Axonal multifocal motor neuropathy without conduction block or other features of demyelination

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Abstract—Background: Conduction block is considered an essential finding for the distinction between motor neuropathies and lower motor neuron disorders. Only a small number of reports describe patients with multifocal motor neuropathies who lack overt conduction block, although in these cases other features of demyelination still suggest the presence of a demyelinating disorder. In contrast, a purely axonal multifocal motor neuropathy has not been described. Methods: This report describes nine patients with slowly or nonprogressive multifocal motor neuropathies who had purely axonal electrophysiologic features. Results: GM1 antibodies titers were normal in all nine cases. Six patients were treated with either prednisone or IV immunoglobulin and three showed convincing improvement. Conclusions: These findings suggest an immune-mediated motor neuropathy with axonal electrophysiologic features that appears to be distinct from both multifocal motor neuropathy and established motor neuron disorders.

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The demonstration of conduction block in at least one motor nerve has long been considered the essential finding for the distinction of motor neuropathy from lower motor neuron disorders. However, over-reliance on strict conduction block criteria can result in underrecognition of potentially treatable motor neuropathies. Patients with multifocal motor neuropathy (MMN) who do not meet electrophysiologic criteria for conduction block still show other features of a demyelinating neuropathy, including focal temporal dispersion, small-amplitude drops between proximal and distal stimulation sites that do not meet subjectively defined limits, prolonged distal latencies, and focal conduction slowing. As opposed to true conduction block, these other demyelinating abnormalities are not likely to explain the presence of weakness but can still be useful in distinguishing MMN from motor neuron disorders. Patients lacking overt conduction block may have elevated titers of anti-GM1 antibodies and respond to IV immunoglobulin (IVIg) with frequencies similar to MMN with conduction block.

Although earlier reports have established the concept of MMN without overt conduction block, none have clearly demonstrated a chronic motor neuropathy without conduction block or any other electrophysiologic features of demyelination. In this report we describe our experience with patients with a slowly or nonprogressive multifocal motor phenotype who had purely axonal electrophysiologic findings. We refer to this syndrome as multifocal acquired motor axonopathy (MAMA) and attempt to distinguish it both from MMN and from common motor neuron disorders that may have similar clinical and electrophysiologic features.

Methods. Patients. We included patients seen during 1999 and 2000 in our neuromuscular clinics who met combined clinical and electrophysiologic criteria. These patients had evidence of a slowly progressive or nonprogressive, asymmetric, purely motor neuropathy. At the time of the initial examination, weakness had to be multifocal with preferential involvement of individual peripheral nerves by clinical examination. All patients were followed up for at least 2 years and had documentation that weakness remained stable over that time, or if there was progression or spread to new areas, the initial multifocal pattern of weakness had to remain evident throughout follow-up or patients had to demonstrate regression of weakness with treatment. Patients who showed bulbar signs or definite upper motor neuron involvement (spasticity, hyperreflexia, extensor plantar response) at any time were excluded.

Motor and sensory nerve conduction studies were performed in each patient. At a minimum, two limbs were evaluated. Studies included median, ulnar, radial, peroneal, and tibial motor responses and median, ulnar, superficial radial, and sural sensory responses, as indicated for each individual. Motor nerve conduction studies were recorded with surface electrodes as follows: the median nerve
was stimulated at the wrist, antecubital fossa, axilla, and Erb’s point recording over the abductor pollicis brevis; the ulnar nerve was stimulated at the wrist, below the elbow, above the elbow, axilla, and Erb’s point, recording over the abductor digiti minimi; the radial nerve was stimulated proximal to the lateral epicondyle, in the axilla, and at Erb’s point, recording over the extensor digitorum communis; the peroneal nerve was stimulated at the ankle, fibular head, and popliteal fossa, recording over the extensor digitorum brevis; and the tibial nerve was stimulated at the ankle and popliteal fossa, recording over the abductor hallucis. Measurements included distal latency, negative peak duration, baseline-to-peak amplitude, and conduction velocity. F-wave latencies were recorded in the median, ulnar, peroneal, and tibial distributions.

