Human Immunodeficiency Virus–Associated Pure Motor Lumbosacral Polyradiculopathy

Michael G. Benatar, MBChB, DPhil; Roland W. Eastman, MBChB, FRCP

Background: Neuromuscular disease is a common manifestation of human immunodeficiency virus infection and acquired immunodeficiency syndrome, but isolated and severe pathology confined to the motor roots or anterior horn cells are not a recognized clinical entity.

Objective: To describe the novel clinical presentation of human immunodeficiency virus–related polyradiculopathy manifesting as isolated severe motor symptoms confined to the legs.

Design: A case series comprising 4 patients identified prospectively during a 6-month period.

Setting: Patients were seen in the Department of Neurology, Groote Schuur Hospital, Cape Town, South Africa. This is an 800-bed teaching hospital, with approximately 5000 patients seen annually in the Department of Neurology.

Patients: Patients were identified by their unique presentation with a severe isolated motor neuropathy in the lower limbs. All were Xhosa-speaking African women.

Result: Early human immunodeficiency virus infection may be associated with pure motor lumbosacral polyradiculopathy.

Conclusion: It remains unclear whether this clinical syndrome should be regarded as a variant of the Guillain-Barré syndrome or whether it represents a unique disorder associated with early human immunodeficiency virus infection.

Arch Neurol. 2000;57:1034-1039

In recent years, there has been a dramatic shift in our understanding of the Guillain-Barré syndrome (GBS) at clinical, electrophysiologic, immunological, and pathological levels. Most notable has been the shift from the conceptualization of GBS as a demyelinating disorder to the recognition of a primary axonal form of the disease. Guillain-Barré syndrome has also come to be well recognized as a manifestation of human immunodeficiency virus (HIV) infection, usually occurring at seroconversion. Cerebrospinal fluid (CSF) pleocytosis of HIV-associated GBS stands in contrast to the characteristic albumin-cytologic dissociation typical of non–HIV-associated disease. The clinical and electrophysiologic findings of HIV-associated GBS, however, are not thought to differ substantially from the non–HIV-associated forms of the disease.

In this report, we describe 4 patients with a clinical presentation and electrophysiologic findings that may further broaden the spectrum of GBS. These were all African women who presented with severe weakness of the legs with absolute sparing of the arms and sensory nerves. All were diagnosed as having HIV infection at presentation and had previously been entirely well.

These 4 patients were identified prospectively during a 6-month period in the Department of Neurology, Groote Schuur Hospital, Cape Town, South Africa. This is an 800-bed teaching hospital with approximately 5000 patients seen annually in the Department of Neurology. All 4 patients were examined in detail by an experienced neurologist and clinical electrophysiologist (R.W.E.). All 4 patients had been entirely well previously, without antecedent HIV-defining illness, and their HIV infection was diagnosed at presentation with leg weakness. For this reason, none of the patients were receiving antiretroviral therapy at presentation.
REPORT OF CASES

CASE 1

A previously well 25-year-old African woman presented with a 6-week history of progressive weakness in both legs, being unable to walk for 3 weeks before admission. She reported neither sensory disturbance nor symptoms to suggest sphincter involvement. She maintained that her arms were entirely asymptomatic, and described no preceding constitutional upset. On examination, she was afebrile, and the abnormal findings on physical examination were confined to the nervous system. Her muscle tone was normal, and the power in her leg muscles was Medical Research Council grade 2/5 proximally and 0/5 distally. Deep tendon reflexes were absent in the legs and well preserved in the arms. The plantar reflexes were flexor. Motor examination results of the upper limbs, the mental state, and the cranial nerves were normal, as were the results of the examination of proprioception, vibration sense, light touch, and pinprick sensation. Laboratory investigation results are summarized in Table 1 and Table 2. This patient was lost to follow-up.

CASE 2

A previously well 40-year-old African woman presented with a 9-day history of weakness in both legs, resulting in difficulty with walking. The development of these symptoms was preceded by a 1-day history of nausea, vomiting, and diarrhea. She reported no symptoms to suggest sensory or sphincter involvement. Abnormal findings were confined to the motor examination of the lower limbs. Her muscle tone was normal, but the power in her legs was reduced to Medical Research Council grade 4/5 proximally and 3/5 distally. Deep tendon reflexes were absent, and plantar responses were flexor. Examination results of the upper limbs, cranial nerves, and modalities of sensation were entirely normal. Laboratory investigation results are summarized in Table 1 and Table 2. Follow-up 2 months later revealed a slight improvement (half a Medical Research Council grade) in muscle power, but significant weakness was still present. The patient was not seen subsequently, but her family has reported full recovery.

