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CASE REPORT

Early-onset peripheral sensorimotor neuropathy associated with systemic lupus erythematosus: a case report

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ABSTRACT

Background: Peripheral neuropathy is an uncommon but potentially disabling manifestation of systemic lupus erythematosus (SLE), particularly when it occurs early in the disease course. Its clinical and electrophysiological heterogeneity often makes diagnosis challenging. **Case Presentation:** We report the case of a 21-year-old man presenting with inflammatory arthralgia followed by progressive sensorimotor peripheral neuropathy predominantly affecting the lower limbs. Electroneuromyography revealed a diffuse sensorimotor polyneuropathy with severe axonal involvement and demyelinating features. Immunological investigations showed high-titer antinuclear antibodies, positive anti-double-stranded DNA and anti-nucleosome antibodies, and normal complement levels. A structured diagnostic work-up excluded alternative metabolic, infectious, toxic, and drug-related etiologies. The diagnosis of active SLE was established according to the 2019 ACR/EULAR classification criteria. Treatment with high-dose corticosteroids, hydroxychloroquine, and immunosuppressive therapy resulted in progressive clinical and electrophysiological improvement. **Conclusion:** This case highlights that peripheral sensorimotor neuropathy may represent an early manifestation of SLE. Early recognition and individualized immunosuppressive management are essential to improve neurological outcomes and limit long-term disability.

Keywords: systemic lupus erythematosus, peripheral neuropathy, neuropsychiatric lupus, sensorimotor neuropathy, immunosuppressive therapy.

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1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic inflammatory autoimmune disease characterized by multisystem involvement, driven by a loss of immune tolerance, polyclonal B-cell activation, and the production of autoantibodies targeting various cellular components. The broad spectrum of clinical manifestations makes this disease a considerable diagnostic and therapeutic challenge. Among these manifestations, neurological involvement, grouped under the term neuropsychiatric lupus (NPSLE), occupies a particular position. It encompasses a wide range of conditions, from seizures to lupus psychosis, as well as cerebrovascular accidents and peripheral neuropathies. The latter are relatively rare, with an estimated prevalence of 2–6% according to published studies (1), but they represent a significant source of functional morbidity.

Peripheral neuropathies associated with SLE are heterogeneous, both clinically and pathophysiologically. They may be axonal, demyelinating, sensory, motor, mixed, or present as mononeuritis multiplex. A notable feature concerns peripheral neuropathies occurring early in the disease course: although uncommon, they are often described as more severe, typically axonal, and associated

with high autoimmune activity. In this context, we report the case of a young man presenting with a sensory–motor peripheral neuropathy as an early manifestation associated with active systemic lupus erythematosus. This observation illustrates the diagnostic complexity of such atypical presentations and provides an opportunity for a comprehensive review of the recent literature.

2. CASE REPORT

A 21-year-old male university student with no significant past medical history was admitted for the evaluation of inflammatory arthralgia evolving for approximately one month. The pain involved both large and small joints in a bilateral and symmetrical distribution, was predominantly nocturnal, associated with prolonged morning stiffness, and was partially and transiently relieved by nonsteroidal anti-inflammatory drugs.

The clinical course was subsequently marked by the progressive onset of neurological symptoms characterized by hypoesthesia predominantly affecting the lower limbs, followed by gradually worsening motor weakness. Functional impairment increased over time, resulting in gait instability and inability to maintain single-leg stance. On history taking, the patient reported no recent infection, whether respiratory, gastrointestinal, or otorhinolaryngological, no recent vaccination, no febrile episodes, no radicular pain, and no symptoms suggestive of dysautonomia (palpitations, orthostatic hypotension, or sphincter disturbances). There was no history of alcohol consumption, illicit drug use, or recent or chronic intake of medications known to have potential neurotoxicity.