For the purpose of this study, we used strict electrodiagnostic criteria for a purely axonal polyneuropathy. Electromyography had to demonstrate motor unit potentials or spontaneous activity suggestive of a neuropathic process. Patients were excluded if they met even minimal criteria for conduction block or abnormal focal temporal dispersion. We defined conduction block as any reduction of amplitude or area of >30% across a standard segment, regardless of the change in waveform duration. Abnormal temporal dispersion was defined as the duration of the waveform increasing >30% across a standard segment. In addition, all patients had conduction velocities, distal latencies, and F-wave latencies that were within normal limits for all segments tested, with the exception of a single patient whose conduction velocity of 46 m/s (normal is >48 m/s) across the forearm in one median nerve, although his remaining study showed no conduction slowing or latency abnormalities in any segment.

A commercial laboratory performed testing for anti-GM1 antibodies (Athena Diagnostics, Worcester, MA). The standard assay was used, as opposed to the more recently available Co-GM1 (Athena) technique. Other laboratory tests were ordered at the discretion of the attending physician and were reviewed in our analysis. All nine patients were offered treatment with either IVIg or prednisone but only six received therapy.

Illustrative case (Patient 8). A 20-year-old right-handed man was referred for evaluation of weakness of 5 years’ duration in the hands and feet. Weakness began in the right hand and was followed within weeks by a milder degree of weakness in the left hand that has remained asymmetric over time. Two years later, the weakness progressed to the left foot and then to the right foot. He denied any sensory symptoms. Nerve conduction studies and CSF examination were normal. Nevertheless, chronic inflammatory demyelinating polyneuropathy was diagnosed (CIDP) and the patient was treated with prednisone, 80 mg daily. His strength returned to normal after about 2 months, and prednisone was slowly tapered. However, he seemed to relapse every time his dose was decreased to 30 mg per day.

At his first visit, he had worsening strength in both hands, right worse than left, and continued distal weakness in the lower extremities. On neurologic examination he had normal muscle bulk and tone. Muscle strength testing revealed the following Medical Research Council scores: orbicularis oculi 5, neck flexion and extension 5, shoulder abduction and elbow extension 5, elbow flexion 4 on the right and 4+ on the left, wrist extension 4 on the right and 3− on the left, wrist flexion 4 on the right and 4+ on the left, finger extension zero on the right and 2 on the left, finger flexion 5 bilaterally, interossei 4 on the right and 4+ on the left, abductor pollicis brevis 3− on the right and 3 on the left, and abductor pollicis longus 0 on the right and 3 on the left. In the lower extremities, hip flexion, abduction, and extension, knee extension, and knee flexion were 5, ankle dorsiflexion was 3−, and plantar flexion was 5. Sensory examination revealed intact light touch, vibration, pinprick, and proprioception. Deep tendon reflexes were 2+ in the biceps, triceps, and brachioradialis, and 2 at the knees and ankles. Plantar responses were flexor.

Anti-GM1 antibodies were absent in the serum. Motor conduction studies (right median, ulnar, peroneal, and posterior tibial) revealed slightly reduced compound muscle action potential (CMAP) amplitudes in the median (3 mV) and peroneal nerves (0.9 mV). The tibial and ulnar motor amplitudes were normal. There was no evidence of conduction block or abnormal temporal dispersion. Conduction velocities, distal latencies, and F-waves were entirely within normal limits. Median and ulnar sensory nerve conduction studies of the right arm were normal. Electromyography demonstrated 1 to 2+ fibrillation potentials and decreased recruitment of long-duration, high-amplitude motor units in the biceps brachii, extensor digitorum communis, first dorsal interosseus, tibialis anterior, and gastrocnemius consistent with active denervation and chronic reinnervation.

We increased his prednisone dosage from 30 to 100 mg daily. After 2 months, he had subjective and objective improvement in his distal arm and leg strength: finger and wrist extension 3− on the right, 4− on the left; finger extension 2 bilaterally, interossei 4+ bilaterally, abductor pollicis brevis 4− bilaterally; abductor pollicis longus 3− on the right, 3 on the left, and 4− in ankle dorsiflexion bilaterally. Maintaining prednisone at a dose of 100 mg every other day for 6 months did not lead to further improvement and no benefit was noted with the addition of azathioprine. Therefore, he received IVIg 2 g/kg and then 0.4 g/kg every month for 5 more months. He noted significant improvement in strength and function in his hands beginning after the first infusion. He denied any functional limitation, although he still had mild objective weakness in his hands.

