CASE REPORT

A case of progressive myelopathy with multisystem involvement suggestive of autoimmune etiology with neurocutaneous and vascular complications- A Case Report

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disorder with diverse clinical manifestations involving the skin, joints, nervous system, and vasculature. Neurological involvement, particularly lupus myelitis presenting as longitudinally extensive transverse myelitis (LETM), is rare but associated with significant morbidity. Vascular complications such as digital ischemia further indicate severe disease activity and possible overlap with antiphospholipid syndrome.

Case Presentation: A 38-year-old female presenting with an 8-year history of inflammatory polyarthritis, followed by progressive sensory disturbances, bilateral lower limb weakness, and bowel and bladder dysfunction. She also had a photosensitive facial rash and dysphagia. Neurological evaluation suggested myelopathy, and MRI spine revealed longitudinally extensive spinal cord involvement consistent with transverse myelitis. Autoimmune workup showed positivity for antinuclear antibodies, anti-dsDNA, and anti-Smith antibodies. Additionally, examination revealed digital ischemia of the toe, suggestive of vascular involvement. The constellation of findings was consistent with systemic lupus erythematosus with lupus myelitis and probable vasculitic or thrombotic complications.

Conclusion: This case emphasizes the importance of recognizing multisystem involvement in SLE, particularly rare neurological and vascular manifestations, to enable early diagnosis and timely management.

Keywords: Systemic lupus erythematosus, lupus myelitis, longitudinally extensive transverse myelitis, neuropsychiatric SLE, digital ischemia, vasculitis, antiphospholipid syndrome, inflammatory polyarthritis, autoimmune disease, spinal cord involvement

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Introduction

Systemic autoimmune diseases represent a heterogeneous group of disorders characterized by immune-mediated damage to multiple organ systems. Among these, systemic lupus erythematosus (SLE) is one of the most clinically diverse conditions, with manifestations ranging from mild mucocutaneous involvement to severe, life-threatening neurological and vascular complications (1). The disease predominantly affects women in the reproductive age group and is marked by the production of autoantibodies against nuclear and cytoplasmic antigens, leading to immune complex deposition and widespread tissue inflammation (2).

Neurological involvement in SLE, termed neuropsychiatric SLE (NPSLE), encompasses a wide spectrum of central and peripheral nervous system manifestations. One of the rare but severe neurological complications is lupus myelitis, which typically presents as acute or subacute spinal cord dysfunction (3). Patients may develop motor weakness, sensory disturbances, and autonomic dysfunction including bowel and bladder involvement. In many cases, magnetic resonance imaging (MRI) reveals longitudinally extensive transverse myelitis (LETM), characterized by spinal cord lesions extending over multiple vertebral segments (4). This form of myelopathy is associated with significant morbidity and often requires prompt recognition and aggressive immunosuppressive therapy to prevent irreversible neurological deficits (5).

Cutaneous manifestations are another hallmark of SLE, with photosensitivity and malar rash being among the most characteristic features. These dermatological signs often serve as important clinical clues to the underlying autoimmune pathology (6,7). In addition, musculoskeletal involvement, particularly inflammatory

polyarthritis, is commonly observed and may precede systemic manifestations by several years (8). The presence of chronic joint pain with morning stiffness is frequently misdiagnosed as other rheumatological conditions, thereby delaying appropriate evaluation and management (9).

Vascular involvement in SLE further complicates the clinical picture. Patients may develop vasculitis or antiphospholipid antibody syndrome (APS), leading to thrombotic events and ischemic complications such as digital gangrene (10,11). The coexistence of neurological deficits and peripheral vascular compromise often indicates severe disease activity and necessitates a multidisciplinary approach to management (12).

The diagnosis of SLE is based on a combination of clinical features and immunological markers, including antinuclear antibodies (ANA), anti-double stranded DNA (anti-dsDNA), and anti-Smith (anti-Sm) antibodies (13). Early identification of organ involvement, particularly of the nervous system, is crucial for initiating timely treatment. Despite advances in diagnostic modalities and therapeutic strategies, atypical and delayed presentations remain a significant challenge in clinical practice (14,15).

In this report, we present a case of a young female with long-standing inflammatory arthritis who developed progressive neurological deficits, autonomic dysfunction, and vascular complications, ultimately suggestive of an underlying systemic autoimmune disorder with spinal cord involvement. This case underscores the importance of recognizing multisystem involvement in autoimmune diseases and highlights the need for early diagnosis and prompt intervention to improve patient outcomes.