On physical examination, the patient was afebrile, hemodynamically stable, and eupneic. His weight was 122 kg and height 180 cm (body mass index 37.6 kg/m²). Cutaneous examination revealed pallor of the skin and mucous membranes associated with photosensitivity of the forearms, without typical lupus rash or vasculitic lesions. Ophthalmologic examination showed no abnormalities. Joint examination demonstrated pain at the end range of motion of the shoulders and wrists, a positive squeeze test of the hands, with no clinically evident synovitis or sacroiliac joint involvement. Gait was waddling, with marked postural instability.

Neurological examination revealed motor weakness predominantly affecting the lower limbs, graded 3/5, mainly involving proximal muscles (iliopsoas and quadriceps) as well as the ankle dorsiflexors. Moderate motor involvement of the upper limbs was also noted (4/5), predominantly affecting proximal muscles. Barré and Mingazzini tests were positive in less than ten seconds. Deep tendon reflexes were preserved and symmetric in both the upper and lower limbs. Sensory examination demonstrated thermo-algesic hypoesthesia of the right thigh, with preserved deep sensation and no proprioceptive ataxia. Cranial nerve examination was unremarkable.

Initial laboratory investigations revealed a marked inflammatory syndrome, with an erythrocyte sedimentation rate of 85 mm in the first hour and a C-reactive protein level of 78 mg/L. Complete blood count showed moderate normocytic normochromic anemia (hemoglobin 11–12 g/dL), without leukopenia or thrombocytopenia. The direct Coombs test was negative. Serum ferritin, vitamin B12, and folate levels were within normal ranges. Serum electrolytes and thyroid function tests (TSH) were normal. Renal function was preserved, with minimal proteinuria (urine protein-to-creatinine ratio < 0.3 g/g) and an unremarkable urinary sediment. Fasting plasma glucose was mildly elevated, with a glycated hemoglobin (HbA1c) level of 6.08%, without a prior diagnosis of diabetes mellitus.

Immunological investigations were highly suggestive of systemic lupus erythematosus, revealing antinuclear antibodies at a titer > 1:1000, elevated anti–double-stranded DNA antibodies, and positive anti-nucleosome and anti-histone antibodies. Antiphospholipid antibodies, ANCA, anti-SSA/SSB, anti-Sm, and anti-RNP antibodies were negative. Complement levels (C3 and C4) were within normal ranges. Serum protein electrophoresis demonstrated polyclonal hypergammaglobulinemia. Cerebrospinal fluid analysis showed mild lymphocytic pleocytosis (10 cells/mm³), with normal protein and glucose levels, and no albuminocytologic dissociation. Infectious serologies (HIV, hepatitis B and C viruses, Epstein–Barr virus, cytomegalovirus) were negative. Cryoglobulinemia testing was negative.

Electroneuromyography, performed on three occasions during follow-up, demonstrated a diffuse sensorimotor peripheral neuropathy combining severe axonal involvement with demyelinating features, predominantly affecting the lower limbs, with persistent signs of partial denervation on follow-up studies. Brain and spinal magnetic resonance imaging did not reveal any central inflammatory lesions. Chest radiography was normal. Transthoracic echocardiography showed no abnormalities. Plain radiographs of the hands and wrists showed no erosive changes. Musculoskeletal ultrasound revealed multiple synovitides, particularly involving the wrists and hands, without erosions.

Taken together, the clinical, biological, immunological, and electrophysiological findings supported the diagnosis of active systemic lupus erythematosus according to the 2019 ACR/EULAR classification criteria, with peripheral neurological and articular involvement. The SLEDAI-2K score was assessed at 6. High-dose systemic corticosteroid therapy (1 mg/kg/day) was initiated, combined with hydroxychloroquine at a dose of 400 mg/day, vitamin supplementation (vitamins B1, B6, and B12), and functional rehabilitation. Adjunctive measures were implemented concomitantly with high-dose corticosteroid therapy, including calcium and vitamin D

supplementation, gastric protection, and close metabolic monitoring, particularly in view of the patient's elevated body mass index and mildly increased glycated hemoglobin level. Azathioprine was subsequently introduced at an immunosuppressive dose of 2 mg/kg/day but was discontinued due to digestive intolerance associated with moderate hepatic cytolysis. Treatment was then switched to mycophenolate mofetil at a dose of 2 g/day.