CASE 3

A previously well 19-year-old African woman was referred with a 4-week history of progressive weakness in both legs, being unable to walk for the 2 weeks preceding admission. There were no sensory or sphincter symptoms to suggest sphincter involvement. She maintained that her arms were entirely asymptomatic, and described no preceding constitutional upset. On examination, she was afebrile, and the abnormal findings on physical examination were confined to the nervous system. Her muscle tone was normal, and the power in her leg muscles was Medical Research Council grade 2/5 proximally and 0/5 distally. Deep tendon reflexes were absent in the legs and well preserved in the arms. The plantar reflexes were flexor. Motor examination results of the upper limbs, the mental state, and the cranial nerves were normal, as were the results of the examination of proprioception, vibration sense, light touch, and pinprick sensation. Laboratory investigation results are summarized in Table 1 and Table 2. A significant improvement in power followed during the ensuing 2 weeks, and this woman was able to walk (with assistance) at discharge from the hospital.

CASE 4

A 19-year-old African woman with no medical history of note presented with a 2-week history of progressive weakness in both legs. She reported neither pain nor sensory disturbance, and there were no symptoms to suggest sphincter dysfunction or cranial nerve weakness. The abnormal findings were confined to the motor examination of the lower limbs. Her muscle tone was reduced, her knee reflexes were depressed symmetrically (being

Table 1. Laboratory Data

<table>
<thead>
<tr>
<th>Variable†</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cell count, ×10^9/L</td>
<td>4.8</td>
<td>7.1</td>
<td>10.6</td>
<td>7.5</td>
</tr>
<tr>
<td>T-lymphocyte count, cells/µL</td>
<td>Not done</td>
<td>Not done</td>
<td>512</td>
<td>336</td>
</tr>
<tr>
<td>CD4 (500-2010)</td>
<td>Not done</td>
<td>Not done</td>
<td>1375</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>CSF examination</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein level, g/L</td>
<td>2.1</td>
<td>1.5</td>
<td>1.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Globulin</td>
<td>2+</td>
<td>2+</td>
<td>Trace</td>
<td></td>
</tr>
<tr>
<td>Polymorphonuclear leukocytes, per microliter</td>
<td>1</td>
<td>13</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lymphocytes, per microliter</td>
<td>21</td>
<td>67</td>
<td>81</td>
<td>0</td>
</tr>
<tr>
<td>Undifferentiated cells, per microliter</td>
<td>0</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CK level, U/L (10-110)</td>
<td>132</td>
<td>21</td>
<td>81</td>
<td>58</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate, mm/h</td>
<td>14</td>
<td>84</td>
<td>2</td>
<td>61</td>
</tr>
<tr>
<td>Polio viral culture and serologic results</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myelogram results</td>
<td>Not done</td>
<td>Not done</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Magnetic resonance imaging results</td>
<td>Not done</td>
<td>Not done</td>
<td>Not done</td>
<td>Not done</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ventral root enhancement</td>
<td>Ventral root enhancement</td>
</tr>
</tbody>
</table>

*All patients had the human immunodeficiency virus. The results of the VDRL test, Gram, Ziehl-Neelsen, and cryptococcal Indian ink stains; Mycobacterium tuberculosis and fungal cultures; and cytomegalovirus, varicella-zoster virus, and herpes simplex polymerase chain reactions were negative for all patients. The results of the porphyria screen (urine and stool) were negative for all patients. CSF indicates cerebrospinal fluid; CK, creatine phosphokinase; and ellipses, data not available.

†Normal values are given in parentheses.
elicited only with reinforcement), and her ankle reflexes were absent. Power was graded at 2/5 to 4/5 throughout both legs, with foot and toe dorsiflexion weakest, at 2/5. Laboratory investigation results are summarized in Tables 1 and 2. This patient was reexamined 3 months later, at which time full power in the legs and normal tendon reflexes were found.

RESULTS

Motor and sensory nerve conduction studies (NCSs) were performed in the upper limbs (in 3 of the 4 patients) and in the lower limbs (in all 4 patients). Motor NCSs were carried out in the median, ulnar, peroneal, and posterior tibial nerves. Sensory NCSs were performed in the sural, median, ulnar, and peroneal nerves. Late (F) responses were examined in the posterior tibial, median, and ulnar nerves. Apart from a mild amplitude reduction in the peroneal motor NCSs, these study results were all normal. In particular, the F responses were normal. A needle electromyographic examination was performed in the tibialis anterior, the vastus lateralis, and L4-5 paraspinal muscles (in 3 patients). Mild denervation changes were identified. These findings are summarized in detail in Table 2.