CASE PRESENTATION

Identification: A 38-year-old female, a farmer by occupation and resident of Mudagar, Lingasur, presented to the hospital with chronic joint pain, progressive weakness of both lower limbs, sensory disturbances, bladder and bowel dysfunction, and recent onset imbalance while walking. She is a right-handed individual with no formal education and communicates primarily in Kannada.

Chief Complaints: The patient presented with complaints of multiple joint pains for the past 8 years, tingling and burning sensation in both lower limbs for 6 years, progressive weakness of both lower limbs for 5 years, bowel and bladder disturbances for 5 years, and imbalance while walking for the last 9 months.

History of Present Illness: The patient was apparently asymptomatic 8 years ago, when she gradually developed pain in multiple joints. The onset was insidious, initially involving large joints such as bilateral shoulders, elbows, and knees. Over time, the disease progressed to involve the ankle joints and subsequently the small joints of the hands. The joint pain was episodic in nature, progressively increasing in severity with each episode. It was characteristically worse in the early morning and associated with stiffness lasting more than 30 minutes, which improved with activity. These features were suggestive of an inflammatory arthritis. There was no history of joint swelling, deformity, redness, or restriction of joint movements. Additionally, there were no associated constitutional symptoms such as fever, weight loss, or loss of appetite, making infectious or malignant causes less likely.

Approximately 6 years prior to presentation, the patient began experiencing sensory disturbances in the lower limbs, described as tingling and burning sensations. These symptoms initially started in the plantar aspect of the left foot and gradually progressed in an ascending manner to involve both lower limbs up to the level of the hips. Over time, the symptoms became bilateral and symmetrical, suggesting involvement of long tracts.

Around 5 years prior to presentation, she developed progressive weakness of both lower limbs. Initially, she experienced difficulty in rising from a squatting position and required support from her upper limbs. Gradually, her condition worsened, and she developed difficulty getting up from a supine position, indicating proximal muscle weakness. She also complained of heaviness and stiffness in the lower limbs while walking, which interfered with her daily activities.

During the same period, she developed autonomic dysfunction in the form of bladder and bowel disturbances. She reported increased urinary frequency, passing urine approximately 10–12 times during the day and 6–7 times at night, along with urgency and hesitancy. There was also difficulty initiating the stream of urine. She complained of chronic constipation, requiring excessive straining during defecation and often needing laxatives, with bowel movements occurring only once weekly.

Nine months prior to presentation, she noticed imbalance while walking, which was initially mild and more pronounced in low-light conditions or darkness, suggestive of sensory ataxia. Over time, the imbalance progressed, requiring support while walking, and she developed a tendency to fall backward. She also reported difficulty in turning and negotiating uneven surfaces.

Additionally, she complained of dysphagia, particularly for solid foods, requiring water intake with each swallow. She described a sensation of dryness in the throat and a foreign body sensation in the eyes, which may indicate associated mucosal involvement.

Cutaneous History: The patient developed an erythematous rash over the face involving the cheeks, forehead, and upper lip. The onset was insidious, and over time the rash became darker and more prominent. It was associated with photosensitivity, with worsening

on exposure to sunlight. There was no associated itching, pain, or scaling. There were no similar lesions elsewhere on the body, and no history of oral ulcers or alopecia.

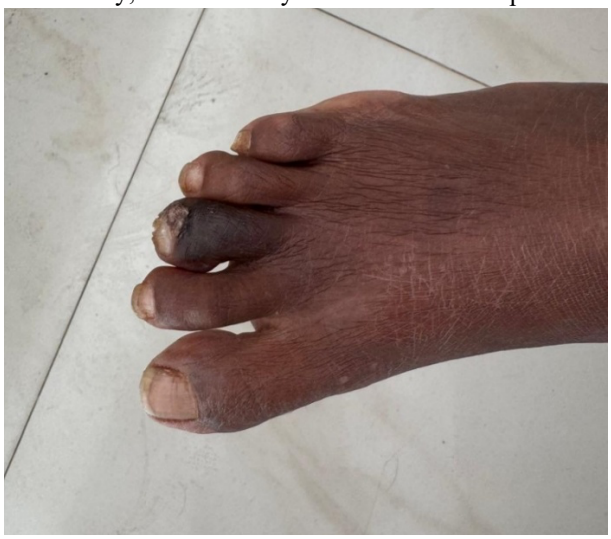

Figure 1: Digital Ischemia of Toe Suggestive of Vasculitis/Thrombotic Complication in Systemic Lupus Erythematosus

Negative History: There was no history suggestive of involvement of the upper limbs, including weakness, sensory disturbances, or difficulty performing fine motor activities such as combing hair, buttoning clothes, or lifting objects. There were no cranial nerve symptoms such as diplopia, facial asymmetry, slurring of speech, or hearing impairment. There was no history of back pain, radicular pain, or band-like sensation around the trunk. There were no involuntary movements such as fasciculations. There was no history suggestive of autonomic instability such as excessive sweating, palpitations, or postural dizziness apart from bladder involvement. There was also no history of trauma, recurrent infections, or respiratory complaints.