Clinical evolution under corticosteroids and mycophenolate mofetil was favorable, with progressive improvement in muscle strength, regression of joint pain, stabilization followed by partial improvement of sensory symptoms, normalization of inflammatory markers, and improvement of electrophysiological parameters on follow-up studies.

3. DISCUSSION

Peripheral neuropathy is a recognized but relatively uncommon manifestation of SLE, particularly when it occurs early in the disease course or contributes to its diagnosis. Large prospective cohorts and inception studies report a prevalence of peripheral nervous system involvement ranging from approximately 2% to 7%, markedly lower than that of central nervous system manifestations, which likely contributes to under-recognition of these presentations in clinical practice (1–4). Nevertheless, peripheral neuropathies may be associated with significant functional morbidity and therefore warrant careful evaluation.

From a clinical and electrophysiological standpoint, lupus-associated peripheral neuropathies are highly heterogeneous. Sensorimotor polyneuropathies with predominant axonal involvement are the most frequently reported patterns, followed by mononeuritis multiplex, often related to immune-mediated small-vessel vasculitis of the vasa nervorum (2-6). Purely demyelinating forms remain uncommon; however, mixed axonal–demyelinating neuropathies have been increasingly described in both cohort studies and case reports, reflecting the complexity of the underlying immunopathological mechanisms (2,3,5).

A structured diagnostic approach integrating clinical presentation, neurological examination, targeted laboratory testing, and electrophysiological studies is essential to accurately classify peripheral neuropathies and exclude alternative etiologies, as proposed in widely accepted diagnostic algorithms (7).

In the present case, the combination of a subacute onset, symmetrical sensorimotor involvement affecting both proximal and distal muscle groups, preserved deep tendon reflexes, and the absence of marked asymmetry or multifocal deficits argues against mononeuritis multiplex or a purely vasculitic neuropathy. Likewise, the presence of severe axonal damage associated with demyelinating features on electroneuromyography, in the absence of cerebrospinal fluid albuminocytologic dissociation, makes a primary demyelinating neuropathy or Guillain–Barré syndrome unlikely (5,7).

The electrophysiological findings are therefore consistent with a diffuse sensorimotor polyneuropathy with predominant axonal involvement and secondary demyelinating changes, a pattern classically reported in lupus-associated peripheral neuropathy and often correlated with higher systemic immune activity (2,3,6). Electroneuromyography remains the cornerstone for lesion characterization, severity assessment, and longitudinal monitoring, allowing differentiation between axonal, demyelinating, and mixed patterns, and guiding both etiological attribution and therapeutic decisions (2,6,7).

An additional point worth discussing concerns the preservation of deep tendon reflexes in our patient despite the presence of severe axonal involvement on electroneuromyography. Although reduced or absent reflexes are commonly described in peripheral neuropathies, preserved reflexes may still be observed in early or partial nerve involvement. Moreover, mixed axonal–demyelinating patterns may lead to clinical–electrophysiological discrepancies. In addition, although the predominance of proximal weakness could raise the possibility of an inflammatory myopathy, muscle enzyme levels were within normal ranges and myositis-specific antibodies were negative. Furthermore, electroneuromyographic findings supported a neuropathic rather than a myopathic process. These elements support the diagnosis of peripheral neuropathy in the present case.

The pathophysiology of lupus-related peripheral neuropathy is complex and involves both innate and adaptive immune mechanisms (Figure 1). Polyclonal B-cell activation, partly driven by plasmacytoid dendritic cells, leads to increased production of pathogenic autoantibodies, including anti–double-stranded DNA, anti-nucleosome, and anti-histone antibodies, as observed in our patient (6,8,9). These autoantibodies promote immune complex formation, which may deposit in perineural tissues or the vasa nervorum, resulting in local inflammation and ischemic nerve injury (8,9).