Sural nerve biopsies were not performed in any of the patients in view of the anticipated low diagnostic yield of sensory (sural) nerve biopsy results in patients with a purely motor syndrome. Campylobacter jejuni serology were similarly not obtained.

A magnetic resonance imaging scan was performed at presentation in patients 3 and 4. Uncontrasted T1-weighted axial images at the levels of the L1 through L3 vertebral bodies were normal, but enhancement of the ventral roots was demonstrated following the administration of gadolinium. These findings are illustrated in the Figure.

COMMENT

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.

We have described 4 patients who presented with either acute or subacute severe lower motor neuron weakness confined to the lower limbs. In all patients, reflexes were absent or depressed, sensory examination was normal, and sphincters were unaffected. The weakness was sufficiently severe in all 4 patients to prevent independent ambulation, but spontaneous recovery was noted in those patients for whom follow-up data are available. All were diagnosed as having HIV infection at presentation. To our knowledge, this is a clinical presentation not previously reported in the literature and represents either an independent clinical entity or a further variant of GBS.

Guillain-Barré syndrome is traditionally conceptualized as a particular clinical syndrome associated with characteristic CSF and electrophysiologic variables. The typical clinical presentation is that of areflexia and symmetrical ascending weakness, with the legs more affected than the arms and with relative sparing of the sensory nerve fibers. Examination of the CSF is notable for the presence of an elevated protein concentration but no pleocytosis. Nerve conduction studies typically demonstrate evidence of demyelination with prolonged distal latency; slowing of conduction velocity, temporal dispersion, and conduction block; and absent or delayed F responses.
More recently, the boundaries of GBS have expanded, and variations that differ clinically, electrophysiologically, or with regard to the CSF albumin-cytologic dissociation are recognized. However, the designation of these variants as falling within the limits of GBS is made because they retain the other essential features of this syndrome.

For example, a primary axonal form of GBS is recognized. In 1986, Feasby et al described a series of 5 patients with a particularly severe neuropathy characterized by tetraparesis with areflexia, less severe sensory disturbance, variable involvement of the cranial nerves, and a generally poor outcome. Electrophysiologic and pathological studies indicated primary axonal degeneration. The researchers believed that the clinicopathologic features of these patients warranted their consideration as a separate entity distinct from GBS. This syndrome has since been termed acute motor and sensory axonal neuropathy and is thought to represent part of a spectrum of GBS. The concept of an axonal variant of GBS is further supported by reports of cases of what has been termed acute motor axonal neuropathy. These cases were descriptions of summer epidemics of acute ascending paralysis among rural children in northern China and were characterized by a high rate of seropositivity for C. jejunii. Electrophysiologic and pathological studies confirmed this entity as a pure motor and axonal neuropathy. The clinical presentation and CSF findings of these patients were similar to those of patients with the more typical demyelinating form of the disease and unlike the clinical features of the present patients.

A further variant is that of GBS associated with HIV seroconversion. These patients demonstrate a similar clinical and electrophysiologic pattern, but the presence of CSF pleocytosis is recognized and even regarded as characteristic. Ropper has also described several additional unusual clinical variants of GBS. These include patients with predominant or pure pharyngeal-cervicobrachial weakness, severe ptosis without ophthalmoplegia, facial diplegia with paresthesiae, and motor and sensory disturbance in the legs, resembling a spinal cord lesion; and a single patient with a lumbar plexopathy. This latter presentation is of particular relevance to the patients described herein. The patient was a 70-year-old woman who developed profound weakness of both legs during a period of 1 week. Her reflexes were depressed, and sensory examination was normal. Her CSF protein level was elevated. Compound muscle action potential amplitudes, latencies, and velocities were normal in the arms and legs, as was the sural sensory nerve conduction. F-wave latencies, however, were greatly prolonged. The lumbar roots and thoracic spinal cord were normal, as determined by a magnetic resonance imaging scan with gadolinium. The patient, despite the highly unusual clinical presentation of severe motor dysfunction confined to the legs, is regarded as having GBS based on the associated characteristic CSF and electrophysiologic findings.

There are several reasons for believing that the patients described herein either do not have GBS or represent a spectrum of the syndrome not previously reported. The primary reason for making this claim is that...
the clinical scenario of isolated severe weakness confined to the legs is not a recognized presentation of GBS. In addition, the electrophysiologic studies indicate some degree of axonal loss. While it is acknowledged that GBS may manifest with primarily axonal pathological features, the patients designated as having acute motor axonal neuropathy or acute motor and sensory axonal neuropathy differed clinically from the patients described herein.