Results. Nine patients were identified fulfilling inclusion criteria, including five men and four women (table). The average age at onset was 39 years (range, 15 to 65 years) and the mean duration of illness was 7.3 years (range, 3 to 20 years). The weakness began in a hand in six patients and simultaneously in a hand and ankle in two. One patient first noticed weakness in the left shoulder. As the condition progressed, weakness remained limited to the upper limbs in six patients and spread to involve both the upper and lower limbs in three. Weakness eventually spread to more than one limb in seven patients. In each case, the progression of weakness at the time of onset was relatively insidious with focal deficits that evolved over weeks to months in one or more nerve distributions. This was followed by periods of stability ranging from months to a few years before other distributions became affected.

Multifocal weakness and atrophy were characteristic on
examination. The degree of weakness appeared to exceed the degree of atrophy in two cases. Fasciculations were present in six patients but were never prominent. The median, ulnar, or radial nerve distributions of distal upper limbs were affected with approximately equal frequencies. Involvement of the tibial or peroneal distributions was less common. Weakness at the elbows, shoulders, or hips was evident in four patients. In two of these, there was diffuse but asymmetric weakness affecting all four limbs. In the other two patients, the proximal weakness had a focal pattern, affecting the axillary nerve distribution in one and proximal radial nerve distribution in the other. At times, weakness was limited to only partial nerve distributions such as the posterior aspect of the deltoid muscle, sparing the anterior portion or the dorsal interosseous muscles, sparing the abductor digitii minimi.

In each of the nine cases, the electrodiagnostic studies suggested a relatively indolent axonal process. Nerve conduction studies generally revealed low-amplitude CMAP in muscles with clinical weakness, although in a few instances the distal CMAP amplitudes were larger than would be expected for the degree of weakness. On electrodiagnostic testing, fast-firing, neuropathic motor unit potentials were present in clinically affected muscles, and at times these abnormalities were seen in muscles without obvious clinical involvement. Fibrillation potentials and positive sharp waves were low grade or absent.

### Table Clinical and diagnostic findings

<table>
<thead>
<tr>
<th>Subject no./age at onset, y/sex</th>
<th>Duration, y</th>
<th>Onset distribution</th>
<th>Maximum involvement*</th>
<th>Studies†</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/30/M</td>
<td>7</td>
<td>R hand</td>
<td>R median; R ulnar; L median; L ulnar</td>
<td>Gene for SMA; paraspinous EMG: C, Th</td>
<td>None</td>
</tr>
<tr>
<td>2/23/M</td>
<td>4</td>
<td>L posterior deltoid muscle</td>
<td>R radial (including triceps); L radial; R median; L median; L axillary nerve (affecting only posterior portion of the deltoid muscle)</td>
<td>Gene for SMA; paraspinous EMG: C</td>
<td>IVIg</td>
</tr>
<tr>
<td>3/24/F</td>
<td>19</td>
<td>R radial</td>
<td>R radial; R median; L median; R peroneal</td>
<td>Gene for HNPP/CMT; paraspinous EMG: C, Th; spinal fluid analysis; superficial radial nerve biopsy</td>
<td>None</td>
</tr>
<tr>
<td>4/42/F</td>
<td>10</td>
<td>L ulnar</td>
<td>L ulnar; L median; L superficial peroneal; L tibial</td>
<td>Genes for HNPP/CMT, SMA; paraspinous EMG: C; spinal fluid analysis</td>
<td>IVIg</td>
</tr>
<tr>
<td>5/64/F</td>
<td>3</td>
<td>L ulnar, L peroneal</td>
<td>Diffuse asymmetric pattern with bilateral asymmetric weakness affecting shoulders, upper arms and hands; the L median and radial distributions were most severely involved; asymmetric distal greater than proximal weakness in the lower limbs, maximal in the R peroneal distribution</td>
<td>Paraspinuous EMG: C, Th, L; spinal fluid analysis</td>
<td>IVIg‡</td>
</tr>
<tr>
<td>6/47/M</td>
<td>12</td>
<td>R ulnar; R median</td>
<td>R ulnar; R median</td>
<td>Paraspinuous EMG: C; gene for HNPP/CMT; Spinal fluid protein of 78 mg/dL</td>
<td>IVIg</td>
</tr>
<tr>
<td>7/32/M</td>
<td>8</td>
<td>R radial</td>
<td>R radial; R median (involving wrist flexor and pronator teres)</td>
<td>Paraspinuous EMG: C (1+ fibs), Th</td>
<td>IVIg‡</td>
</tr>
<tr>
<td>8/15/M</td>
<td>5</td>
<td>Hands R &gt; L</td>
<td>Diffuse asymmetric pattern affecting elbows, wrists, fingers, and ankles; the R radial distribution (including triceps) was most involved</td>
<td>None</td>
<td>Prednisone† Azathioprine IVIg‡</td>
</tr>
<tr>
<td>9/16/M</td>
<td>3</td>
<td>R radial</td>
<td>R radial; L peroneal; R peroneal; L radial</td>
<td>Paraspinuous EMG: C, L</td>
<td>None</td>
</tr>
</tbody>
</table>

