Case Report

"Polyradiculopathy" - A Rare Neurological Manifestation in Systemic Lupus Erythematosus

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ABSTRACT

The relationship of the neurological manifestations to the lupus disease process is not always clear. We present a case of systemic lupus erythematosus (SLE) with subacute onset quadriparesis, which was due to polyradiculopathy, a rarely described neurological manifestation of SLE.

Introduction -

Systemic lupus erythematosus (SLE) is an inflammatory, multisystem disease of unknown etiology with diverse clinical and laboratory manifestations. CNS manifestations are seen in 24-54% of cases[1]. The diagnosis of neuropsychiatric involvement in SLE has been difficult and their relationship to the lupus disease process is not always clear. Polyradiculopathy presenting primarily as muscle weakness has rarely been reported in patients suffering from SLE[2]. We report a case of SLE who presented with quadriparesis because of polyradiculopathy.

Case Report -

A 23 years old female presented with weakness in all four limbs since one and half month along with malaise & hyperpigmented rash all over her body of 15 days duration. On physical examination she had pallor, alopecia and edema feet. There was hyperpigmented macular rash all over her body, predominantly over the face. The musculoskeletal system examination showed waddling gait, inability to get up from squatting position. Her neurological examination revealed Proximal muscle weakness in both upper & lower limbs but normal muscle power in distally. (wrists and elbows, Ankles) There was no sensory impairement. All deep tendon reflexes were absent, & bilateral plantars were flexors.

Laboratory investigations revealed hemoglobin of 7gm%, WBC count 10,500 / cmm, and platelets count of 2.4 lakhs/cmm. Positive CRP and ESR of 40mm at the end of one hour. Qualitative urine analysis revealed presence of 3+ Protein. 24-hour urine protein was found to be 817mg%. Her blood urea was 67mgm% and serum creatinine was 2.2mgm%. Liver functions, ECG and X-ray Chest were within normal limits. USG Abdomen showed Grade-I Renal Parenchymal Disease with mild ascites. ANA and ds-DNA were positive. CSF examination revealed proteins 127.4 mg/dl, glucose 59 mg/dl and cells 02/mm3. NCV revealed reduced CMAP amplitude affecting lower limbs more than upper limbs and absent F-waves in lower limbs suggestive of Radiculopathy. ***(Table 1)*** A final diagnosis of Systemic Lupus Erythematosus with Lupus Nephritis with Pure Motor Quadriparesis "Polyradiculopathy" was kept.

The patient was put on with monthly pulse therapy of Cyclophosphamide and Methyl prednisone followed by Hydroxychloroquine and prednisolone. Patient showed improvement in two weeks and a complete response (normal muscle power and normal reflexes) was seen in 12 weeks time. The steroids were slowly tapered thereafter.

Discussion -

Proximal muscle weakness involving both upper and lower limbs in a patient of SLE can be of varied etiology like myositis, spinal cord involvement, peripheral neuropathy, or drug-induced myopathies due to steroids and chloroquine or overlap syndrome