The electrophysiologic findings and their relevance to the clinical presentation of the patients described in this report merit some additional discussion. The findings of mildly decreased compound muscle action potential amplitude and fibrillations indicate some degree of axonal loss. However, the degree of weakness and the absent reflexes in the face of relatively preserved compound muscle action potential amplitudes and mild denervation changes, together with the rapid clinical recovery, suggest that the primary pathology involves the proximal motor segments. The pathology may be a conduction block despite the preserved F responses. The presence of proximal motor nerve pathology is supported by the ventral root enhancement on magnetic resonance imaging. While we cannot exclude the possibility, there is no direct evidence for distal axonal loss. It is not necessary to implicate proximal sensory conduction block, a process that might also lead to areflexia, as the clinical and electrophysiologic findings are adequately explained by the combination of proximal motor pathology and mild axonal loss. Furthermore, the absence of any sensory symptoms, signs, or sensory nerve action potential changes is consistent with relative sparing of sensory fibers.

The finding of motor root enhancement on the magnetic resonance imaging scan in these patients is useful in localizing the site of the pathology to this region. However, although it is recognized that a similar appearance has been described in patients with GBS, this appearance is nonspecific. Those patients with GBS in whom these radiological changes have been described do not bear clinical resemblance to the patients described herein but rather present with clinical syndromes typical of GBS.

Human immunodeficiency virus–associated lumbosacral polyradiculopathy is a syndrome characterized by asymmetrical leg weakness involving proximal and distal muscles and developing during a period of 1 to 6 weeks. Urinary retention or a mild sensory disturbance is invariable. The CSF is typically abnormal, with an elevated protein level and pleocytosis (either polymorphonuclear or lymphocytic). The electromyogram invariably reveals denervation, and the NCS results are usually abnormal (small or unobtainable compound muscle action potentials or prolonged or absent F responses). Cytomegalovirus infection (viral invasion of the lumbosacral roots) is the most commonly identified cause (demonstrated by culture results from the CSF) and usually occurs in the setting of polymorphonuclear CSF pleocytosis. This syndrome usually occurs in the setting of established disease.

In contrast to patients with lumbosacral polyradiculopathy, the 4 patients described herein all presented with a motor syndrome that was remarkable for the absence of sensory and sphincter symptoms. Furthermore, they presented early in the course of HIV infection (no acquired immunodeficiency syndrome–defining illnesses and preserved CD4 cell counts in patients 3 and 4), and study results for cytomegalovirus (CSF and polymerase chain reaction) were negative in all 4 patients. The NCS and electromyographic findings in the patients described herein also differed from those described in patients with lumbosacral polyradiculopathy.
Of possible relevance to the syndrome described in this article is the report of Verma et al., who described an HIV-positive patient with progressive weakness of the arms and legs, widespread fasciculations, normal tendon reflexes, and normal sensation. The results of motor NCSs were normal, and an electromyographic examination demonstrated widespread denervation, suggesting a diagnosis of polyradiculopathy or lower motor neuron disease. An autopsy 2 years later revealed scarring of the ventral roots, moderate loss of myelinated nerve fibers in the peripheral nerves, and mixed features of neurogenic and myopathic muscle atrophy. These findings led to the all-encompassing diagnosis of myeloradiculoneuropathy and myopathy. This report raises the possibility of anterior horn cell dysfunction, in the context of HIV infection, as a cause of weakness. Although the radiological changes in the patients presented herein indicate motor root pathology, the clinical, CSF, and electrophysiologic features are compatible with anterior horn cell dysfunction.

In summary, therefore, we have described 4 patients who presented with a previously unrecognized clinical syndrome in the context of recent HIV infection. The clinical, CSF, electrophysiologic, and radiological findings cohere to implicate the motor roots as the primary site of the pathology. Although we have been unable to define a specific etiologic factor in these patients, their presentation in the context of newly diagnosed HIV infection raises the suspicion that the motor neuropathy is somehow related to this infection. The spontaneous recovery recorded in those patients for whom follow-up is available is consistent with that observed in patients with GBS. It remains unclear whether this syndrome is best regarded as a variant of GBS or as a distinct clinical entity.

Accepted for publication January 24, 2000.

Corresponding author: Michael G. Benatar, MBChB, DPhil, Department of Medicine, Suite KS-406, Beth Israel Deaconess Medical Center, 330 Brookline Ave, Boston, MA 02215 (e-mail: mbenatar@caregroup.harvard.edu).

REFERENCES