Arsenic-induced polyneuropathy is traditionally classified as an axonal-loss type, electrodiagnostically resulting in low amplitude or absent sensory and motor responses, relatively preserved proximal and distal motor conduction rates, and distal denervation. We report four patients with a subacute onset progressive polyradiculoneuropathy following high-dose arsenic poisoning. In three patients, early electrodiagnostic testing demonstrated findings suggestive of an acquired segmental demyelinating polyradiculoneuropathy. Serial testing confirmed evolution into features of a distal dying-back neuropathy. We hypothesize that arsenic toxicity and the resultant biochemical derangement of the peripheral nerve cell leads to subtle changes in axonal function that produce, initially, segmental demyelination and eventually distal axonal degeneration. Acute arsenic toxicity must be suspected in patients with clinical and electrodiagnostic features supporting Guillain-Barré syndrome.

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ACUTE ARSENIC INTOXICATION PRESENTING AS GUILLAIN-BARRÉ–LIKE SYNDROME

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Peripheral neuropathy is a common and greatly feared complication of arsenic toxicity. The earliest clinical features of acute, high-dose arsenic poisoning reflect multiorgan involvement, which is manifested often as an acute gastroenteritis variably associated with encephalopathy, pancytopenia, hepatitis, cardiomyopathy, and dermatitis.[12,16,17,33] In many patients, a subacute polyneuropathy begins 7–14 days following arsenic exposure, eventually leading to a severe chronic painful sensorimotor polyneuropathy.[2,3,12,16,17,33] Long-term low-level arsenic exposure may result in a chronic neuropathy without history of a preceding gastrointestinal disturbance.[2,16,27]

Most reports of the electrodiagnostic findings in arsenic polyneuropathy describe profound sensorimotor axonal-loss involvement. We report four cases of subacute polyradiculoneuropathy following single or multiple high-dose arsenic intoxication. Early electrodiagnostic testing revealed features indistinguishable from a segmental demyelinating polyradiculoneuropathy, typical of those commonly recorded in acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome). Serial electrodiagnostic testing in these patients documented evolution into features commonly reported in chronic arsenic neuropathy, i.e., a sensorimotor distal axonal-loss neuropathy.

CASE REPORTS

Case 1. A 61-year-old man, his wife, and daughter developed nausea, vomiting, and diarrhea following ingestion of their usual Sunday meal. The daughter's symptoms resolved after 24 hours. Six days later, because of persistence of symptoms, the patient and his wife sought medical consultation; they were hospitalized for a preliminary diagnosis of viral hepatitis. She subsequently expired following the sudden onset of an intractable ventricular arrhythmia. On developing paresthesia in his hands and feet, the patient was transferred to the Cleveland Clinic for further evaluation.

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Initial physical examination revealed normal vital signs and decreased breath sounds at both bases. Strength was mildly decreased in the distal lower extremities. Muscle stretch reflexes were normal in the upper extremities, reduced at the knees, and absent at the ankles. Sensory examination demonstrated severe vibratory and position loss in a stocking-glove distribution.

Initial laboratory studies revealed marked anemia (Hb 10.6 g), leukopenia (WBC 2900), elevated liver function studies (SGOT 608), and azotemia. Chest x-ray confirmed bilateral pleural effusions. Electrodiagnostic testing revealed findings suggestive of a developing acquired segmental demyelinating polyradiculoneuropathy, most consistent clinically with Guillain-Barré syndrome.

Within days of hospitalization, all reflexes disappeared and sensory signs progressed to the midforearm and midthigh regions. Weakness progressed in an ascending fashion to involve proximal muscles. Approximately 1 week following admission, skin desquamation developed over his soles and palms. Initial spinal fluid analysis was unremarkable, but a repeat tap disclosed acellular fluid and a protein of 193 mg/dl. A sural nerve biopsy obtained in the seventh week of hospitalization demonstrated severe axonal and myelin degeneration. The patient's clinical status stabilized 6 weeks following onset of gastrointestinal symptoms. Exhaustive toxicologic screening uncovered a pubic hair arsenic level of 2895 μg/g of hair (normal <65). Arsenic was also discovered in liver and kidney of the patient's wife on autopsy.

Case 2. A 63-year-old man was transferred to the University of Michigan Hospital with a 2-week history of headache, confusion, nausea, anorexia, weakness, and distal extremity dysesthesia. Previous hospitalization at an outside hospital had disclosed elevated liver function studies, renal insufficiency, pancytopenia, pulmonary infiltrates, and a skin rash.