Past History: The patient has no history of similar complaints in the past. She is not a known case of diabetes mellitus, hypertension, coronary artery disease, or cerebrovascular accident. There is no history suggestive of tuberculosis, malignancy, or chronic systemic illness. She has not been on any long-term medications and was not on regular follow-up for her joint symptoms.

Personal History: The patient consumes a mixed diet and reports adequate appetite and sleep. She has a significant history of tobacco chewing for approximately 25 years. There is no history of alcohol consumption or substance abuse.

Family History: She is married and has three children, all of whom are healthy. There is no significant family history of autoimmune diseases, neurological disorders, or similar complaints in close relatives.

Menstrual and Obstetric History: She attained menarche at the age of 11 years and reports regular menstrual cycles with normal flow and no dysmenorrhea. Obstetric history reveals three full-term normal vaginal deliveries, with no history of abortions or stillbirths.

General Physical Examination: On general examination, the patient was conscious, coherent, and cooperative. She was well oriented to time, place, and person. Her anthropometric measurements revealed a height of 156 cm, weight of 54 kg, and a body mass index of 22.2 kg/m², indicating normal nutritional status. Her pulse rate was 78 beats per minute, regular in rhythm with normal volume and character. Blood pressure was recorded as 126/82 mmHg in the right arm in the supine position. Respiratory rate was 16 breaths per minute, and she was afebrile.

Peripheral pulse examination revealed that all pulses were palpable except for the left dorsalis pedis artery, suggesting possible peripheral vascular compromise.

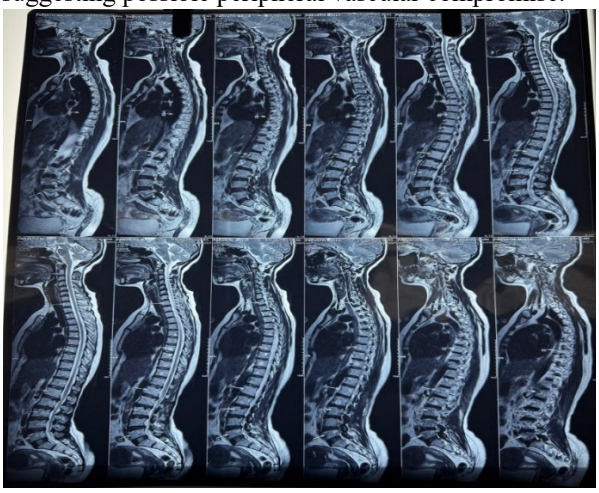

Figure 2: MRI Spine Showing Longitudinally Extensive Spinal Cord Lesion Suggestive of Transverse Myelitis (Lupus Myelitis)

Local Examination: Examination of the right foot revealed blackish discoloration of the third toe, which appeared dry and non-tender, suggestive of digital ischemia or gangrene. This finding is indicative of vascular involvement, possibly secondary to vasculitis or thrombotic events associated with autoimmune disease.

Systemic Examination: Based on the clinical presentation, the neurological examination is suggestive of involvement of the spinal cord. The patient demonstrates features of progressive spastic paraparesis, with bilateral lower limb weakness, ascending sensory disturbances, and significant autonomic dysfunction involving bladder and bowel control. The presence of imbalance, particularly worsening in darkness, suggests involvement of posterior columns in addition to corticospinal tracts, indicating a combined tract involvement consistent with myelopathy.

Investigations: Laboratory evaluation revealed a positive autoimmune profile, including antinuclear antibodies, anti-double stranded DNA antibodies, and anti-Smith antibodies, strongly suggestive of systemic lupus erythematosus. Other autoimmune markers were either negative or borderline.

Magnetic resonance imaging of the spine demonstrated long segment involvement of the spinal cord, consistent with longitudinally extensive transverse myelitis. This supports an inflammatory or autoimmune etiology of spinal cord dysfunction.

Provisional Diagnosis: The clinical presentation is most consistent with systemic lupus erythematosus with lupus myelitis, associated with neurogenic bladder and vascular involvement, possibly due to vasculitis or antiphospholipid syndrome.

