Motor Radiculopathy — An Unusual Presentation of Multiple Sclerosis

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SUMMARY: *A 28 year old female presented with clinical and electrical evidence indicative of L5 motor radiculopathy. Subsequently she was found to have symptoms and signs of multiple sclerosis. When clinical assessment and investigation fail to disclose a cause then demyelinating disease should be considered in the differential diagnosis of motor radiculopathy.*

RÉSUMÉ: *Nous rapportons le cas d'un femme de 28 ans qui présentait les signes cliniques et électriques d'une radiculopathie en L_5 mais qui, subséquemment s'avéra souffrir d'une sclérose en plaques certaine. Il faut donc savoir considérer la possibilité d'une maladie démyélinisante comme diagnostic différentiel lorsqu'une radiculopathie motrice ne trouve pas de cause.*

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INTRODUCTION

When weakness is the initial symptom of multiple sclerosis it is almost invariably of an upper motor neuron type. We report a patient, however, who presented with motor radiculopathy who subsequently developed signs and symptoms of multiple sclerosis.

CASE REPORT

A 28 year old white female presented in February, 1979 with the acute onset of weakness about the right ankle. She had noted difficulty with dorsiflexion and a tendency to invert the ankle spontaneously. She also noted an increased sensitivity to light touch over the dorsum of the foot.

Review of her past health revealed that she had had one normal pregnancy and cone biopsy therapy for stage 0 carcinoma of the cervix six months previously.

Examination revealed grade 1/5 strength of dorsiflexion, inversion and eversion of the right ankle. There was trivial weakness of the plantar flexors of the right ankle with grade 4/5 strength of the right hamstring and gluteus medius groups. Bulk and tone were normal. The right hamstring reflex was diminished. The remainder of the deep tendon reflexes were normal and both plantar responses were flexor. Straight leg raising was unremarkable to 90 degrees bilaterally. Sensory examination was normal. She walked with a drop-foot gait. A small deeply pigmented birthmark and tuft of hair were present in the midline over the lower lumbar spine with minimal tenderness at this site. A detailed general and neurological examination was otherwise normal.

Laboratory studies on admission revealed a hemoglobin of 14.9 gm%; WBC 8,700 with a normal differential; erythrocyte sedimentation rate (ESR) was 7 mm/hr. Routine biochemistry, serology, and urinalysis were negative. Radiographs of the chest, lumbosacral spine and cone views of L5 and S1 were normal.

Nerve conduction study of the right peroneal nerve revealed normal motor parameters (conduction velocity 44.5 meters per second, distal motor latency 5.2 milliseconds and amplitude 3 milliVolts). Needle examination (table 1) demonstrated, in the pattern as outlined, evidence of abnormal spontaneous activity (+) manifested as fibrillation potentials and/or positive waves signifying the presence of denervation and abnormalities of number, shape, and rate of firing of the motor unit potentials indicative of neurogenic impairment. The alterations were detected in those muscles supplied predominantly by L5 (Heffernan, 1979). It was not possible to study the paraspinal musculature adequately due to significant patient distress.

A pantopaque lumbar myelogram was normal but as no satisfactory views of the conus medullaris were obtained a metrizamide myelogram including tomography of the conus was performed and was normal. The CSF protein was 47 mg%, sugar 60 mg%, VDRL nonreactive, WBC count 4. A protein electrophoresis was not performed. A Gallium scan of the chest, abdomen and pelvis and a computerized body scan were normal. The patient was discharged.

When seen two months later she was recovering from a three week episode of paresthesias involving the left arm distal to the elbow. She had difficulty

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with the use of the left arm describing it as feeling "heavy, stiff, clumsy and frozen". She then recalled that for several months she had been aware of "instantaneous shock-like sensations" spreading down her back to her buttocks and occasionally into her legs on neck flexion. Similarly she remembered that six months prior to the onset of weakness she had been aware of hypersensitivity to light touch over the L5 distribution of both legs which had subsided spontaneously after two months.

Examination revealed unchanged findings in the L5 distribution on the right. The deep tendon reflexes, however, were quite brisk particularly on the left side. Testing of the biceps and brachioradialis reflexes induced marked distal finger flexion suggesting pathological spreading of the reflex arc. The legs demonstrated bilateral crossed adductor responses. There was a left Babinski sign and an equivocal extensor toe sign on the right. There was diminution of vibratory sensation in the left fingers and toes. Position sense was minimally disturbed in the toes, left greater than right. Sudden full flexion of the head and neck evoked a classical L'Hermitte's phenomenon.