On general physical examination, the patient was afebrile and in mild respiratory distress. An erythematous maculopapular rash was present on his trunk and proximal legs, and mild pitting edema was noted at the ankles. Diffuse inspiratory and expiratory wheezes, scattered rhonchi, and dullness to percussion at both bases were present.

The patient was alert, but irritable, inattentive, and uncooperative. Motor examination revealed mild atrophy of intrinsic hand muscles and mild weakness distally in all extremities. Muscle stretch reflexes were absent at the ankles and decreased elsewhere. Sensory examination revealed a gradient loss to all modalities in a stocking-glove distribution.

Initial laboratory studies included WBC 1300, hematocrit 27, platelet count 129,000, blood urea nitrogen (BUN) 18, creatinine 1.6, total bilirubin 0.8, SGOT 120, and SGPT 271. On chest x-ray, vascular changes consistent with congestive heart failure were noted. Electroencephalogram (EEG) was diffusely abnormal, suggesting a metabolic or toxic disturbance. Cerebral spinal fluid (CSF) analysis was normal. Electrodiagnostic findings were those of an acute motor and sensory demyelinating polyradiculoneuropathy.

Several days after admission, his mental status and rash improved; however, sensory and motor loss slowly progressed, resulting in a flaccid quadriplegia with areflexia. Repeat electrophysiologic studies indicated worsening of proximal conduction block and conduction velocity slowing and severe axonal involvement. Progressive hypoxic ventilatory failure ensued secondary to superimposed pneumonia, congestive heart failure, and bilateral phrenic nerve dysfunction. Subacute progression of the quadriplegia, with respiratory failure, strongly suggested the diagnosis of Guillain-Barré syndrome. Arsenic poisoning was confirmed by the following abnormal laboratory tests: urine arsenic 35,000 ppb (>50 is toxic), fingernail arsenic level 8.6 μg/g (normal 0.030–0.36), and pubic hair arsenic level of 160 ppm (normal 0.05–0.5).

Treatment with British anti-Lewisite (BAL) resulted in a brisk arsenic diuresis, but no clinical amelioration. The patient remained quadriplegic for months.

Case 3. A 62-year-old man was briefly hospitalized for 3 days because of shortness of breath, nausea, vomiting, diarrhea, malaise, myalgia, and arthralgia. These symptoms abated after 3 weeks, but were followed by the onset of paresthesia and dysesthesia in his fingers and toes. Two weeks after the onset of sensory complaints, the patient was rehospitalized for impaired dexterity in his hands and feet, progressive weakness in climbing stairs, and difficulty in appreciating the position of his extremities.

On systems review, the patient complained of a recent change in his coffee flavor and stated that he was afraid his daughter may have poisoned him.

Physical examination revealed distal greater than proximal weakness in all limbs, areflexia, and a stocking-glove gradient loss to pin, vibration, and position sense.

Extensive laboratory investigation failed to

identify an etiology for the subacute polyneuropathy. Lumbar puncture was unremarkable, except for a mild elevation of protein (51 mg/dl, normal <45). A 24-hour urine heavy metal screen disclosed an arsenic level of 76 μg/liter (normal <50). Pubic hair arsenic level was 4.2 μg/g of hair (normal 0.05–1.9). Electrodiagnostic studies revealed abnormalities consistent with a severe sensory and motor axonal and demyelinating polyradiculoneuropathy.

Prior to discovery of the toxicology results, acute idiopathic demyelinating polyradiculoneuropathy (Guillain-Barré syndrome) was considered to be the most likely diagnosis. The patient underwent intensive physical therapy and occupational rehabilitation during hospitalization, and at the time of discharge, was able to ambulate using a walker.

Case 4. A 31-year-old man abruptly developed nausea, vomiting, and diarrhea lasting 1 week. Nine days following the onset of gastrointestinal symptoms, he began to appreciate numbness and tingling of both feet. Within days, these sensory complaints progressed, accompanied by hand weakness and gait unsteadiness.

On transfer to the University of Michigan Medical Center, 3 weeks after disease onset, his general physical examination disclosed sinus tachycardia and a loud systolic murmur. Neurologic examination was noteworthy for distal greater than proximal quadriparesis, lower extremity hyporeflexia, and marked vibratory and position sense loss at the fingers, ankles, and toes.