Differential Diagnosis: The differential diagnoses to be considered include neuromyelitis optica spectrum disorder, Sjögren's syndrome with neurological involvement, mixed connective tissue disease, and antiphospholipid antibody syndrome presenting with vascular and neurological manifestations.

DISCUSSION

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disorder with highly variable clinical manifestations involving the musculoskeletal, dermatological, neurological, and vascular systems. The present case demonstrates a classical yet severe spectrum of SLE, with progression from inflammatory arthritis to neurovascular complications, consistent with observations reported in recent literature. Dai et al. (2025) described SLE as a disease driven by complex immune dysregulation involving autoantibody production and immune complex deposition, leading to multiorgan damage (1). The combination of joint involvement, cutaneous manifestations, and neurological deficits in our patient aligns with this pathophysiological framework.

The initial presentation of chronic inflammatory polyarthritis in this case closely resembles the pattern described by Cush and Dao (2025), where polyarticular involvement, particularly affecting both large and small joints with morning stiffness, is characteristic of inflammatory rheumatological disorders (8). Similarly, Orange et al. (2020) emphasized that prolonged morning stiffness reflects active synovial inflammation, supporting the inflammatory nature of the patient's joint symptoms (9). In our case, the long-standing arthritis preceding systemic manifestations highlights the diagnostic challenge and potential delay in recognizing underlying SLE.

Cutaneous involvement in the form of photosensitive facial rash further supports the diagnosis. Fijałkowska et al. (2024) and Uva et al. (2012) have described photosensitivity and malar rash as hallmark features of SLE, often serving as early clinical indicators (6,7). The patient's rash, localized to sun-exposed areas and associated with pigmentation changes, is consistent with these findings, reinforcing the multisystem nature of the disease.

Neurological involvement in SLE, particularly lupus myelitis, is rare but carries significant morbidity. Jayasinghe et al. (2025) highlighted that neuropsychiatric SLE can present with spinal cord involvement manifesting as motor weakness, sensory deficits, and autonomic dysfunction (3). Our patient exhibited progressive spastic paraparesis, ascending sensory symptoms, and bladder dysfunction, which are classical features of myelopathy. Furthermore, Banjade et al. (2024) reported longitudinally extensive transverse myelitis (LETM) as a recognized but uncommon manifestation of SLE, often associated with severe disease activity (4). The MRI findings in our case demonstrating long segment spinal cord involvement are in direct concordance with their observations.

Vascular involvement, as evidenced by digital ischemia in this patient, further supports the severity of systemic disease. Pyrpasopoulou et al. (2012) described vascular complications in SLE as resulting from both inflammatory vasculitis and thrombotic mechanisms (11). Cravero et al. (2022) reported digital ulcers and ischemia as potential manifestations of secondary antiphospholipid syndrome, which may coexist with SLE (10). The presence of gangrenous changes in our patient's toe strongly suggests an underlying vascular or thrombotic pathology, possibly linked to antiphospholipid antibodies.

The diagnosis of SLE in this case is supported by serological markers including ANA, anti-dsDNA, and anti-Sm antibodies, which are well-established diagnostic biomarkers. Yu et al. (2021) emphasized the importance of these immunological markers in confirming SLE and assessing disease activity (13). The coexistence of serological positivity with clinical manifestations involving multiple organ systems strengthens the diagnosis.

In comparison to other neurological conditions such as cervical myelopathy described by Afifi et al. (2025), the presence of systemic features and autoimmune markers in our case clearly favors an inflammatory autoimmune etiology rather than a purely structural cause (5). Overall, this case highlights a rare but severe presentation of SLE with simultaneous neurological and vascular involvement, emphasizing the importance of early recognition and multidisciplinary management.

CONCLUSION

This case highlights a rare and severe presentation of systemic lupus erythematosus characterized by progressive myelopathy with multisystem involvement, including musculoskeletal, cutaneous, neurological, and

vascular manifestations. The patient's long-standing inflammatory polyarthritis, photosensitive rash, and strongly positive autoimmune serology were suggestive of SLE, while the development of longitudinally extensive transverse myelitis and digital ischemia indicated advanced disease with significant morbidity. The coexistence of neurological deficits and vascular compromise raises the possibility of associated vasculitis or antiphospholipid syndrome, further emphasizing the complexity of the disease process.

This case underscores the importance of maintaining a high index of suspicion for autoimmune etiology in patients presenting with progressive neurological symptoms and systemic features. Early diagnosis, supported by imaging and serological markers, is crucial to initiate timely immunosuppressive therapy and prevent irreversible neurological damage. A multidisciplinary approach is essential for optimal management and improved clinical outcomes.

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