Type I interferon signaling plays a central amplifying role in this process. Interferon-α enhances T-helper cell activation, plasma cell differentiation, and endothelial expression of adhesion molecules, thereby facilitating immune cell recruitment into peripheral nerve tissues (6,8,10). Pro-inflammatory cytokines such as interleukin-6, tumor necrosis factor-α, and interleukin-17 further contribute to macrophage activation, sustained perineural inflammation, and myelin damage, accounting for the mixed axonal and demyelinating patterns observed in some patients (10–12).

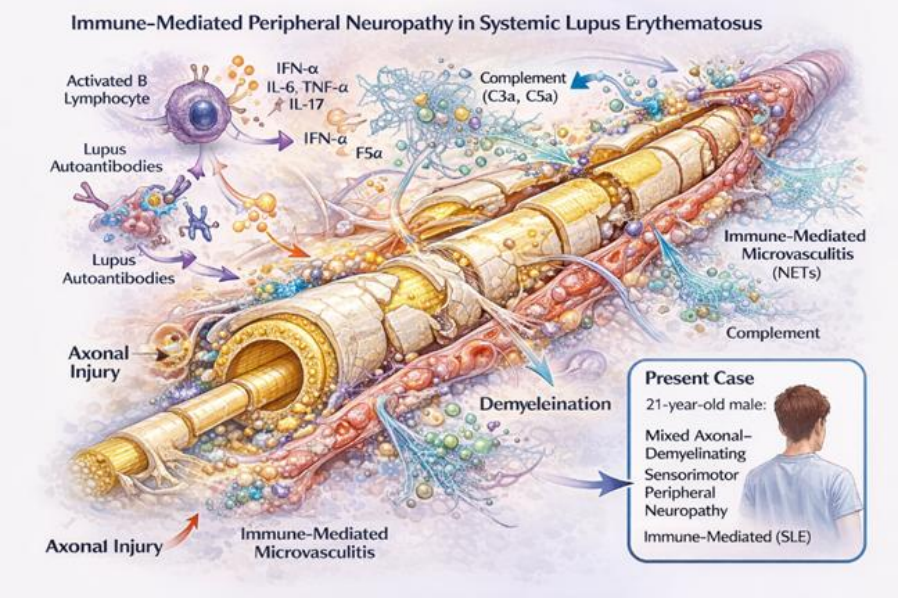

Figure 1. Immunopathological mechanisms of peripheral nerve involvement in SLE. Schematic illustration created by the authors based on the mechanisms described in the literature. *(Schematic overview of immune-mediated peripheral nerve injury highlighting autoantibody production, inflammatory pathways, and mixed axonal–demyelinating damage in the present case.)*

Complement activation and thrombo-inflammatory mechanisms also play an important role. Although hypocomplementemia is commonly associated with active SLE, normal circulating complement levels do not exclude local complement activation within affected tissues. In addition, the formation of neutrophil extracellular traps (NETs), a hallmark of SLE, promotes endothelial dysfunction, microvascular injury, and immunothrombosis, potentially affecting the vasa nervorum even in the absence of antiphospholipid antibodies (12–14). These mechanisms provide a pathophysiological basis for the frequent predominance of axonal damage and the occurrence of mixed neuropathic patterns.

Histopathological data from reported nerve biopsies in patients with SLE have demonstrated small-vessel vasculitis, perivascular and perineural inflammatory infiltrates, and immune complex deposition, supporting these mechanistic hypotheses (15,16). Although nerve biopsy was not performed in our patient, the convergence of clinical, immunological, and electrophysiological findings strongly supports an immune-mediated lupus-related peripheral neuropathy.