Initial laboratory tests disclosed a prominent pancytopenia and abnormal liver (SGPT 86, normal <35) and pancreatic (amylase 452, normal 23–100) function tests. A bone marrow aspirate was markedly hypocellular, with megaloblastic changes and basophilic stippling. Westergren sedimentation rate was 70 mm/hr (normal <10) and antinuclear antibody (ANA) was positive in a speckled pattern at 1:80. CSF analysis disclosed clear acellular fluid and a protein of 270 mg/dl (normal <45). Cardiomegaly and bilateral pleural effusions were noted on chest x-ray. Early electrodiagnostic testing results were consistent with an acquired sensory and motor demyelinating polyradiculoneuropathy.

Initial treatment consisted of six plasma exchanges for the presumptive diagnosis of Guillain-Barré syndrome. Nevertheless, the patient continued to deteriorate, eventually attaining a state of quadriplegia and respiratory dependency 6 weeks after disease onset. A toxic cause for his condition was confirmed on receiving results of an initial 24-hour urine heavy metal screen, which reported an arsenic level of 1500 μg/ml (toxic is >50). In the course of time, a brown desquamated palm and sole rash and finger and toe nail Mee's lines evolved. Penicillamine treatment was initiated, but was discontinued after 2 weeks because of a drug-induced fever.

MATERIALS AND METHODS

Motor and sensory conduction studies were performed using standard techniques of supramaximal percutaneous stimulation and surface electrode recording. Motor and sensory nerve evoked response amplitudes were measured from baseline to negative peak. Conduction velocity was measured in the forearm and leg segment in all cases and across the shoulder and upper arm in case 1. Temporal dispersion and/or conduction block along motor nerves was expressed as the ratio of the proximal to the distal compound muscle action potential (CMAP) amplitude. F-response latencies were recorded as the minimal latency in a series of 10 F-responses using distal (wrist or ankle) antidromic motor nerve stimulation. For all conduction studies, skin temperature was measured and maintained above 32°C using warm compresses.

Criteria for demyelination have been published previously and are similar to those reported by Kelly.[4,19] This required identification of at least one of the following in two or more nerves (exceptions noted): (1) conduction velocity (CV) less than 95% of lower limit of normal (LLN) if amplitude exceeded 50% of LLN, less than 85% if amplitude less than 50% of LLN; excluded were isolated ulnar or peroneal nerve abnormalities at the elbow or knee, respectively; (2) distal latency exceeding 110% of upper limit of normal (ULN) if amplitude normal, exceeding 120% of ULN if amplitude less than LLN; excluded were isolated median nerve abnormalities at the wrist; (3) evidence of excessive motor temporal dispersion and/or conduction block, defined as a proximal to distal CMAP amplitude ratio less than 70%; excluded were isolated ulnar or peroneal lesions as above, as well as any studies with evidence of anomalous innervation (e.g., median to ulnar nerve crossover); and (4) F-response latency exceeding 120% of ULN with combined exclusions from above.

Electromyography was performed using standard concentric needle electrodes. Insertional activity, spontaneous activity at rest, and motor unit recruitment and morphology were analyzed and scored subjectively.

Table 1. Electrodiagnostic results.