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and anticardiolipin antibody syndrome. Overall, symptoms of muscle weakness can occur in 30-50% of patients suffering with SLE. Overt myositis can be present in 3-5% of SLE patients[3]. Our patient did not show any symptoms of muscle pain or tenderness. Subacute or chronic painless myopathy in patients with SLE can occur during treatment with chloroquine and steroid therapy. Chloroquine therapy can cause myopathy and a mild peripheral neuropathy. Although the myopathy is reversible when the drug is withdrawn, the recovery is slow. Peripheral neuropathy occurs in about 10% of cases of SLE, usually with established disease. The incidence of subclinical nerve disease is higher (21%) as suggested by abnormal NCV studies[4]. Three patterns of peripheral nerve involvement are usually recognized - symmetrical distal sensorimotor neuropathy; GullianBarre syndrome (GBS) and mononeuritis multiplex[5]. Anticardiolipin antibodies are associated with various neurological manifestations. Differentiation of GBS from chronically progressive symmetrical distal sensorimotor neuropathy (CIDP) is occasionally difficult. These cases, have a subacute onset and follow a chronic course, the so called CIDP. CIDP has been reported rarely in SLE. Rechtand et al described a report of two young women with progressive weakness, areflexia, elevated CSF proteins and slow nerve conduction velocity as the first manifestation of SLE[6]. Our patient did not have any sensory phenomenon like tingling, numbness and burning sensation or any objective sensory loss. The muscle weakness was also predominantly proximal, which is rarely seen in peripheral neuropathy. The presence of raised CSF proteins, NCV confirming radicular involvement, EMG evidence of denervation in same myotomes and suggests that the diagnosis is demyelinating polyradiculopathy. The patient was given pulse therapy of methylprednisolone with cyclophosphamide and subsequently put on prednisolone 1 mg/kg. Patient showed improvement in two weeks and a complete response (normal muscle power and normal reflexes) was seen in 12 weeks time. The steroids were later slowly withdrawn. This is an unusual case of subacute muscle weakness in a patient suffering from SLE, emphasizing that polyradiculopathy can be a cause of proximal muscle weakness in patients of SLE which can present early in the course of disease.

References :

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Table 1 : MNC Studies
Nerve : Median-Lt R-Site : APB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1. Wrist	3.50	13.25	9.75	3.62 mV	11.94 mVmS	APB-Wrist		3.50	
2. Elbow	7.75	21.50	13.75	6.63 mV	27.73 mVmS	Wrist-Elbow	210	4.25	49.41

Nerve : Median-Rt R-Site : APB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1. Wrist	3.75	12.00	8.25	4.14 mV	11.72 mVmS	APB-Wrist		3.75	
2. Elbow	8.25	18.00	9.75	4.26 mV	14.02 mVmS	Wrist-Elbow	220	4.50	48.89

Nerve : Peroneal-Lt R-Site : EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Ankle	3.88	11.25	7.38	0.30 mV	1.92 mVmS	EDB-Ankle		3.88	
2.Knee	11.38	20.38	9.00	0.70 mV	2.01 mVmS	Ankle-Knee	310	7.50	41.33

Nerve: Peroneal-Rt R-Site : EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Ankle	4.75	11.75	7.00	0.18 mV	0.94 mVmS	EDB-Ankle		4.75	
2.Knee	10.50	20.38	9.88	0.29 mV	1.03 mVmS	Ankle-Knee	280	5.75	48.70

Nerve : Tibial-Lt R-Site : EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Ankle	4.25	9.50	5.25	0.12 mV	0.56 mVmS	EHL-Ankle		4.25	
2.Popliteal Fossa	10.75	20.00	9.25	0.56 mV	1.74 mVmS	Ankle-Popliteal Fossa	300	6.50	46.15

Nerve : Tibial-Rt R-Site : EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Ankle	4.88	15.38	10.50	0.73 mV	2.64 mVmS	EHL-Ankle		4.88	
2.Popliteal Fossa	12.25	24.13	11.88	0.92 mV	3.76 mVmS	Ankle-Popliteal Fossa	290	7.38	39.32

Nerve: Ulnar-Lt R-Site: ADM

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Wrist	1.88	4.63	2.75	1.23 mV	2.45 mVmS	ADM-Wrist		1.88	
2.Elbow	6.00	9.00	3.00	1.10 mV	2.44 mVmS	Wrist-Elbow	230	4.13	55.76

Nerve : Ulnar-Rt R-Site : ADM

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Dur (mS)	Amp	Area	Segment	Dist. (mm)	Diff (mS)	CV (m/S)
1.Wrist	2.25	5.75	3.50	1.54 mV	2.48 mVmS	ADM-Wrist		2.25	
2.Elbow	6.13	10.75	4.63	1.43 mV	3.60 mVmS	Wrist-Elbow	220	3.88	56.77