Nerve distributions with most severe involvement are in bold.

* Weakness was limited to hands, wrists, and feet unless specifically mentioned.
† Normal unless in bold.
‡ Treatment associated with favorable therapeutic response.

SMA = spinal muscular atrophy; EMG = electromyography; C = cervical; Th = thoracic; HNPP = hereditary neuropathy with susceptibility to pressure palsies; CMT = Charcot–Marie–Tooth type 1A; L = lumbar; IVIg = IV immunoglobulin.
tomal levels of the weak muscles were normal in seven patients. Thoracic paraspinal muscles were normal in all four patients tested. One patient (Patient 7) had fibrillation potentials in the cervical paraspinal region but had normal thoracic paraspinal study findings and responded to therapy. Paraspinal muscles were not studied in Patient 8 (illustrated case), in whom there was clinical improvement with immune-modulating therapies, negating motor neuron disease as a possible diagnosis.

Anti-GM1 antibody test results were normal in all nine patients. Serum protein electrophoresis was also normal in each case. Creatine kinase levels were normal in eight patients and mildly elevated in one. Three patients had deletion analysis for hereditary neuropathy with susceptibility to pressure palsies and three patients underwent testing for the survival motor neuron gene deletions. These findings were normal. Spinal fluid analysis was unremarkable in the three subjects and there was a mildly elevated protein level in the fourth who was tested. One patient underwent sensory nerve biopsy, and findings were unremarkable. All nine patients had normal MRI findings for the cervical spine.

Three patients showed convincing improvement of at least one Medical Research Council grade with immune-modulating therapy. One patient (illustrated case) initially improved within 2 months of starting high-dose prednisone. However, he had relapses when the drug was tapered and only improved partially as prednisone was increased again. This patient and two others treated with IVIg improved. In all three, the response to IVIg became evident within 2 weeks of completing the initial infusion of 2 g/kg. Two patients maintained the therapeutic response with additional monthly infusions of 0.4 g/kg. One was wheelchair dependent at presentation and regained the ability to walk independently by 4 months (Patient 5). The other regained the use of his hands (illustrated case). Of note, these were the only two patients in our report with the relatively diffuse asymmetric pattern of weakness. The third patient who improved (Patient 7) received IVIg 4 years after the onset of symptoms. At first, he did not receive any additional infusions because of a shortage of IVIg, and his weakness returned to its pretreatment baseline. When he received IVIg, 2 g/kg, for a second time almost a year later, there was no improvement and he declined further treatment. His condition has remained stable without therapy for the past 3 years. Three patients did not show any response to a single infusion of IVIg, 2 g/kg. These three had a longer duration of weakness prior to the initiation of treatment (mean, 8.7 years; range, 4 to 12 years) compared with the responsive patients (mean, 5.3 years; range, 3 to 8 years). Three other patients declined therapy.

