Polyradiculopathy in a Patient of 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 muscle weakness, which was due to polyradiculopathy, a rarely described neurological manifestation of SLE.

INTRODUCTION
Systemic lupus erythematosus (SLE) is an inflammatory, multisystem disease of unknown aetiology with diverse clinical and laboratory manifestations. CNS manifestations are seen in 24-54% of cases. 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. We present a case of SLE from Nepal who developed muscle weakness because of polyradiculopathy. Incidentally this is also the first case report of SLE from Nepal.

CASE REPORT
A 21 years old female medical student presented with two months history of painful swelling of joints and low grade fever of two weeks duration. She also had malar rash, alopecia and malaise. A diagnosis of SLE was made after investigations. These were Hb 12.2%, total leucocyte count 7200/mm³, ESR 68 mm/hr (Westergreen), ANA positive (ELISA) and ant-dsDNA positive. Her urine examination, renal function tests and liver function tests were within normal limits. She was treated with chloroquine daily and NSAIDs. She had good improvement with this treatment. After four months she developed generalized bodyache and proximal muscle weakness of the lower limbs. The musculoskeletal system examination showed waddling gait, inability to get up from squatting position, normal muscle power in upper limbs, grade IV muscle power at hips and knees and normal muscle power at ankles. Knee reflexes were sluggish while both ankle jerks were absent. Results of her investigations were hemoglobin 10.0 gm/dl, total leucocyte count 7700/mm³, platelets 175,000/mm³ and ESR 78 mm. in 1st hour (Westergreen). Urine examination, renal function tests and liver function tests were within normal limits. Her CPK was 60 IU (Normal 25-165 IU/dl), ANA was positive with rim pattern at 1:160 dilution and anti-dsDNA antibody level of 6.7 IU/ml. Her anticardiolipin antibody was 4.5 GPL (normal < 10 GPL). Complement levels were C3 64.8 mg/dl and C4 11.3 mg/dl. CSF examination revealed proteins 73.4 mg/dl, glucose 59 mg/dl and cells 20/mm³ with 96% lymphocytes. NCV (Nerve conduction velocity) showed radicular involvement at the roots L4-5 and S1. Electromyography findings were suggestive of acute denervation in L4-5, S1 myotomes and neurogenic changes in L2-3 myotomes. MRI scan revealed swelling of nerve roots L3-5. The final diagnosis was polyradiculopathy involving L3-5, S1 nerve roots.

DISCUSSION
Proximal muscle weakness involving lower limbs in a patient of SLE can be of varied aetiology like myositis, spinal cord involvement, overlap syndrome, peripheral neuropathy, anticardiolipin syndrome or drug-induced myopathies due to steroids and chloroquine. Overall, symptoms of muscle involvement can occur in 30-50% of patients suffering with SLE. Overt myositis can be present in 3-5% of SLE patients. Our patient did not show any symptoms of muscle pain or tenderness. There were no signs of active disease as is indicated by normal ESR, CRP, anti-dsDNA antibody and complement levels.

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. Initially the diagnosis of chloroquine-induced myopathy was considered in this case but there was no response to...
chloroquine withdrawal.

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 and determination of vibration threshold. Three patterns of peripheral nerve involvement are usually recognized - symmetrical distal sensorimotor neuropathy, Gullian Barre syndrome (GBS) and mononeuritis multiplex. 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.

Our patient did not have any sensory phenomenon like tingling, numbness and burning sensation or any objective sensory loss. The muscle weakness was also predominately proximal, which is rarely seen in peripheral neuropathy. The presence of raised CSF proteins, NCV confirming radicular involvement at roots L4, S1, EMG evidence of denervation in same myotomes and swelling of nerve roots on MR imaging suggests that the diagnosis is chronic demyelinating polyradiculopathy.

The patient was started 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 slowly withdrawn over next 10 months.

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