She was seen again five months later because of the sudden onset of similar sensory dysfunction involving, once again, the left upper extremity. Examination demonstrated a qualitative impairment of touch sensation, i.e., objects felt different, however once the threshold to stimulation was exceeded the resulting sensation was most unpleasant. There was also a return of strength about the right ankle so that she was experiencing no functional disability whatsoever. Formal testing demonstrated, however, minimal residual weakness in the previously detected distribution. The examination otherwise demonstrated no alterations from the previous assessment.

DISCUSSION

The clinical detection of weakness of inversion placed the site of dysfunction outside of the territory of the peripheral peroneal nerve, peroneal palsy being the usual cause of weakness solely of dorsiflexion and eversion. Weakness of all three movements suggested impairment of a shared innervation which must be located proximally, i.e., radicular and not distal, in view of the sparing of the plantar flexor movement. The pattern of clinical weakness was indicative of L5 dysfunction. Denervation (abnormal spontaneous activity manifested by positive waves and fibrillation potentials) and neurogenic alterations of the motor units result from a loss of axonal continuity. The presence of these changes in muscles outside the territory of a single peripheral nerve most often indicates a shared proximal radicular innervation. The changes detected in this patient, on the basis of previous work from this laboratory (Heffernan, 1979), indicated L5 to be the damaged radicle, confirming the clinical impression.

As no appropriate lesion could be demonstrated the patient was treated conservatively.

Subsequently she developed symptoms of cervical dorsal column dysfunction. Examination at that time revealed bilateral upper motor neuron findings and dorsal column impairment. A diagnosis of clinically definite multiple sclerosis was then made (Rose et al, 1976).

Motor weakness of an upper motor neuron type is a common initial symptom of multiple sclerosis. Weakness with reflex diminution or loss is usually accompanied by an associated sensory disturbance suggesting a lesion in the region of the corticospinal tract spreading to the posterior horn or posterior root entry zone. These findings are more common with lesions in the cervical rather than the lumbar cord (McAlpine et al, 1972).

Lower motor neuron weakness often accompanied by muscular atrophy has been well recognized in the chronic phase of this illness (McAlpine et al, 1972). However, this type of weakness is an unusual mode of presentation of multiple sclerosis. Such involvement indicates the presence of a plaque either at the intramedullary anterior root level where "peripheral" motor fibers are enveloped in "central myelin" prior to their exit from the spinal cord or invasion directly of anterior horn cells. Lesions at these sites have been well documented pathologically (Oppenheim, 1911; Davison et al, 1934). It is our contention that a lesion on the right side at the level of the fifth lumbar segment of the cord would explain the lower motor neurone component of this patient's illness.

The possibility exists that the radiculopathy is the result of an entirely separate undetermined disorder. However, no additional evidence has developed to support this contention and extensive investigation has failed to reveal the presence of such a disorder. This supposition would also necessitate making two distinct diagnoses as there appears to be little doubt of the presence of multiple sclerosis.

TABLE 1

Needle Electrode Assessment

Muscle	*Abnormal Spontaneous Activity*	*Motor unit Potentials*
R T. Anterior	+	Neurogenic
R T. Posterior	+	Neurogenic
R Peroneal Group	+	Neurogenic
R M. Gastrocnemius	Increased Irritability	Normal
R L. Gastrocnemius	Increased Irritability	Normal
R V. Lateralis	0	Normal

(+ = Positive Waves &/or Fibrillation Potentials)

Considering the temporal sequence of events it seems more reasonable to invoke the same diagnosis to explain the entire presentation. The subsequent, albeit not complete, improvement of the motor weakness about the right ankle is consistent with the known pathophysiological evolution of multiple sclerosis. Some weakness has persisted which could be explained on the basis that the process was not solely demyelinating suggesting a degree of partial axonal degeneration had occurred.

Electrophoresis of spinal fluid was not done as the diagnosis of multiple sclerosis was not entertained at that time. Though the determination of an elevation of IgG remains the mainstay of cerebrospinal fluid diagnosis in this disorder its overall accuracy in the routine clinical laboratory is only approximately 50% (Poser, 1979).

This case is presented to illustrate the occasional occurrence of motor radiculopathy in multiple sclerosis. Most frequently this occurs late in the illness, serving principally to localize the degree of spinal involvement (McAlpine et al, 1972). On rare occasions, however, a motor radiculopathy may be a presenting feature of this disease.

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