Attribution of peripheral neuropathy to SLE requires a rigorous diagnostic approach combining clinical, biological, electrophysiological, and evolutionary data. In the present case, an extensive evaluation excluded alternative etiologies, including metabolic, nutritional, endocrine, infectious, toxic, and drug-related causes (7).

Diabetic neuropathy was considered in the differential diagnosis given the mildly elevated glycated hemoglobin level (HbA1c 6.08%). However, several clinical, biological, and evolutionary arguments allow this etiology to be reasonably excluded in our patient. Diabetic peripheral neuropathy typically occurs in the context of long-standing chronic hyperglycemia and most often presents as a slowly progressive, distal symmetric polyneuropathy with predominant sensory involvement, usually developing over several years (17,18). In contrast, our patient had no prior history of diabetes mellitus, and the modest elevation of HbA1c remained below levels classically associated with clinically overt diabetic neuropathy.

Moreover, close glycemic monitoring, including a capillary blood glucose profile performed during high-dose systemic corticosteroid therapy, did not reveal significant or persistent hyperglycemia, further arguing against chronic glycemic imbalance as the underlying cause of the neurological manifestations. From a clinical and electrophysiological perspective, the presentation was also inconsistent with diabetic neuropathy, which predominantly affects distal nerve fibers with late proximal extension and limited early motor involvement. The subacute course, early sensorimotor involvement including proximal muscle groups, and the electrophysiological pattern combining severe axonal damage with demyelinating features are atypical for classic diabetic peripheral neuropathy (7,18).

No exposure to neurotoxic substances was identified, and the immunological profile was strongly suggestive of active SLE. Moreover, the favorable clinical and electrophysiological response to immunosuppressive therapy further supports lupus-related neuropathy (2,5).

Several case series and reports describe peripheral neuropathy as an early or revealing manifestation of SLE, particularly in young patients and occasionally in males. A comparison between previously published cases and the present observation is summarized in Table 1. Florica et al. reported predominantly axonal neuropathies with a notable subset of mixed forms, while Bortoluzzi et al. described cases of early sensorimotor neuropathy with favorable outcomes following corticosteroid-based immunosuppressive therapy (2,3). Isolated reports have also documented severe peripheral neuropathy leading to the diagnosis of SLE, with partial or complete recovery after immunosuppressive treatment, although residual deficits may persist in cases with marked axonal involvement (7,16,19,20).

Table 1. Comparison of published cases of peripheral neuropathy associated with systemic lupus erythematosus and the present case.

Reference	Age / Sex	Neuropathy (type)	Time of onset	Immunological profile	Treatment instituted	Outcome
Florica et al 2011 (2)	38 years (median) F > M	Sensorimotor polyneuropathy, predominantly axonal	Early or during the course of SLE	ANA+, anti-dsDNA frequent, variable complement levels	Corticosteroids ± AZA / CYC	Clinical improvement in most cases
Bortoluzzi et al 2017 (20)	34 years (median) F > M	Heterogeneous peripheral neuropathy (axonal, mononeuritis)	Inaugural or early	High immunological activity	Corticosteroids ± CYC	Favorable outcome with early treatment
Sciascia et al 2014 (16)	27 years / F	Severe sensorimotor polyneuropathy	Inaugural	ANA+, anti-dsDNA+	Corticosteroids + CYC	Partial recovery
Bortoluzzi et al 2019 (3)	30–40 years F > M	Axonal ± mixed neuropathy	Variable	Correlated with lupus activity	Corticosteroids ± IS	Frequent improvement
Gumkowska-Sroka et al 2024 (21)	Variable	Inflammatory neuropathies	Variable	Associated systemic activity	Corticosteroids ± IS	Depends on axonal severity
Present case	21 years / M	Diffuse sensorimotor polyneuropathy, severe axonal involvement with demyelinating features	Early	ANA >1:1000, anti-dsDNA+, anti-nucleosome+, normal complement levels	Corticosteroids + AZA → MMF	Progressive clinical and electrophysiological improvement

AZA: azathioprine; CYC: cyclophosphamide; IS: immunosuppressive therapy; MMF: mycophenolate mofetil.