	Patient 1			Patient 2			Patient 3		Patient 4			
Days following onset of sensory complaints	5	12	36	9	15	35	35	11	18	25	38	80
Sensory nerve studies												
Sural												
Amp. (6–47, μV)	NR	NR	NR	7.0	—	NR	NR	NR	NR	NR	—	NR
DL (3.2–4.2 msec)				3.2								
Median												
Amp. (>20 μV)	4.0	NR	NR	12.0	—	—	NR	5.0	NR	NR	NR	NR
CV (53–73 m/sec)	—			—				—				
DL (2.5–3.7 msec)	3.5			3.2				3.5				
Motor nerve studies												
Stimulation point												
Peroneal			0.15/0.10	4.0/3.8	2.0/1.5	0.1	0.5/0.15	4.8/3.8	1.2/1.2	0.4/0.2	0.1/NR	NR
Amp. (2–12 mV) A/K	6.5/4.5	4.4/1.8	24.0	42.0	34.0	—	24.0	36.0	35.0	26.0	—	
CV (41–57 m/sec)	45.0	38.0	8.8	5.6	4.4	7.4	5.3	4.3	5.8	7.2	9.2	
DL (3.3–6.1 msec)	3.4	4.0	NR	—	NR	—	—	76.0	64.0	—	—	
F-response (<55 msec)	55.5	NR										
Tibial			0.20/0.05	5.0	3.0/0.5	0.2	1.6		2.5/1.5	1.3/0.3	NR	NR
Amp. (3–25 mV) A/K	7.0/4.0	5.0/2.0	22.0	—	31.0	—	—		37.0	31.0		
CV (41–53 m/sec)	42.0	38.0	8.8	4.2	3.8	9.5	4.9		4.4	5.8		
DL (2.7–6.1 msec)	4.9	5.9	NR	—	NR	—	—		81.0	NR		
F-response (<55 msec)	73.5	74.0										
Median												
Amp. (4–18 mV) W/E	10.0/8.0	5.0/1.0	0.5/0.05	8.0/7.0	—	NR	4.6/3.0	4/1.5	1.0/0.1	0.1/0.1	NR	NR
CV (49–70 m/sec)	51.0	32.0	28.0	46.0			42.0	48.0	52.0	37.0		
DL (2.4–4.4 msec)	3.7	4.0	6.0	4.0			3.7	3.6	4.4	5.6		
F-response (<31 msec)	51.5	NR	NR				33.1	48.0	42.0	37.4		
Ulnar												
Amp. (6–16 mV)	13/11/10/8	13/6/4.5/2	5.6/1.2/1/0.4	8.5/7.0	—	—	5.0		4.5/3.2	3.8/2.0	1.0/0.2	NR
W/E/AX/Erb												
CV (49–71 m/sec)	55/50/40	40/42/36	31/35/33	49.0			55.0		55.0	38.0	29.0	
DL (1.8–3.5 msec)	2.5	3.2	3.8	3.3			3.6		2.9	3.5	4.2	
F-response (<31 msec)	45.0	42.0	50.0	41.0			25.1		41.4	44.5	NR	
Electromyography (muscle)												
First dorsal interosseous												
Fibrillation potentials	—	0	1+	0	—	2+	4+	—	—	2+	—	4+
Recruitment	—	↓ ↓ #	↓ ↓ ↓ #	Normal	—	Absent	↓ ↓ #	—	—	Absent	—	Absent
Quadriceps												
Fibrillation potentials	—	0	2+	0	—	2+	3+	—	—	3+	4+	3+
Recruitment	—	↓ #	Absent	Normal	—	Absent	↓ ↓ #	—	—	Absent	Absent	Absent
Tibialis anterior												
Fibrillation potentials	—	0	2+	0	2+	4+	4+	—	—	3+	—	4+
Recruitment	—	↓ #	Absent	Normal	↓ ↓ ↓ #	Absent	↓ ↓ ↓ #	—	—	Absent	—	Absent

Stimulation point—A, ankle; K, knee; W, wrist; E, elbow; AX, axilla; Erb, Erb's point.
NR, no response; (—), not done.
Fibrillation potentials—1+, persistent trains, 2 areas; 2+, 3 or more areas; 3+, many, all areas; 4+, filling baseline, all areas.
Recruitment—↓ #, mildly reduced; ↓ ↓ #, moderately reduced; ↓ ↓ ↓ #, markedly reduced.

ELECTRODIAGNOSTIC FEATURES

Table 1 displays serial nerve conduction study (NCS) and electromyography results of each patient, using the number of days following onset of sensory complaints as a point of reference. A definite trend is apparent on separating NCS results into early, intermediate, and late findings. In patients 1, 2, and 4, early testing (days 5, 9, and 11, respectively) identified absent or reduced sensory nerve action potentials, partial conduction block along motor nerves, as reflected in an abnormal proximal/distal CMAP amplitude ratio, and slowed proximal motor nerve conduction (prolonged F-response latency). Repeat evaluation approximately 1 week after initial testing (intermediate phase) disclosed in patients 1, 2, and 4 absent sensory responses and more profound motor conduction dysfunction (Fig. 1). In each case, when compared to the previous study, there was deterioration in the CMAP amplitudes, a greater degree of conduction slowing or block both distally (proximal/distal CMAP amplitude ratio) and proximally (slowed or absent F-response latency), as well as slowing in conventional conduction velocity. One month following the development of polyneuropathy, electrodiagnostic results identified further worsening in the proximal and distal conduction. Needle exam early in arsenic polyneuropathy revealed normal or slightly increased insertional activity at rest and reduced recruitment of voluntary motor unit potentials (MUP). Unequivocal evidence of denervation (fibrillation potentials) was recorded in patients 1 and 2 at 2 weeks and in patient 4 at 3 weeks following the onset of neuropathic symptoms. Late results in all four patients confirmed severe distal and proximal denervation, paraspinal muscle denervation, and profound reduction in MUP recruitment in a similar distribution.