**Discussion.** Our findings allow the concept of chronic acquired multifocal motor polyneuropathy to be expanded to include a subgroup of patients who appear to have purely axonal electrodiagnostic features, thus blurring the lines distinguishing motor neuropathies from neuronopathies. Earlier studies have discussed patients with the phenotype of MMN who lack overt conduction block, but these cases usually have demyelinating findings other than conduction block. These reports have also pointed toward the presence of anti-GM1 antibodies or a response to immune-modulating therapies as additional evidence for an immune-mediated neuropathy. In three of our patients, only the response to immune-modulating therapy suggested an immune-mediated neuropathy was present. However, in the other six, none of these features was present and we can only infer the localization to peripheral nerves and the pathogenic basis. Although we cannot be certain that all nine patients have a single pathophysiologic entity, we include them together because the phenotype of slowly progressive multifocal involvement served as our guideline for recommending therapy. We did not separate responders from nonresponders given that even in classic MMN with conduction block, IVIg is not effective in up to one-third of cases. Moreover, from a practical perspective, the long periods of nonprogression necessitate that our nonresponders be differentiated from patients with ominous forms of motor neuron disease that have similar electrophysiologic findings, in which progression with time is a mandatory diagnostic feature.

Several findings suggest that MAMA may be distinct from MMN, despite the clinical parallels. For example, assuming a 30 to 50% sensitivity of standard anti-GM1 antibody testing in MMN with or without conduction block, the probability of finding no positive samples in nine consecutive tests would fall within a range of between only 0.01 to 3%. Other observations that were relatively atypical of MMN included the three patients who were <20 years of age at disease onset and the single patient who improved with prednisone. Our series also differs from other reports on potentially treatable lower motor neuron syndromes that lack conduction block. One group described a syndrome marked by the presence of “distal greater than proximal weakness and no conduction block,” although detailed electrophysiologic data were not supplied. Anti-GM1 antibodies were present in more than half. Another recent report described 10 patients with an “asymmetric, progressive lower motor neuron disorder” who were prospectively treated with IVIg. Although four patients improved and only two had GM1 antibodies, the methods for patient selection had important differences from ours. The authors did not perform comprehensive testing to rule out other demyelinating features and did not use temporal criteria to ensure a lack of progression over time. In several of these patients ALS was later diagnosed.

Earlier authors proposed that the electrodiagnostic appearance of “MMN without overt conduction block” results from “hard to find” demyelinating lesions in proximal locations that are not easily accessible to basic electrodiagnostic testing or from secondary axonal degeneration that masks the primary demyelinating process. We cannot rule out that the use of root stimulation in our patients may have revealed conduction block localized only to the spinal roots or proximal nerve segments. However, because weakness often affected multiple nerve dis-
tributions, we would have to assume that the primary demyelinating process occurred only at the roots and spared all portions of nerve that were more easily accessible to testing. The focal pattern of weakness, the presence of normal F-waves, and the CSF protein levels further suggested that this localization was unlikely.

