. Clinical manifestations of cutaneous lupus erythematosus. J Dtsch Dermatol Ges. 2007;5(12):1124-37.
3. Razi S, Truong TM, Sekhon P, Ouellette S, Rao BK. Subacute cutaneous lupus erythematosus with a psoriasiform presentation: a diagnostic clinical conundrum. Clin Case Rep. 2023;11(9):e7893.
4. Okon LG, Werth VP. Cutaneous lupus erythematosus: diagnosis and treatment. Best Pract Res Clin Rheumatol. 2013;27(3):391-404.
5. Murphy G, Isenberg DA. Effect of gender on clinical presentation in systemic lupus erythematosus. Rheumatology (Oxford). 2013;52(12):2108-15.
6. Ali M, Abdullah S, Abdulhameed Y, Elbadawi F. Systemic lupus erythematosus and psoriasis overlap: a case series. Cureus. 2025;17(5):e84760.
7. Smiljanić Tomičević L, Barešić M, Mayer M, Anić B. Musculoskeletal manifestations of systemic lupus erythematosus. Acta Clin Croat. 2023;62(4):714-23.
8. Tiniakou E, Goldman D, Corse A, Mammen A, Petri MA. Clinical and histopathological features of myositis in systemic lupus erythematosus. Lupus Sci Med. 2022;9(1):e000635.
9. Jakati S, Rajasekhar L, Uppin M, Challa S. SLE myopathy: a clinicopathological study. Int J Rheum Dis. 2015;18(8):886-91.

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