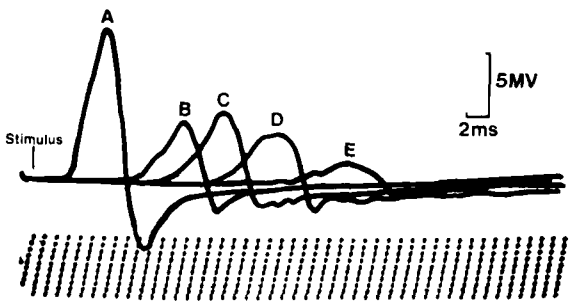

FIGURE 1. Surface recording of the CMAP amplitude from the abductor digiti minimi muscle in case 1 with stimulation of the right ulnar nerve at the wrist (**A**), below elbow (**B**), above elbow (**C**), axilla (**D**), and Erb's point (**E**). Recording was performed 12 days subsequent to the onset of sensory complaints. Note partial conduction block with stimulation at the elbow (**B**) and Erb's point (**E**).

DISCUSSION

Many sources for arsenic poisoning have been reported, including accidental (pesticide exposure, Paris green),[1,2,12,15,18,33] iatrogenic and medicinal (Donovan's solution, arsphenamine, Fowler's solution),[2,22,33] illicit alcohol manufacturing and consumption,[16,17,25] arsenic gas leakage,[32] well water contamination,[7] and cutaneous absorption.[10,15] Most contemporary reviews attest to homicide or suicide attempts or accidental ingestion as the most common causes of arsenic poisoning in our society.[1,16,17,27,33]

High-dose ingestion of arsenic results in severe gastrointestinal symptoms of burning, pain, vomiting, and diarrhea within hours of exposure.[12,16,17,33] These symptoms often last several days. These complaints are invariably followed by a constellation of features reflecting multiple organ involvement, a testimony to arsenic's role as a systemic protoplasmic poison. These may include encephalopathy,[16,32,33] pancytopenia,[1,12,13,15,20,30,32] eosinophilia,[23] hemaglobinuria,[32] hepatic involvement,[1,3,30] jaundice,[3] renal tubular necrosis,[1] burning and dryness of mucus membranes,[1,3,16,17] peripheral edema,[4,15,17,29] respiratory failure,[13] cardiomyopathy,[2,13] electrocardiographic abnormalities,[11] pancreatitis,[17] dermatitis,[1–3,12,16,17,33] and reduced or exaggerated sweating.[2,16,17] On elimination of the arsenic source, these features of toxicity abate. If untreated, a subacute polyneuropathy begins 7–14 days following arsenic exposure, which may progress in an ascending fashion to involve proximal arms and legs. Symmetrical distal weakness, myalgia, cramping, and hypoactive or absent muscle stretch reflexes soon follow. The severity of clinical involvement spans the spectrum from mild paresthesia with preserved ambulation to quadriplegia and respiratory failure.[13] This subacute form of arsenic poisoning may evolve into a severe chronic sensorimotor polyneuropathy with distinctive associated clinical characteristics, such as hyperpathia, Mee's lines, palm and sole hyperkeratosis, and a pigmented dermatitis.

The electrophysiologic findings in chronic arsenic polyneuropathy have been described in most large series and texts as those of an axonal-loss sensorimotor polyneuropathy, i.e., low amplitude/unelicitable sensory and motor conduction responses, often with preserved motor conduction velocities.[3,9,12,33] Some authors have reported slow-

ing in sensory and motor conduction velocity, but specific values and/or the degree of reduction were not provided.[17,29,32] Chhuttani[3] and Goldstein[12] have independently described electrodiagnostic data in patients with subacute or chronic arsenic polyneuropathy. In both groups, some motor slowing was present, yet CMAP amplitudes, the presence or absence of dispersion with proximal stimulation, and motor nerve distal or F-response latencies were unreported.[3,12]

A few reports have described the electrodiagnostic results recorded in patients who developed peripheral polyneuropathy within several weeks of high-dose arsenic exposure. LeQuesne and McLeod[21] described four such patients. In two of them, sensory responses were absent, yet conventional motor conduction velocities were slow, motor distal latencies were markedly prolonged, and a 50% or greater loss in CMAP amplitude was apparent on proximal versus distal nerve stimulation in at least one nerve. Murphy et al.[23] published data on two patients whose clinical presentation and electrophysiologic results were similar to our four cases. Both patients developed a subacute polyneuropathy and electrodiagnostic features of absent sensory responses and moderately reduced motor CVs 2 and 4 weeks, respectively, after arsenic exposure. Maximum slowing was noted 3 months after exposure. A similar case was described by Feit et al.,[8] who documented moderate slowing of motor CVs 3 weeks following disease onset. In this as well as other instances, an initial diagnosis of Guillain-Barré syndrome was made prior to obtaining final results of toxicology screening.