Two of our three patients who responded to immune-modulating therapy had a tendency toward diffuse, generalized weakness as compared with the remaining patients who had more clearly defined multifocal patterns. This diffuse motor pattern is commonly associated with typical CIDP. An earlier series described a steroid-responsive axonal variant of CIDP, although those cases did not have the pure motor features, asymmetry, and normal CSF protein levels seen in our patients. Still, taken together that study and ours argue for a spectrum of immune-mediated peripheral neuropathies with purely axonal electrophysiologic features. The relationship between chronic MMN and MAMA or between axonal and demyelinating forms of CIDP appears analogous to that in the generalized acute inflammatory neuropathies, where Guillain–Barré syndrome and acute motor axonal neuropathy share clinical features but differ by electrodiagnostic findings. There was skepticism about the earlier report of “axonal” CIDP, partly because, like our cases, there was a rapid response to immune therapy. This observation was thought to imply that the underlying disorder was more likely to be one of easily reversible primary demyelination, despite the electrodiagnostic appearance. Although we do not know the underlying physiologic process in our patients, MAMA could also be explained by a relatively indolent and persistent immune attack directed against distal motor nerve terminals and leading to distal conduction block or axonal degeneration, analogous to that described in acute motor axonal neuropathy. Note that in acute motor axonal neuropathy, recovery is often similar to recovery from typical Guillain–Barré syndrome despite the absence of pathologic demyelination. This has been attributed to rapid collateral sprouting near the motor end plate during the recovery phase. Patients with MAMA who have longer disease duration or more severe axonal loss might be less likely to respond, similar to that described in patients with MMN with conduction block. MAMA should be included in the differential diagnosis of motor neuropathies and neuronopathies that present with asymmetric weakness and axonal electrophysiologic features. Progressive muscular atrophy is probably the most common disorder in this category, as estimates suggest that up to one-third of patients with sporadic motor neuron disease lack upper motor neuron signs at the time of presentation. Although the frequency depends on clinical definitions and the interpretation of examination findings, progressive muscular atrophy cases ultimately behave similarly to ALS, with evolution to generalized weakness and widespread denervation. One notable exception is brachial amyotrophic diplegia, in which signs of motor neuron disease can remain isolated to the arms for many years. These patients typically develop flail arms with relatively symmetric proximal and distal arm weakness. This is a distinctly different phenotype from MAMA. Finally, MAMA also shares features with juvenile muscular atrophy of the upper extremities. Patients with juvenile muscular atrophy develop characteristic unilateral or bilateral weakness and wasting limited to the intrinsic hand muscles and the ulnar side of the forearm that remain static after a few years of progression. The majority present between the ages of 18 and 22 years, and onset past age 30 years is rare. Only three of our patients had weakness limited to the hands, although not clearly in a distribution typical of juvenile muscular atrophy. These patients were also between the ages of 30 and 47 years at onset and one responded to IVIg.

References

Ptosis in patients with hemispheric strokes

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Abstract—Background: Cerebral ptosis is considered rare; it has been reported with unilateral, usually right hemispheric lesions. However, the frequency of ptosis in patients with strokes has not received systematic study. Objectives: To determine the frequency of ptosis in patients with acute hemispheric stroke and to identify stroke features associated with ptosis. Methods: Eyelid function was studied in 64 consecutive patients with acute hemispheric stroke and 40 age-matched subjects with no known neurologic disease. All underwent comprehensive neuro-ophthalmologic and general neurologic examination within 48 hours of admission, including measurement of palpebral fissures, marginal reflex distance, and range of upper lid movement. Only patients who could cooperate with eyelid testing were included. Brain CT scans were obtained for all patients who had had strokes. Results: Twenty-four (37.5%) of the patients with strokes had neurogenic ptosis, which was bilateral in 10 and unilateral in 14. None of the control subjects had neurogenic ptosis. All patients with strokes with ptosis had a hemiparesis. Rightward gaze deviation and upgaze paresis were more common (p < 0.05) in the patients with ptosis compared with others who had had strokes. CT evidence of right-sided hemispheric cortical infarction was more common in patients with strokes with ptosis (p < 0.05). In five patients with large hemispheric infarction, complete bilateral or asymmetric ptosis was the first sign of imminent herniation, preceding papillary dilation and ocular motor deficits. Conclusions: Ptosis occurs frequently in patients with hemispheric strokes, especially in association with right hemispheric lesions. Complete bilateral ptosis is usually caused by large infarctions and may be a premonitory sign of an impending herniation.

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The term cerebral ptosis refers to droopy eyelids in association with lesions of the cerebral hemispheres. It has been described with unilateral, usually right, hemispheric lesions.¹,²,⁵ Cerebral ptosis (or blepharoptosis) is considered rare.³ Its frequency, however, may be underestimated because inability to open the eyes in patients with strokes can be mistakenly ascribed to drowsiness. The aims of the present study were to determine the frequency of ptosis in patients with acute hemispheric ischemic stroke, to identify associated clinical findings, and to correlate the type of ptosis with the site of infarction.

Methods. Consecutive patients who were admitted to the Rabin Medical Center, Golda Campus, with the diagnosis of acute stroke during an 8-month period were examined by a neurologist within 48 hours of admission. All underwent brain imaging within 72 hours. Patients with...
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