Therapeutic management of lupus-associated peripheral neuropathy is based primarily on observational data and expert recommendations. Systemic corticosteroids remain the cornerstone of first-line treatment, particularly in inflammatory or progressive forms, allowing rapid reduction of immune-inflammatory activity (21).

Cyclophosphamide is generally reserved for severe, rapidly progressive forms or those associated with documented vasculitis of the peripheral or central nervous system (21). In the present case, despite clinically significant neuropathy, the absence of evidence for severe systemic vasculitis, lack of renal or central nervous system involvement, and early improvement under corticosteroid therapy supported the choice of a less aggressive immunosuppressive strategy.

Azathioprine was therefore selected as maintenance immunosuppressive therapy in combination with corticosteroids, in accordance with common practice for moderate peripheral nervous system involvement in SLE (23,24). Its discontinuation was prompted by digestive intolerance associated with moderate hepatic cytolysis, leading to a switch to mycophenolate mofetil, used as an effective and generally well-tolerated alternative in systemic manifestations of SLE (23,24). Written informed consent was obtained prior to initiation of mycophenolate mofetil, given its off-label use in certain neurological manifestations of SLE and the need to inform the patient about expected benefits, potential risks—particularly infectious and hematological—and the requirements for regular laboratory monitoring.

B-cell–targeted biologic therapies, particularly rituximab, have been used in refractory cases of lupus-associated peripheral neuropathy based on case series and open-label studies (23–25). Their use remains limited to situations of treatment failure or intolerance to conventional immunosuppressive agents or in cases of relapse under maintenance therapy. In our patient, the favorable clinical and electrophysiological response to corticosteroids and conventional immunosuppression did not justify the use of biologic therapy.

Other therapeutic options have been reported more sporadically. Intravenous immunoglobulins may be considered in acute severe forms, particularly in the context of diagnostic uncertainty with Guillain–Barré syndrome or contraindications to immunosuppression, although their efficacy is often transient (26). Plasma exchange is reserved for exceptional situations characterized by rapidly progressive or life-threatening disease and does not represent a standard treatment for lupus-related peripheral neuropathy (27). Data regarding belimumab and other targeted biologic therapies remain insufficient to recommend their use in this specific indication (21,28).

The prognosis of lupus-related peripheral neuropathy depends primarily on initial severity, the extent of axonal damage, and the timeliness of therapeutic intervention. Severe axonal forms are associated with a higher risk of persistent deficits, whereas predominantly inflammatory forms may show substantial functional recovery with appropriate treatment. Long-term management relies on regular clinical and electrophysiological monitoring, combined with functional rehabilitation (2,6).

Several limitations should be acknowledged in the present case. First, no nerve biopsy was performed, and therefore a direct histopathological confirmation of lupus-related neuropathy could not be obtained. In addition, antiganglioside antibodies were not assessed. Consequently, the attribution of the neuropathy to systemic lupus erythematosus remains based on a combination of clinical, immunological, electrophysiological, and evolutionary arguments together with the exclusion of alternative causes, rather than on definitive proof.

4. CONCLUSION

This case highlights an early peripheral neurological involvement in systemic lupus erythematosus in a young patient, underscoring the marked clinical heterogeneity of the disease. Although uncommon, peripheral neuropathy may occur during the initial phases of lupus and represent a significant source of functional impairment. Attribution of the neuropathy to lupus relied on a rigorous diagnostic approach combining clinical, electrophysiological, and immunological data, with systematic exclusion of alternative causes. The favorable response to tailored immunosuppressive therapy further supports a disease-related mechanism. This observation emphasizes the importance of considering a lupus-related etiology in patients presenting with unexplained peripheral neuropathy associated with systemic features, and of initiating early, individualized management to optimize functional outcomes.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical information. The patient's identity has been kept confidential.

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