Serial electrophysiologic data in our four patients suggest that arsenic poisoning of the peripheral nervous system may begin as a segmental demyelinating polyradiculoneuropathy. Supporting this theory are early electrodiagnostic features of (1) partial conduction block, as evidenced by reduced CMAP on proximal versus distal stimulation, (2) sufficient slowing in conventional motor CV to invoke a demyelinating process, and (3) proximal CV slowing or conduction block as determined by prolonged or absent F-responses. Only on subsequent NCS recorded later in the illness were results obtained similar to those reported by other authors as reflecting primarily axonal involvement.[3,9,12,33] Conversely, NCS in chronic ingestion of arsenic do not show a segmental demyelinating component, presumably because, at any one point in time, not enough fibers are demyelinated to produce either detectable conduction block, focal slowing, or differential slowing. This early reduction and disappearance of sensory nerve action potentials prior to motor nerve involvement is atypical for an early, nondemyelinating, axonal-loss polyneuropathy. Wilbourn[31] has demonstrated that the motor amplitude falls and disappears several days before the sensory response in early Wallerian degeneration. The early disappearance of sensory before motor responses in arsenic polyneuropathy is difficult to explain using an axon-loss model and is presumably secondary to distal conduction block or differential slowing in sensory fibers.

Most detailed descriptions of peripheral nerve pathology in chronic arsenic polyneuropathy cite axonal degeneration of large more than small fibers as the major feature.[33] Increased cellularity and fibrous tissue, thickened perineurium, and a reduced number of myelinated fibers have been observed by many authors on examination of sural or lower extremity digital nerve biopsies.[2,3,12,16,21] Longo et al.[22] reported extensive degeneration of distal ramifications of peripheral nerve fibers that were typical of changes seen in Wallerian degeneration. Ohta[24], based on results of routine light microscopy and teased single-fiber preparations, stated that the main pathologic features were those of Wallerian degeneration. Nevertheless, other authors have found pathologic support for a demyelinating process. Teased fiber preparations of three patients described by Chhuttani, citing Chopra's results, showed evidence of segmental demyelination and remyelination of mild, moderate, and severe degree.[3] Goldstein et al.,[12] found a small percentage of teased fibers with segmental demyelination in case 9.

Modern autopsy data of central and peripheral nervous system abnormalities in early or subacute arsenic poisoning is lacking, as patients rarely expire from the early complications of arsenic toxicity. In 1892, Erlicki and Rybalkin[6] reported detailed clinical observations in two patients with subacute arsenic polyneuropathy. An autopsy in one severely affected patient revealed a reduction in the number of anterior horn cells and nuclear and cytoplasmic abnormalities in the remaining anterior horn cells. Pathologic studies in animals receiving intravenous injections of arsenic have also demonstrated cytoplasmic changes and severe destruction in anterior horn cells within 2 weeks of exposure.[14,26]

These autopsy and animal toxicity studies suggest that arsenic may be primarily toxic to the nerve cell body, leading eventually to axonal dysfunction that produces, initially, segmental demyelination (most apparent proximally) before axonal degen-

eration ensues. Possibly other toxic, axon-loss polyneuropathies begin with segmental demyelination. In one model of toxic polyneuropathy, Dyck et al.[5] identified unequivocal evidence of segmental demyelination in teased-fiber preparations from patients with uremia. They postulated that uremic neuropathy evolves through several phases of myelin change, resulting in secondary segmental demyelination before degeneration of the axon is fully manifested. It was their conclusion that segmental demyelination may be present pathologically in neuropathies heretofore considered to be purely axonal. Likewise, Thomas stated that a combination of axonal degeneration and segmental demyelination is found on careful examination of most neuropathies.[28]

In summary, four cases of arsenic-induced subacute polyradiculoneuropathy with early electrodiagnostic studies indistinguishable from an acquired segmental demyelinating polyradiculoneuropathy are reported. All were initially diagnosed as Guillain-Barré syndrome. This potential clinical pitfall cautions us to consider arsenic toxicity in any patient with clinical and electrodiagnostic features of Guillain-Barré syndrome heralded by gastrointestinal symptoms.

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